United States
Consumer Product Safety Commission

July 2, 2024

Via Email and Box
Roman Jankowski
214 Massachusetts Avenue
Washington, District of Columbia 20002
Roman.Jankowski@heritage.org

RE: Freedom of Information Act Request #23-F-00294: I respectfully request the following records:

A. All records related to CPSC Commissioner Richard L Trumka's memo titled NPR Proposing Ban on Gas Stoves (Indoor Air Quality)
B. All records regarding voluntary standards and natural gas stoves
C. All record regarding ANSI Z21.1-2016 or CSA 1.1-2016
D. All communications with other federal agencies, including but not limited to the Department of Energy, Environmental Protection Agency, and the Federal Energy Regulatory Commission, regarding natural gas stoves.
E. All communications with the Office of the President or Executive Office of the President regarding natural gas stoves.
[Please see request for details]. (Date Range for Record Search: From 12/1/2021 To 1/8/2023)

Dear Mr. Jankowski:

Thank you for your Freedom of Information Act (FOIA) request seeking records from the U.S. Consumer Product Safety Commission (CPSC). On June 21, 2023, we sent you correspondence advising you that your request presented unusual circumstances and would take up to an additional nine months to process from that date because the request sought records from several CPSC program offices and involved a volume of potentially responsive records approximating 5,000 pages. By email dated January 3, 2024, we informed you that some of the records responsive to your request were processed in other FOIA requests and posted to our website. Those records are available at the following links (with the corresponding FOIA Request numbers provided in parentheses):

Newsroom - FOIA | CPSC.gov (23-F-00274)

Newsroom - FOIA | CPSC.gov (23-F-00255)

Newsroom - FOIA | CPSC.gov (23-F-00226)

In a supplemental response dated March 29, 2024, we provided additional records – the responsive emails from the CPSC's Office of Executive Director (OEX), consisting of 570 pages of emails and attachments.

This is a second supplemental response. The responsive emails from the CPSC's Office of Legislative Affairs (OLA) have been processed and are enclosed. These consist of 1497 pages of emails and attachments. Additional records are being reviewed and will be made available on a rolling basis in another supplemental response.

Portions of the enclosed records are withheld pursuant to FOIA Exemptions 5 and 6, 5 U.S.C. §§ 552(b)(5) and (b)(6). CPSC considered the foreseeable harm standard when reviewing these records and applying FOIA exemptions.

Exemption 5. FOIA Exemption 5 permits withholding from disclosure inter-agency and intra-agency memoranda that would not be available, by law, to a party other than an agency in litigation with the agency. The staff memoranda and analyses being withheld are pre-decisional and deliberative, consisting of recommendations, opinions, suggestions, and analyses of technical and/or legal staff. Any factual materials in the memoranda not covered by some other exemption are inextricably intertwined with exempt materials, or the disclosure of the factual materials would expose the deliberative process and/or violate the attorney-client privilege. It would not be in the public interest to disclose these materials because disclosure would impair the frank exchange of views necessary for such matters. Accordingly, we are withholding portions of the enclosed emails which contain pre-decisional draft documents and staff deliberations under this exemption where noted.

Exemption 6. FOIA Exemption 6 permits withholding personnel and medical files and similar files, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy. Absent authorizations to disclose the records or personally identifying information from the persons identified in the records or their representatives, such information falls within the protection of this FOIA exemption to disclosure and is withheld accordingly.

FOIA Administrative Procedures

Right to appeal. According to the CPSC's regulations implementing the FOIA at 16 C.F.R. § 1015.7, a partial denial of access to records may be appealed. If you are not satisfied with the response to this request, you may administratively appeal in writing, addressed to FOIA APPEAL, Office of the General Counsel, ATTN: Division of Information Access, U.S. Consumer Product Safety Commission, 4330 East West Highway, Room 820, Bethesda, MD 20814-4408.

Your appeal must be postmarked or electronically transmitted (cpscfoiarequests@cpsc.gov) within 90 days of the date of the response to your request. You may also fax your appeal to 301-504-0127. You may contact us Monday – Friday from 8:00AM – 4:30PM ET, by telephone at 1-800-638-2772, by fax to 301-504-0127, or by email to cpsc-foia@cpsc.gov.

Before filing a formal appeal with the CPSC, you may contact me, CPSC's FOIA Public Liaison, via email or at 1-800-638-2772, for any further assistance, or to discuss any aspect of your request. Assistance may include guidance on reformulation of your request or an alternative period for processing the request.

Right to Mediation. Additionally, you may contact the Office of Government Information Services (OGIS) at the National Archives and Records Administration to inquire about the FOIA mediation services they offer. The contact information for OGIS is as follows: Office of Government Information Services, National Archives and Records Administration, 8601 Adelphi Road-OGIS, College Park, MD 20740-6001; email: ogis@nara.gov; telephone: 202-741-5770; toll free: 1-877-684-6448; fax: 202-741-5769.

Fees. We are not charging you fees in this instance to cover the costs to the CPSC in processing this request, performing the file searches and preparing the information.

Sincerely,

ROBERT DALTON

Digitally signed by ROBERT DALTON
Date: 2024.07.02 10:54:45 -04'00'

Robert Dalton

Supervisory Attorney
Office of the General Counsel
Division of Information Access
(301) 504-7246
rdalton@cpsc.gov

Enclosures: Office of Legislative Affairs Emails (with Attachments) – 1497 pages

From: Jankowski, Roman
To: CPSC FOIA
Cc: OversightProject
Subject: New FOIA request part 1 of 4
Date: Monday, February 6, 2023 3:52:41 PM
Attachments: CSPC request 2-6-2023.pdf

Attached is a new FOIA request part 1 of 4.

Roman Jankowski

Senior Investigative Counsel, Oversight Project, Government Relations
The Heritage Foundation
214 Massachusetts Avenue, NE
Washington, DC 20002
202-608-1503

heritage.org

Email secured by Check Point

The Heritage Foundation
214 Massachusetts Avenue, NE
Washington, DC 20002
(202) 546-4400
heritage.org

February 6, 2023

Dear Abioye Mosheim,

Pursuant to the Freedom of Information Act, 5 U.S.C. § 552, and the implementing FOIA regulations of the Consumer Safety Product Commission ("CSPC"), 16 CFR Part 1015, I respectfully request the following records:

A. All records related to CPSC Commissioner Richard L Trumka's memo titled *NPR Proposing Ban on Gas Stoves (Indoor Air Quality)*
B. All records regarding voluntary standards and natural gas stoves
C. All record regarding ANSI Z21.1-2016 or CSA 1.1-2016
D. All communications with other federal agencies, including but not limited to the Department of Energy, Environmental Protection Agency, and the Federal Energy Regulatory Commission, regarding natural gas stoves.
E. All communications with the Office of the President or Executive Office of the President regarding natural gas stoves

Please limit the search for this request from December 1. 2021 to Jan 8, 2023. Please include the following custodians in any search:

A. Office the Chairman, Alexander Dennis "Alex" Hoehn-Saric,
B. Office of Commissioner Peter Feldman
C. Office of Commissioner Mary T. Boyle
D. Office of Commissioner Richard L. Trumka Jr.
E. Office of Communications
F. Office of the Inspector General
G. Office of Equal Employment Opportunity and Minority Enterprise
H. Office of the Executive Director
I. Office of the General Counsel
J. Office of Legislative Affairs

The term "record" means any written, recorded, or graphic matter of any nature whatsoever, regardless of how recorded, and whether original or a copy, including, but not limited to, the following: memoranda, reports, expense reports, books, manuals, instructions, financial reports, working papers, records, notes, letters, notices, confirmations, telegrams, receipts, appraisals, pamphlets, magazines, newspapers, prospectuses, inter-office and intra-office communications, electronic mail (emails), MMS or SMS text messages, instant messages, messaging systems (such as iMessage, Microsoft Teams, WhatsApp, Telegram, Signal, Google Chat,

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Twitter Direct Messages, Lync, Slack, and Facebook Messenger), contracts, cables, telexes, notations of any type of conversation, telephone call, voicemail, meeting or other communication, bulletins, printed matter, computer printouts, teletypes, invoices, transcripts, diaries, analyses, returns, summaries, minutes, bills, accounts, estimates, projections, comparisons, messages, correspondence, press releases, circulars, financial statements, reviews, opinions, offers, studies and investigations, questionnaires and surveys, work sheets (and all drafts, preliminary versions, alterations, modifications, revisions, changes, and amendments of any of the foregoing, as well as any attachments or appendices thereto), graphic or oral records or representations of any kind (including without limitation, photographs, charts, graphs, microfiche, microfilm, videotape, recordings and motion pictures), electronic or mechanical records or representations of any kind (including, without limitation, tapes, cassettes, disks, and recordings), and other written, printed, typed, or other graphic or recorded matter of any kind or nature, however produced or reproduced, and whether preserved in writing, film, tape, disk, videotape or otherwise. A record bearing any notation not a part of the original text is to be considered a separate record. A draft or non-identical copy is a separate record within the meaning of this term.

The term "communication" means each manner or means of disclosure or exchange of information (in the form of facts, ideas, inquiries, or otherwise), regardless of means utilized, whether oral, electronic, by document or otherwise, and whether in an in-person meeting, by telephone, facsimile, e-mail (desktop or mobile device), MMS or SMS text message, instant messages, messaging systems (such as iMessage, Microsoft Teams, WhatsApp, Telegram, Signal, Google Chat, Twitter Direct Messages, Lync, Slack, and Facebook Messenger), regular mail, telexes, releases, or otherwise.

"Communications with," "communications from," and "communications between" means any communication involving the related parties, regardless of whether other persons were involved in the communication, and includes, but is not limited to, communications where one party is cc'd or bcc'd, both parties are cc'd or bcc'd, or some combination thereof.

The terms "and" and "or" should be construed broadly and either conjunctively or disjunctively to bring within the scope of this request any information which might otherwise be construed to be outside its scope. The terms "all," "any," and "each" should each be construed as encompassing any and all. The singular includes

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the plural number, and vice versa. The present tense includes the past and vice versa. The masculine includes the feminine and neuter genders.

Please consider all members of a document "family" to be responsive to the Request if any single "member" of that "family" is responsive, regardless of whether the "family member" in question is "parent" or "child."

In the interest of expediency and to minimize the research and/or duplication burden on your staff, please send records electronically if possible. If this is not possible, please notify me before sending to the mailing address listed below. If access to this request takes longer than twenty (20) business days, please let me know when I might receive records or be able to inspect the requested records. Please produce responsive documents as soon as they become available.
In all cases, please communicate with me at the email address below.

Please comply fully with 5 U.S.C. § 552 (b). Accordingly, without limitation to the foregoing, if any portion of this request is denied for any reason, please provide written notice of the records or portions of records that are being withheld and cite each specific exemption of the Freedom of Information Act on which the Agency relies. Moreover, to the extent the Agency believes that responsive records may be withheld in part, produce all reasonably segregable portions of those records.

Additionally, please provide all responsive documents even if they are redacted in full.

Fee Waiver Request

This request is primarily and fundamentally for non-commercial purposes. As a 501(c)(3) nonprofit, Heritage Foundation does not have a commercial purpose and the release of the information requested is not in Heritage Foundation's commercial interest. Heritage Foundation's mission is to is to formulate and promote public policies based on the principles of free enterprise, limited government, individual freedom, traditional American values, and a strong national defense. Heritage Foundation uses the information requested and analyzes it in order to educate the public through social media,[1] broadcast media[2] (traditional and nontraditional), and

[1] Heritage Foundation. [@ Heritage] (Accessed: 2022, February 18). 626.8K Followers Twitter. https://twitter.com/Heritage
[2] Fox News. (Accessed: 2022, February 18). Heritage Foundation launches Conservative Oversight

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press releases.[3] The requested information is in the public interest because the U.S. CPSC commissioner, Richard Trumka Jr., stated in an interview with Bloomberg, that CPSC is considering banning gas stoves amid health fears.[4] This announcement generated a great deal of national news coverage of this issue. *See* Appendix 1.

This is a request by a member of the news media for information of public interest, made in my capacity as an author for the Daily Signal[5] (a major news outlet[6]), I actively gather information of potential interest to our Daily Signal audience, I use my editorial skills to turn raw materials into a distinct work, and I distribute that work to our Daily Signal audience through podcasts[7] and articles. Therefore, I request that you waive all applicable fees associated with this request. If you deny this request for a fee waiver, please advise me in advance of the estimated charges if they are to exceed $50. Please send me a detailed and itemized explanation of those charges.

Expedited Processing is Warranted under 16 C.F.R. 1015.5

1. This provision provides that expedited processing shall be granted regarding: "With respect to a request made by a person primarily engaged in disseminating information, that there is an urgency to inform the public concerning actual or alleged Federal Government activity."

Even though expedited processing under 36 C.F.R. § 1250.28 (a)(4) hasn't been judicially construed under 16 C.F.R. 1015.5, courts have held that similar DOJ Regulation requires the requester to show: (1) that the request involves a "matter of widespread and exceptional media interest" (28 C.F.R. § 16.5(e)(1)(iv)); and (2) that the matter is one "in which there exists possible questions about the integrity of the government that affect public confidence" (*id.*). *See Edmonds v. FBI*, No. 02-cv-1294 (ESH), 2002 WL 32539613, *3 (D.D.C. Dec. 3, 2002). It is not necessary to show "prejudice or a matter of current exigency to the American public." *Id.*

First, the DOJ Regulation requires showing that the matter about which questions of integrity have been raised is the subject of widespread national media attention. *See Am. Oversight v. DOJ*, 292 F.Supp.3d 501, 507–508 (D.D.C. 2018)

Project aimed at 'exposing' Biden admin, leftist policies. https://www.foxnews.com/politics/heritage-conservative-oversight-project-biden-admin-leftist-policies

[3] Heritage Foundation. (Accessed: 2022, February 18). Press. https://www.heritage.org/press .

[4] Bloomberg. (Accessed: 2023, January 27). US Safety Agency to Consider Ban on Gas Stoves Amid Health Fears. https://www.bloomberg.com/news/articles/2023-01-09/us-safety-agency-to-consider-ban-on-gas-stoves-amid-health-fears?leadSource=uverify%20wall

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(denying motion for expedited processing because general media interest in Solicitor General's nomination is insufficient to show media interest in possible ethics questions concerning the nomination). There need not be a showing that the disclosure would shed considerable light on agency operations; only that there is "exceptional" and "widespread" media interest. *See CREW v. DOJ*, 870 F.Supp.2d 70, 81 (D.D.C. 2012), *rev'd on other grounds*, 746 F.3d 1082 (D.C. Cir. 2014). While the media interest need be "widespread" and "exceptional" it need not be overwhelming. *See ACLU*, 321 F.Supp.2d at 31–32 (rejecting DOJ's position that requester's citation to what the court described as "only a handful of articles" was insufficient to show "widespread and exceptional media interest" because those articles "were published in a variety of publications and repeatedly reference the ongoing national discussion about the Patriot Act and Section 215" (second quotation added)); *Edmonds*, 2002 WL 32539613, at *3 (numerous national newspaper and network television broadcasts concerning whistleblower's allegations of security lapses in FBI translator program met test).[5]

Second, the DOJ Regulation requires showing that "'there exists *possible* questions about the government's integrity that affect public confidence.'" *CREW v. DOJ*, 436 F.Supp.3d 354, 361 (D.D.C. 2020) (*quoting* 28 C.F.R. § 16.5(e)(1)(4)) (emphasis by Court).[6] It does not "require the requester to prove wrongdoing by the government in order to obtain documents on an expedited basis." *Id.* at 362. "The primary way to determine whether such possible questions exist is by examining the state of public coverage of the matter at issue, and whether that coverage surfaces possible ethics issues so potentially significant as to reduce public confidence in governmental institutions." *Am. Oversight v. DOJ*, 292 F.Supp.3d 501, 508 (D.D.C. 2018). This is not an extraordinarily high bar. *See, e.g.*, CREW, 436 F.Supp.3d at 361 (complaint sufficient to survive a motion to dismiss where it alleged Attorney General's action regarding disclosure of Mueller Report "supported an inference that at best, the Attorney General undertook to frame the public discussion on his own terms, and at worst that he distorted the truth"); *ACLU v. DOJ*, 321 F.Supp.2d 24, 32 (D.D.C. 2004) (allegations in press that Section 215 of the Patriot Act may be unconstitutional and reports that Members of Congress have alleged abuses of Section 215 "implicate[] government integrity" and hence are sufficient to meet test); *Edmonds*, 2002 WL 32539613, at *3–4 (test met where plaintiff alleged security

[5] *Cf.* 28 C.F.R. § 16.5(e)(3) ("The existence of numerous articles published on a given subject can be helpful in establishing the requirement that there be an 'urgency to inform' the public on the topic.").
[6] To be sure, this standard does not require expedition of any questions concerning government integrity. *See, e.g.*, *White v. DOJ*, 16 F.4th 539, 544 (7th Cir. 2021) (test not met in case where records sought to cast doubt on requestors' criminal conviction where requestor claimed he was subject to an elaborate government sting operation).

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lapses in FBI translators program, national news covered the issue, and two Senators expressed concern regarding "the significant security issues raised by plaintiff's allegations and the integrity of the FBI").[7]

2. The requested information is in the public interest because the U.S. CPSC commissioner, Richard Trumka Jr., stated in an interview with Bloomberg, that CPSC is considering banning gas stoves amid health fears.[8] As Attachment A (000002-000087) demonstrates, the press interest in these revelations is immense and on-going.

There has been Congressional interest in the stated comment. The Energy and Commerce Committee is conducting oversight and has transmitted oversight letters to the CSPC and the White House. *See* Appendix B. Senate Energy and Natural Resources Committee Chairman Joe Manchin announced Thursday that he is working with Sen. Ted Cruz to introduce bipartisan legislation to prohibit the Consumer Product Safety Commission from banning gas stoves.[9]

Press coverage and public Congressional oversight has repeatedly surfaced "possible questions about the integrity of the government that affect public confidence."

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[7] DOJ has granted expedition under the DOJ Regulation in a number of circumstances. *See, e.g.*, *CREW v. DOJ*, 870 F.Supp.2d at 81 n. 14 (expedition granted to request seeking records on FBI's closed investigation of Congressman DeLay for misconduct which did not result in charges, but received considerable media attention (subsequent history omitted)); *CREW v. DOJ*, 820 F.Supp.2d 39, 42, 46 (D.D.C. 2011) (expedition granted to request seeking information concerning possible deletion of Office of Legal Counsel emails where the possible deletion was flagged as a hindrance in an internal investigation, covered in the media, and was the subject of Congressional concerns); *Elec. Frontier Found. v. DOJ*, 565 F.Supp.2d 188, 189–91 (D.D.C. 2008) (expedition granted to request seeking information regarding storage of information obtained by National Security Letters in FBI's Data Warehouse); *CREW v. DOJ*, No. 05-cv-2078 (EGS), 2006 WL 1518964, *1 (D.D.C. June 1, 2006) (expedition granted to request concerning government's decision to seek a reduced penalty in tobacco litigation where government's decision was subject to intensive news coverage and prompted concern from "several Congressman" which caused a request for an Inspector General investigation of "improper political interference" with the decision).

[8] Bloomberg. (Accessed: 2023, January 27). US Safety Agency to Consider Ban on Gas Stoves Amid Health Fears. https://www.bloomberg.com/news/articles/2023-01-09/us-safety-agency-to-consider-ban-on-gas-stoves-amid-health-fears?leadSource=uverify%20wall

[9] Washington Examiner. (Accessed: 2023, February 2). Manchin teams up with Cruz on bill to block gas stove ban. https://www.washingtonexaminer.com/restoring-america/faith-freedom-self-reliance/manchin-cruz-bill-block-gas-stove-ban

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First. The proposed CPSC action to ban gas stoves has repeatedly been attacked as an extreme action undertaken without regards the CPSC's proper role and authority[10]. Indeed, many Members of Congress and members of the media have explicitly said the rule is motivated solely to advance and extreme climate agenda without regard to any applicable metrics. (Attachment A and B 000002-0000109)

Second. Many have raised serious concerns that a proposed ban would directly cause safety issues. (Attachment A 000002-000109)

Third. Many have argued that the proposed ban would potentially increase costs as it relates to energy(Attachment A 000002-000087). Some have suggested that this could have a more acute impact on certain disadvantaged segments of society

Thank you in advance for considering my request. If you have any questions, or feel you need clarification of this request please contact me at oversightproject@heritage.org.

Sincerely,

Mike Howell
Director of the Oversight Project and
Author at The Daily Signal
The Heritage Foundation
214 Massachusetts Ave, NE
Washington, D.C. 20002

[10]Daily Mail. (Accessed: 2023, February 3). Biden's Consumer Product Safety Commission WAS serious about banning gas stoves before public backlash forced a rethink - as it's claimed it would cost over $65BN to make every US household electric. https://www.dailymail.co.uk/news/article-11707473/Internal-memo-shows-Biden-banning-gas-stoves-public-backlash.html

From: Crockett, David
Sent: Wed, 10 Aug 2022 18:00:02 +0000
To: catherine.francois@mail.house.gov
Cc: CPSC Legislative Affairs
Subject: FW: Letter for Mr. Alexander Hoehn-Saric, Chairman, Consumer Product Safety Commission
Attachments: House Oversight Krishnamoorthi respsonse Gas Stoves 8.10.22 Final.pdf

Good afternoon,

Please find the attached correspondence and acknowledge receipt.

Thanks,
Dave

David Crockett
Congressional Liaison
U.S. Consumer Product Safety Commission | Office of Legislative Affairs
4330 East West Highway | Bethesda, MD 20814
Office: (301) 504-7489
dcrockett@cpsc.gov

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From: McGarvey, Carla <CMcGarvey@cpsc.gov>
Sent: Wednesday, August 10, 2022 1:12 PM
To: Mpanju, Melanie <Melanie.Mpanju@mail.house.gov>; CPSC Legislative Affairs <(b)(@cpsc.gov>
Cc: Misk, Jonathan <Jonathan.Misk@mail.house.gov>; Vruwink, Jonathan <Jonathan.Vruwink@mail.house.gov>; Baldwin, Wilson <Wilson.Baldwin@mail.house.gov>; Kilvington, John <John.Kilvington@mail.house.gov>; LaNier, Elisa <Elisa.LaNier@mail.house.gov>; Stratton, Amy <Amy.Stratton@mail.house.gov>; Solomon, Morgan <Morgan.Solomon@mail.house.gov>; Miller, Aidan <Aidan.Miller@mail.house.gov>; Stopek, Emma <Emma.Stopek@mail.house.gov>; Ginsburg, Andrew <AGinsburg@cpsc.gov>; Crockett, David <DCrockett@cpsc.gov>
Subject: RE: Letter for Mr. Alexander Hoehn-Saric, Chairman, Consumer Product Safety Commission

Hi All,

Please find attached to this email a response from CPSC Chair Hoehn-Saric to Chairman Krishnamoorthi to his August 1st letter on gas stoves.

Let me know if you have questions.

Thanks,
Carla

Carla McGarvey
Director, Office of Legislative Affairs
U.S. Consumer Product Safety Commission | Office of Legislative Affairs
4330 East West Highway | Bethesda, MD 20814
Mobile: (b)(6)
cmcgarvey@cpsc.gov

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From: Mpanju, Melanie <Melanie.Mpanju@mail.house.gov>
Sent: Monday, August 1, 2022 12:09 PM
To: McGarvey, Carla <CMcGarvey@cpsc.gov>; CPSC Legislative Affairs (b)(6)@cpsc.gov>
Cc: Misk, Jonathan <Jonathan.Misk@mail.house.gov>; Vruwink, Jonathan <Jonathan.Vruwink@mail.house.gov>; Baldwin, Wilson <Wilson.Baldwin@mail.house.gov>; Kilvington, John <John.Kilvington@mail.house.gov>; LaNier, Elisa <Elisa.LaNier@mail.house.gov>; Stratton, Amy <Amy.Stratton@mail.house.gov>; Solomon, Morgan <Morgan.Solomon@mail.house.gov>; Miller, Aidan <Aidan.Miller@mail.house.gov>; Stopek, Emma <Emma.Stopek@mail.house.gov>
Subject: Letter for Mr. Alexander Hoehn-Saric, Chairman, Consumer Product Safety Commission

Hello—

Please see the attached letter from Chairman Raja Krishnamoorthi, Subcommittee on Economic and Consumer Policy, for Mr. Alexander Hoehn-Saric, Chairman, Consumer Product Safety Commission.

Please acknowledge receipt of the letter. Thank you.

Best regards,
Melanie Mpanju
Staff Assistant | Committee on Oversight & Reform
Chairwoman Carolyn B. Maloney
melanie.mpanju@mail.house.gov

Email secured by Check Point

UNITED STATES
CONSUMER PRODUCT SAFETY COMMISSION
4330 EAST WEST HIGHWAY
BETHESDA, MD 20814

CHAIR ALEXANDER HOEHN-SARIC

August 10, 2022

The Honorable Raja Krishnamoorthi
Chairman
Subcommittee on Economic and Consumer Policy
Committee on Oversight and Reform
2157 Rayburn House Office Building
U.S. House of Representatives
Washington, D.C. 20515

Dear Chairman Krishnamoorthi:

Thank you for your letter dated August 1, 2022, asking several questions, and seeking a number of documents concerning gas stoves. This letter responds to your initial set of questions.

The safety of consumers in their homes is a vital part of CPSC's mission, which has led to stove safety being an area of concern for the agency for many years. Based on fatality data, one of the leading causes of death associated with consumer products in the home is fire, which claims an average of 2,379 lives annually. The leading cause of home fires is cooking, generally involving stoves. More specifically, based on the most recent available data, fire deaths associated with stoves totaled 480 during the period 2017 through 2019. A closer analysis of that data indicates that approximately 85 percent of those deaths were associated with electric stoves, and 15 percent were associated with gas stoves, despite gas stoves representing 40 percent of the market. To mitigate these risks, CPSC has worked with voluntary standards organizations to address ignitions from electric coil ranges, and staff has addressed smoke alarm standards to ensure that smoke alarms sound when a fire begins, without nuisance alarms; staff has also conducted related recalls, and we have pursued information and education campaigns about these acute risks.

As your letter suggests, carbon monoxide (CO) deaths and injuries remain a significant problem as well. According to a recent CPSC report, there have been over 500 fatalities from 2016-2018 due to CO poisonings associated with consumer products. CPSC is currently tackling the risks of injury or death from acute poisoning due to carbon monoxide emissions from furnaces, generators, and other products through rulemakings, voluntary standards work, and consumer education.

CPSC has undertaken work regarding chronic hazards targeted at addressing health risks associated with indoor air quality (IAQ), including chronic hazards associated with gas stoves, but the work has been limited due in large part to funding constraints. Significant resources are necessary to conduct the research required to determine the levels of exposure from identified consumer products that present measurable harms to human health. Where Congress has set strict statutory limits on the presence of substances such as lead and phthalates, CPSC has been able to move quickly and effectively to enforce those provisions and limit use in consumer products. But where CPSC must make the scientific findings needed to begin and sustain a rulemaking that produces a standard that is defensible in court, a significant research investment is necessary.

Responses to the specific questions raised in your letter, which are repeated in bold, are included below.

1) **Has the Commission considered issuing mandatory or recommending voluntary standards or warning labels to address the health risks of indoor air pollution from gas stoves? If it has, please explain the status of the Commission's work and explain why the Commission has not yet issued any such mandatory—or recommended any such voluntary—standards or warning labels? If it has not, will the Commission consider taking such action based on the publicly available evidence of health harms from gas stoves?**

   In recognition of the importance of indoor air emissions and their impact on consumer health, CPSC staff is working on scoping voluntary standards to address the potential health risks of indoor air emissions from gas stoves creating unreasonable risks of injury to consumers. Based on Commission direction in its FY 2022 Operating Plan,[1] this year CPSC staff has been participating in voluntary standards activity for indoor air quality and for gas

[1] FY 2022 Operating Plan

ranges, which include staff participation in various ASTM and American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) work groups and subcommittees[2]. Over the course of FY 2022, CPSC staff has begun to consider how to address emissions from gas appliances, including CPSC staff acting as Chair of the ANSI/CSA voluntary standard technical subcommittee on gas ranges. This work is described in the FY 2022 Mid-Year Voluntary Standards Activity Report[3]. As part of this effort, work has begun on areas including: developing an inventory of existing voluntary standards related to indoor air quality and gas range stoves; compiling acute and chronic toxicity reference values for carbon monoxide, nitrogen dioxide, and particulate matter; and describing the relationship of emissions from gas range stoves and indoor air concentrations. Additional technical work and data analysis are necessary before staff can make conclusions regarding this research.

Relatedly, CPSC recently proposed investments to conduct additional research of the potential hazards associated with gas ranges and unvented gas fireplaces. CPSC had planned a significant project on Healthy Indoor Environment in FY 2022, as described in the agency's Performance Budget Request[4] to Congress and in the Commission-approved FY 2022 Operating Plan[5]. Unfortunately, CPSC's appropriated levels for FY 2022, which were not finalized until the FY 2022 Omnibus passed in March 2022, were significantly lower than both the Performance Budget Request, as well as the House-passed appropriations bill. Therefore, CPSC did not have the funds available to take on this research project. Consequently, CPSC has not been able to move forward in developing the findings that would be necessary to support a mandatory standard.

In the past, CPSC has studied or contributed to voluntary standards for many residential combustion appliances, such as furnaces, unvented gas space heaters, kerosene space heaters, portable generators, wood stoves, and camping heaters, given clear hazards within the Commission's jurisdiction, including death, that these products pose. CPSC plans to continue its involvement in the voluntary standards activity that began in FY 2022 for indoor air quality and gas ranges.

---

[2] https://cpsc.gov/s3fs-public/Volunary-Standards-Activities-Fiscal-Year-2022-Midyear-Report.pdf?VersionId=bYWjN1aNfD7GmmJ5BSWa0IENK6D_ustI

[3] *Id.*

[4] FY 2022 Performance Budget Request

[5] FY 2022 Operating Plan | CPSC.gov

2) **Please describe the Commission's plans, if any, to issue mandatory standards or to facilitate the adoption of voluntary standards addressing indoor air pollution from gas stoves.**

For the Commission to issue mandatory standards, the agency must follow its statutory authorities. Two statutes that CPSC administers, the Consumer Product Safety Act (CPSA) and the Federal Hazardous Substances Act (FHSA), are particularly relevant to a potential mandatory standard addressing hazards associated with gas stoves.

Under sections 7 and 9 of the CPSA, the Commission has the authority to issue consumer product safety standards consisting of performance requirements and warnings or instructional requirements, if the Commission finds that the requirements of the standard are "reasonably necessary to prevent or reduce an unreasonable risk of injury associated with the consumer product." The regulatory analysis required to issue a mandatory standard under the CPSA includes an assessment of the potential benefits and costs of the rule; reasons a voluntary standard submitted to the Commission was not published as part of the rule; and evaluation of alternatives to the rule. Additional findings to issue a mandatory standard under sections 7 and 9 of the CPSA include an assessment of the degree and nature of the risk intended to be addressed by the rule; an approximate number of products that are subject to the rule; the need of the public and effect of the rule on utility, cost, and availability of the product; and other means of achieving the objective of the rule while minimizing adverse effects on competition, manufacturing, and commercial practices. The rule must be in the public interest, the expected benefits of the rule must bear a reasonable relationship to the costs, and the rule must impose the least burdensome requirement that prevents or adequately reduces the risk of injury.

The FHSA addresses household articles that are or contain "hazardous substances."15 U.S.C. §§ 1261–1278. The FHSA defines a "hazardous substance" as a substance or mixture that (*i*) is toxic, (*ii*) is corrosive, (*iii*) is an irritant, (*iv*) is a strong sensitizer, (*v*) is flammable or combustible, or (*vi*) generates pressure through decomposition, heat, or other means, if the substance "may cause substantial personal injury or substantial illness during or as a proximate result of any customary or reasonably foreseeable handling or use, including reasonably foreseeable ingestion by children."

A product that is intended or packaged in a form suitable for household use and meets the definition of "hazardous substance" must be appropriately labeled to warn consumers of relevant hazards or the item is considered a "misbranded hazardous substance." Manufacturers whose household articles meet the definition of a hazardous substance are responsible for appropriately labeling their products. The Commission may also issue a rule declaring a substance to be a hazardous substance in order to avoid or resolve uncertainty.

The FHSA provides the Commission with the authority to ban a hazardous substance if the Commission determines that, notwithstanding the cautionary labeling required for hazardous substances, the degree or nature of the hazard involved is such that the objective of the protection of public health and safety can be adequately served only by keeping such substance out of the channels of interstate commerce. Issuing a rule to ban a hazardous substance requires findings similar to those under section 7 and 9 of the CPSA, such as a regulatory analysis; a determination that the expected benefits from the rule bear a reasonable relationship to the costs; a determination that the rule imposes the least burdensome requirement that prevents or adequately reduces the risk of injury. If the risk at issue with the household substance or article is cancer, birth defects, or gene mutations, the Commission must convene a chronic hazard advisory panel (CHAP) prior to beginning a rulemaking.

Under both the CPSA and the FHSA, moreover, in order to promulgate a final rule, the Commission must determine that any voluntary standard is unlikely to result in the elimination or adequate reduction of the risk of injury, or that substantial compliance with the voluntary standard is unlikely.

CPSC has not yet determined what voluntary standards may be within its jurisdiction and appropriate for gas stoves. At this time, CPSC does not have sufficient information to develop the scientific and economic findings necessary to support a mandatory standard or to recommend a specific course of action in a voluntary standard, although collaborations as described in response to the preceding question are underway. Next steps would include CPSC undertaking a project, such as the Healthy Indoor Environment project, which was proposed for FY 22, but did not receive funding.

Within the limited appropriated funds available for chronic hazard research, CPSC staff have been working to scope and begin development of future

voluntary standards for gas range stoves. A potential voluntary standard to measure emissions from gas stoves and relate those emissions to indoor air concentrations has resulted in discussion of detailed technical topics, such as the amount of fuel used per test for scalability, appropriate test chambers, types of sampling equipment, and appropriate analytical methods. Additional work is required and is ongoing, despite limited resources under current appropriations to advance the project.

CPSC staff is working with EPA staff and other stakeholders, with ASTM, a standards development organization (SDO), and with ASHRAE to develop and review more broadly voluntary standards related to IAQ, indoor air emissions and consumer products. Staff is also working with many stakeholders, including CSA (another SDO), to develop gas range standards. These efforts include staff research on gas range emissions, subject to available resources. Participation in a voluntary standards process is open to consumers and the public in addition to CPSC staff and stakeholders.

3) **Please describe the Commission's plans, if any, to require a mandatory warning label or facilitate the adoption of voluntary warning labels addressing the health risks of indoor air pollution from gas stoves.**

See response 2.

4) **Please describe the Commission's plans, if any, to publish public educational materials specifically focused on the significant health risks posed by gas stove emissions.**

Since the early 1990s, CPSC has worked with other federal agencies like EPA, state health departments, and the American Lung Association to issue warnings and release informational booklets to the public and physicians to raise awareness on the risk of nitrogen dioxide, carbon monoxide, and other toxic gases in the indoor environment. Staff has published informational material about the potential hazards of gas ranges, including "The Inside Story: A Guide to indoor Air Quality."[6] Staff will produce additional materials for the public to share, as new information and new standards are developed.

[6] Available at: https://www.cpsc.gov/Safety-Education/Safety-Guides/Home/The-Inside-Story-A-Guide-to-Indoor-Air-Quality.

5) **Are there any legislative or other measures that the Commission believes are necessary for it to issue regulations concerning indoor gas stove emissions?**

If Congress were to set a standard for permissible levels of gas stove emissions similar to what it did for lead and phthalates and give the agency rulemaking authority consistent with the Administrative Procedure Act to implement these levels, CPSC would act quickly to ensure compliance with the law. As discussed above, by contrast, regulating indoor gas stove emissions with respect to risk of injury under CPSC's existing authorities would require significant resources and time.

CPSC's FY 2023 Performance Budget Request includes a request for additional staff for chronic hazard analysis and funding at a level of $195.5 million to address otherwise unfunded priorities, including chronic hazards. CPSC requires consistent funding that is sufficient to perform the research on a range of healthy indoor environmental and other chronic hazard issues. Chronic hazards are often complex, with work required to establish exposure, bioavailability, and toxicity, all requiring extensive research often conducted over many years to review existing data and collect missing data.

6) **Are the following substances toxic: (i) nitrogen dioxide; (ii) benzene; (iii) toluene; (iv) heptane; (v) hexane; and (vi) cyclohexane?**

These substances likely meet the definition of "toxic" as defined in the FHSA and its implementing regulations. The FHSA concerns household articles that are or contain "hazardous substances." Under the FHSA, a "toxic substance" is a "hazardous substance" if it may cause substantial personal injury or substantial illness as a result of customary or reasonably foreseeable handling or use. This assessment of the potential to cause substantial personal injury or illness involves consideration of the intensity, frequency, and duration of human exposure to the toxic substance at issue and any resulting adverse health effects from the exposure. CPSC lacks data on the intensity, extent or duration of exposure to these chemicals for users of gas stoves or household members possibly exposed to varied levels of these chemicals in other locations in the home. Before the CPSC could regulate household articles such as stoves that contain or emit the gas or the chemical as "hazardous substances," the Commission would need to make findings as to the toxicity (or other enumerated characteristic in the statute) and whether customary or

reasonably foreseeable handling or use of the substance has the capacity to cause substantial injury or illness.

As requested in your letter, CPSC is working to provide requested documents at a future date. Should you or your staff have any additional questions, please do not hesitate to contact me, or Carla McGarvey, Director of Legislative Affairs, at cmcgarvey@cpsc.gov or 240-997-8580.

Sincerely,



Alexander Hoehn-Saric
Chair

cc: The Honorable Michael Cloud, Ranking Member, Subcommittee on Economic and Consumer Policy

From: Laitin, Anna
Sent: Wed, 21 Dec 2022 21:24:50 +0000
To: Ray, DeWane
Cc: Levine, Jason; Ginsburg, Andrew; Crockett, David
Subject: FW: Letter to CPSC re: indoor air pollution from gas stoves
Attachments: Letter to CPSC on gas stoves Dec 2022.pdf

Hey DeWane –

This just came through to OLA. It doesn't ask for documents or a briefing. We will learn more about what they actually want in the new year and will follow up.

Anna

From: CPSC Legislative Affairs <(b)(6)@cpsc.gov>
Sent: Wednesday, December 21, 2022 3:38 PM
To: Laitin, Anna <ALaitin@cpsc.gov>; Ginsburg, Andrew <AGinsburg@cpsc.gov>
Subject: FW: Letter to CPSC re: indoor air pollution from gas stoves

Please see the attached.

From: Fish, Carina (Booker) <Carina_Fish@booker.senate.gov>
Sent: Wednesday, December 21, 2022 3:28 PM
To: CPSC Legislative Affairs(b)(6)@cpsc.gov>
Cc: Schisler, Kate <Kate.Schisler@mail.house.gov>; Spawn, Ariana (Booker) <Ariana_Spawn@booker.senate.gov>
Subject: Letter to CPSC re: indoor air pollution from gas stoves

Hi Carla,

I hope you're well. Please find attached a letter from Senator Booker, Representative Beyer, and colleagues to the CPSC urging the Commission to address indoor air pollution generated by gas stoves. Thanks in advance for routing this letter appropriately.

Happy holidays,

Carina

Carina Fish, Ph.D. | she/her
NOAA Sea Grant Knauss Fellow
U.S. Senator Cory A. Booker
717 Hart Senate Office
+1.202.228.1327

Email secured by Check Point

Congress of the United States

Washington, DC 20510

December 21, 2022

Alexander Hoehn-Saric
Chair
U.S. Consumer Product Safety Commission
4330 East West Highway
Bethesda, MD 20814

Dear Chairman Hoehn-Saric:

We write to express our concern regarding the risks posed to consumers from indoor air pollution generated by gas stoves, and to encourage the Consumer Product Safety Commission (CPSC) to take action to address these risks.

Over a third of American households—more than 40 million homes—use a gas stove for cooking. Gas stoves emit high levels of pollutants such as nitrogen dioxide (NO_2), carbon monoxide (CO), and fine particulate matter ($PM_{2.5}$). A range of studies have shown that, when used without adequate ventilation, cooking with a gas stove can raise indoor concentrations of these pollutants to levels that the Environmental Protection Agency considers to be unsafe even outdoors. Further, methane leaks from gas stoves inside U.S. homes were recently found to contribute the equivalent climate impacts as about 500,000 gasoline-powered cars and, importantly, more than three-quarters of the leaks occurred while the appliance was not in use.

In addition to the climate impacts, these emissions represent real health risks to millions of Americans. Both NO_2 and $PM_{2.5}$ are known to exacerbate respiratory conditions. Short-term exposure to NO_2 is linked to worsening asthma in children, and long-term exposure has been determined to likely cause the development of asthma. Both pollutants have also been linked to cardiovascular impacts and a range of other adverse health issues. Even low-level CO exposure can exacerbate cardiovascular illness among people with coronary heart disease and other vulnerable populations.

These emissions can create a cumulative burden to households that are already more likely to face higher exposure to both indoor and outdoor air pollution. Statistics show that Black, Latino, and low-income households are more likely to experience disproportionate air pollution, either from being more likely to be located near a waste incinerator or coal ash site, or living in smaller homes with poor ventilation, malfunctioning appliances, mold, dust mites, secondhand smoke, lead dust, pests, and other maintenance deficiencies.

As you know, the CPSC has broad authority under the Consumer Product Safety Act to regulate consumer products that pose an unreasonable risk of injury. We urge the Commission to protect consumers from these harmful emissions. The Commission itself expressed concerns over gas

stove emissions in 1985. Since then, evidence for the danger of these emissions has only increased.

We understand that the CPSC adopted the following provision to this year's operating plan:

> "Chronic Hazards: Rulemaking Activities: By March 1, 2023, staff will prepare and submit to the Commission a Request for Information (RFI) to seek public input on hazards associated with gas stoves and proposed solutions to those hazards."

We are encouraged by this addition to the operating plan and urge the CPSC to move quickly on this RFI and use the responses to swiftly initiate impactful rulemaking. As part of this process, we ask the CPSC to explicitly evaluate the disparate health outcomes that occur from the coupling of gas stoves with the material realities to which the most vulnerable Americans are subjected, as well as evaluate the health impacts of gas leaks due to gas stoves connections. Robust consideration of the distributional effects of regulatory action is a long-recognized best practice for agency decision making that is consistent with Executive Orders and guidance from the Office of Management & Budget.

We also request that the CPSC consider taking the following actions to help protect Americans from the hazards of gas stove emissions:

- Require gas stoves to be sold with range hoods that meet mandatory performance standards, assessing their efficiency of removing the pollutants,
- Where feasible, issue mandatory performance standards for gas stoves that address steady-state-off leakage, including requiring automatic shut-off valves,
- Where feasible, issue mandatory performance standards for gas stoves that address the health impacts of hazardous emissions,
- Require labels on gas stoves that educate consumers about their exposure risks,
- Launch a public education campaign on the health risks of cooking with a gas stove, and steps that consumers can take to minimize their risk.

Thank you for your attention on this matter. We look forward to supporting the Commission in your efforts to address this consumer product safety and critical health issue.

Sincerely,

Cory A. Booker
United States Senator

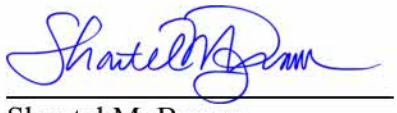
Donald S. Beyer Jr.
Member of Congress



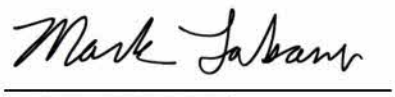
Jared Huffman
Member of Congress



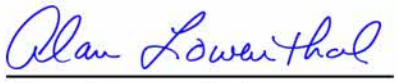
Dianne Feinstein
United States Senator



Shontel M. Brown
Member of Congress



MARK TAKANO
Member of Congress



Alan S. Lowenthal
Member of Congress
Chair, Subcommittee on
Energy and Mineral
Resources



Ted W. Lieu
Member of Congress



André Carson
Member of Congress



Martin Heinrich
United States Senator



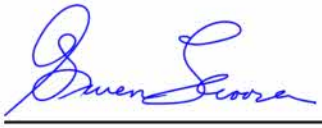
Barbara Lee
Member of Congress



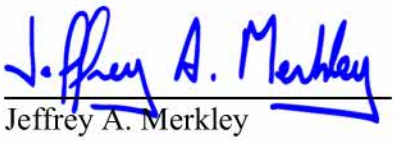
Eleanor Holmes Norton
Member of Congress



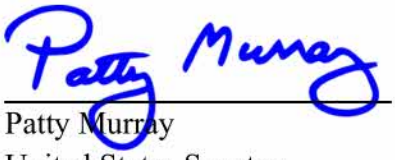
Edward J. Markey
United States Senator



Gwen S. Moore
Member of Congress



Jeffrey A. Merkley
United States Senator



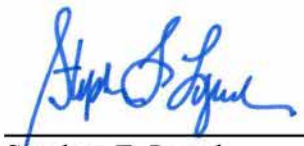
Patty Murray
United States Senator



Elizabeth Warren
United States Senator



Bernard Sanders
United States Senator



Stephen F. Lynch
Member of Congress



Katie Porter
Member of Congress

From: Crockett, David
Sent: Thu, 4 Aug 2022 20:28:22 +0000
To: McGarvey, Carla
Subject: RE: Gas stove questions
Attachments: Krishnamoorthi responses.docx

Oh, didn't know you wanted the whole format. See attached.

From: McGarvey, Carla <CMcGarvey@cpsc.gov>
Sent: Thursday, August 4, 2022 4:03 PM
To: Crockett, David <DCrockett@cpsc.gov>
Subject: RE: Gas stove questions

Thanks. Could you make it like a letter so with the address line etc?

From: Crockett, David <DCrockett@cpsc.gov>
Sent: Thursday, August 4, 2022 4:01 PM
To: McGarvey, Carla <CMcGarvey@cpsc.gov>
Subject: Gas stove questions

Hi Carla – Please see the attached.

David Crockett
Congressional Liaison
U.S. Consumer Product Safety Commission | Office of Legislative Affairs
4330 East West Highway | Bethesda, MD 20814
Office: (301) 504-7489
dcrockett@cpsc.gov

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UNITED STATES
CONSUMER PRODUCT SAFETY COMMISSION
4330 EAST WEST HIGHWAY
BETHESDA, MD 20814

CHAIR ALEXANDER HOEHN-SARIC

August X, 2022

The Honorable Raja Krishnamoorthi
Chairman
Subcommittee on Economic and Consumer Policy
Committee on Oversight and Reform
2157 Rayburn House Office Building
U.S. House of Representatives
Washington, D.C. 20515

Dear Chairman Krishnamoorthi:

Your questions are repeated below in bold, followed by our responses.

1) **Has the Commission considered issuing mandatory or recommending voluntary standards or warning labels to address the health risks of indoor air pollution from gas stoves? If it has, please explain the status of the Commission's work and explain why the Commission has not yet issued any such mandatory—or recommended any such voluntary—standards or warning labels? If it has not, will the Commission consider taking such action based on the publicly available evidence of health harms from gas stoves?**

   Insert answers here.

2) **Please describe the Commission's plans, if any, to issue mandatory standards or to facilitate the adoption of voluntary standards addressing indoor air pollution from gas stoves.**

   Insert answer here.

3) **Please describe the Commission's plans, if any, to require a mandatory warning label or facilitate the adoption of voluntary warning labels addressing the health risks of indoor air pollution from gas stoves.**

   Insert answer here.

4) **Please describe the Commission's plans, if any, to publish public educational materials specifically focused on the significant health risks posed by gas stove emissions.**

   Insert answer here

5) **Are there any legislative or other measures that the Commission believes are necessary for it to issue regulations concerning indoor gas stove emissions?**

   Insert answer here.

6) **Are the following substances toxic: (i) nitrogen dioxide; (ii) benzene; (iii)toluene; (iv) heptane; (v) hexane; and (vi) cyclohexane?**

   Insert answers here.

Sincerely yours,

Alexander Hoehn-Saric
Chair

Enclosure

cc: The Honorable Michael Cloud, Ranking Member, Subcommittee on Economic and Consumer Policy

From: McGarvey, Carla
Sent: Mon, 1 Aug 2022 16:16:23 +0000
To: Mpanju, Melanie; CPSC Legislative Affairs
Cc: Misk, Jonathan; Vruwink, Jonathan; Baldwin, Wilson; Kilvington, John; LaNier, Elisa; Stratton, Amy; Solomon, Morgan; Miller, Aidan; Stopek, Emma; Ginsburg, Andrew; Crockett, David
Subject: RE: Letter for Mr. Alexander Hoehn-Saric, Chairman, Consumer Product Safety Commission

Hi All,

Confirming that we have received the letter.

Thanks,
Carla

Carla McGarvey
Director, Office of Legislative Affairs
U.S. Consumer Product Safety Commission | Office of Legislative Affairs
4330 East West Highway | Bethesda, MD 20814
Mobile: (b)(6)
cmcgarvey@cpsc.gov

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From: Mpanju, Melanie <Melanie.Mpanju@mail.house.gov>
Sent: Monday, August 1, 2022 12:09 PM
To: McGarvey, Carla <CMcGarvey@cpsc.gov>; CPSC Legislative Affairs (b)(6)@cpsc.gov>
Cc: Misk, Jonathan <Jonathan.Misk@mail.house.gov>; Vruwink, Jonathan <Jonathan.Vruwink@mail.house.gov>; Baldwin, Wilson <Wilson.Baldwin@mail.house.gov>; Kilvington, John <John.Kilvington@mail.house.gov>; LaNier, Elisa <Elisa.LaNier@mail.house.gov>; Stratton, Amy <Amy.Stratton@mail.house.gov>; Solomon, Morgan <Morgan.Solomon@mail.house.gov>; Miller, Aidan <Aidan.Miller@mail.house.gov>; Stopek, Emma <Emma.Stopek@mail.house.gov>
Subject: Letter for Mr. Alexander Hoehn-Saric, Chairman, Consumer Product Safety Commission

Hello—

Please see the attached letter from Chairman Raja Krishnamoorthi, Subcommittee on Economic and Consumer Policy, for Mr. Alexander Hoehn-Saric, Chairman, Consumer Product Safety Commission.

Please acknowledge receipt of the letter. Thank you.

Best regards,
Melanie Mpanju

Staff Assistant | Committee on Oversight & Reform
Chairwoman Carolyn B. Maloney
melanie.mpanju@mail.house.gov

Email secured by Check Point

From: Hampshire, Melissa
Sent: Wed, 17 Aug 2022 14:13:38 +0000
To: McGarvey, Carla
Cc: Ginsburg, Andrew; Crockett, David; Austin Schlick
Subject: RE: Update on Document Production Request (CONGRESSIONAL MALONEY/Krishnamoorthi)
Attachments: (FOIA) RE: Letter for Mr. Alexander Hoehn-Saric, Chairman, Consumer Product Safety Commission

Good morning – (b)(5)

(b)(5)

Please let me know immediately if you have any questions or concerns. Thank you!

From: McGarvey, Carla <CMcGarvey@cpsc.gov>
Sent: Monday, August 15, 2022 3:57 PM
To: Hampshire, Melissa <MHampshire@cpsc.gov>
Cc: Ginsburg, Andrew <AGinsburg@cpsc.gov>; Crockett, David <DCrockett@cpsc.gov>
Subject: RE: Update on Document Production Request (CONGRESSIONAL MALONEY)

(b)(5)

Thanks!

From: Hampshire, Melissa <MHampshire@cpsc.gov>
Sent: Monday, August 15, 2022 11:05 AM

To: McGarvey, Carla <CMcGarvey@cpsc.gov>
Subject: RE: Update on Document Production Request (CONGRESSIONAL MALONEY)

Hi Carla – (b)(5)

(b)(5)

From: McGarvey, Carla <CMcGarvey@cpsc.gov>
Sent: Monday, August 15, 2022 10:26 AM
To: Hampshire, Melissa <MHampshire@cpsc.gov>
Cc: Mosheim, Abioye <AMosheim@cpsc.gov>
Subject: RE: Update on Document Production Request (CONGRESSIONAL MALONEY)

Ok, Abi can you give me a call? I think I just got the FOIA request that Melissa mentioned.

(b)(5)

Thanks!

From: Hampshire, Melissa <MHampshire@cpsc.gov>
Sent: Monday, August 15, 2022 10:12 AM
To: McGarvey, Carla <CMcGarvey@cpsc.gov>
Cc: Mosheim, Abioye <AMosheim@cpsc.gov>
Subject: RE: Update on Document Production Request (CONGRESSIONAL MALONEY)

Hi Carla – the FOIA is not my issue its Abi's. Can you touch base with her? Thanks

---

From: McGarvey, Carla <CMcGarvey@cpsc.gov>
Sent: Monday, August 15, 2022 10:07 AM
To: Hampshire, Melissa <MHampshire@cpsc.gov>
Subject: RE: Update on Document Production Request (CONGRESSIONAL MALONEY)

Hi Melissa,

Can you give me a call when you have a moment—either on my cell (below) or on Webex?

(b)(5)

Thanks!
Carla

Carla McGarvey

Director, Office of Legislative Affairs
U.S. Consumer Product Safety Commission | Office of Legislative Affairs
4330 East West Highway | Bethesda, MD 20814
Mobile: (b)(6)
cmcgarvey@cpsc.gov

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From: Hampshire, Melissa <MHampshire@cpsc.gov>
Sent: Monday, August 15, 2022 9:14 AM
To: McGarvey, Carla <CMcGarvey@cpsc.gov>; Ginsburg, Andrew <AGinsburg@cpsc.gov>; Recht, Joel <jrecht@cpsc.gov>
Cc: Boniface, (b)(6) Boniface@cpsc.gov>; Crockett, David <DCrockett@cpsc.gov>; Austin Schlick (b)(6) @cpsc.gov>; Mosheim, Abioye <AMosheim@cpsc.gov>
Subject: RE: Update on Document Production Request (CONGRESSIONAL MALONEY)

Abi – meant to copy you. Thanks

From: Hampshire, Melissa
Sent: Monday, August 15, 2022 9:05 AM
To: McGarvey, Carla <CMcGarvey@cpsc.gov>; Ginsburg, Andrew <AGinsburg@cpsc.gov>; Recht, Joel <jrecht@cpsc.gov>
Cc: Boniface (b)(6) Boniface@cpsc.gov>; Crockett, David <DCrockett@cpsc.gov>; Austin Schlick (b)(6) @cpsc.gov>
Subject: RE: Update on Document Production Request (CONGRESSIONAL MALONEY)

(b)(5)

From: McGarvey, Carla <CMcGarvey@cpsc.gov>
Sent: Thursday, August 11, 2022 3:36 PM
To: Hampshire, Melissa <MHampshire@cpsc.gov>; Ginsburg, Andrew <AGinsburg@cpsc.gov>; Recht, Joel <jrecht@cpsc.gov>
Cc: Boniface, (b)(6) Boniface@cpsc.gov>; Crockett, David <DCrockett@cpsc.gov>
Subject: Re: Letter for Mr. Alexander Hoehn-Saric, Chairman, Consumer Product Safety Commission

Hi All,

(b)(5)

Thanks
Carla

Get Outlook for iOS

From: Hampshire, Melissa <MHampshire@cpsc.gov>
Sent: Thursday, August 11, 2022 2:38:09 PM
To: Ginsburg, Andrew <AGinsburg@cpsc.gov>; Recht, Joel <jrecht@cpsc.gov>
Cc: Boniface(b)(6) Boniface@cpsc.gov>; McGarvey, Carla <CMcGarvey@cpsc.gov>; Crockett, David <DCrockett@cpsc.gov>
Subject: RE: Letter for Mr. Alexander Hoehn-Saric, Chairman, Consumer Product Safety Commission

Thanks Andrew – (b)(5)

(b)(5)

From: Ginsburg, Andrew <AGinsburg@cpsc.gov>
Sent: Thursday, August 11, 2022 2:33 PM
To: Recht, Joel <jrecht@cpsc.gov>; Hampshire, Melissa <MHampshire@cpsc.gov>
Cc: Boniface(b)(6) Boniface@cpsc.gov>; McGarvey, Carla <CMcGarvey@cpsc.gov>; Crockett, David <DCrockett@cpsc.gov>; Mosheim, Abioye <AMosheim@cpsc.gov>
Subject: RE: Letter for Mr. Alexander Hoehn-Saric, Chairman, Consumer Product Safety Commission

I would defer to Carla. She can assess the document production and decide how to proceed when she returns.

Andrew Ginsburg
Deputy Director
U.S. Consumer Product Safety Commission | Office of Legislative Affairs
4330 East-West Highway | Bethesda, MD 20814
Mobile: (b)(6)

Follow Us: Facebook, Twitter, Instagram, YouTube

From: Recht, Joel <jrecht@cpsc.gov>
Sent: Thursday, August 11, 2022 12:56 PM
To: Hampshire, Melissa <MHampshire@cpsc.gov>
Cc: Boniface(b)(6) Boniface@cpsc.gov>; McGarvey, Carla <CMcGarvey@cpsc.gov>; Ginsburg, Andrew <AGinsburg@cpsc.gov>; Crockett, David <DCrockett@cpsc.gov>; Mosheim, Abioye <AMosheim@cpsc.gov>
Subject: RE: Letter for Mr. Alexander Hoehn-Saric, Chairman, Consumer Product Safety Commission

I saw that, which is why I copied Andrew and Dave. Hopefully they can address for OLA.

From: Hampshire, Melissa <MHampshire@cpsc.gov>
Sent: Thursday, August 11, 2022 12:55 PM
To: Recht, Joel <jrecht@cpsc.gov>
Cc: Boniface(b)(6) Boniface@cpsc.gov>; McGarvey, Carla <CMcGarvey@cpsc.gov>; Ginsburg, Andrew <AGinsburg@cpsc.gov>; Crockett, David <DCrockett@cpsc.gov>; Mosheim, Abioye <AMosheim@cpsc.gov>
Subject: RE: Letter for Mr. Alexander Hoehn-Saric, Chairman, Consumer Product Safety Commission

Carla is out till Monday 8/15 according to the email.

From: Recht, Joel <jrecht@cpsc.gov>
Sent: Thursday, August 11, 2022 12:55 PM
To: Hampshire, Melissa <MHampshire@cpsc.gov>
Cc: Boniface(b)(6) Boniface@cpsc.gov>; McGarvey, Carla <CMcGarvey@cpsc.gov>; Ginsburg, Andrew <AGinsburg@cpsc.gov>; Crockett, David <DCrockett@cpsc.gov>; Mosheim, Abioye <AMosheim@cpsc.gov>
Subject: RE: Letter for Mr. Alexander Hoehn-Saric, Chairman, Consumer Product Safety Commission

(b)(5)

From: Hampshire, Melissa <MHampshire@cpsc.gov>
Sent: Thursday, August 11, 2022 12:46 PM
To: Recht, Joel <jrecht@cpsc.gov>
Cc: Boniface(b)(6) Boniface@cpsc.gov>; McGarvey, Carla <CMcGarvey@cpsc.gov>; Ginsburg, Andrew <AGinsburg@cpsc.gov>; Crockett, David <DCrockett@cpsc.gov>; Mosheim, Abioye <AMosheim@cpsc.gov>
Subject: RE: Letter for Mr. Alexander Hoehn-Saric, Chairman, Consumer Product Safety Commission

Thanks Joel – (b)(5)
(b)(5)

From: Recht, Joel <jrecht@cpsc.gov>
Sent: Thursday, August 11, 2022 12:37 PM
To: Hampshire, Melissa <MHampshire@cpsc.gov>; Mosheim, Abioye <AMosheim@cpsc.gov>
Cc: Boniface(b)(6) Boniface@cpsc.gov>; McGarvey, Carla <CMcGarvey@cpsc.gov>; Ginsburg, Andrew <AGinsburg@cpsc.gov>; Crockett, David <DCrockett@cpsc.gov>
Subject: RE: Letter for Mr. Alexander Hoehn-Saric, Chairman, Consumer Product Safety Commission

Melissa and Abi –

(b)(5)

Thanks.

Joel

From: Mosheim, Abioye
Sent: Tue, 16 Aug 2022 21:11:58 +0000
To: Recht, Joel; Hampshire, Melissa
Cc: Boniface, Duane; McGarvey, Carla; Ginsburg, Andrew; Crockett, David
Subject: (FOIA) RE: Letter for Mr. Alexander Hoehn-Saric, Chairman, Consumer Product Safety Commission
Attachments: RE: (Urgent) 2022-08-01.RK to Hoehn-Saric-CPSC re Gas Stoves.pdf
Importance: High

Angie was able to find the attached documents as possibly responsive. Please let me know if you need anything else on this. Thanks.

Abioye E. Mosheim
Assistant General Counsel, Division of Information Access
U.S. Consumer Product Safety Commission | Office of the General Counsel
4330 East West Highway | Bethesda, MD 20814
Office: (301) 504-7454 | **Mobile:** (b)(6)

Follow Us: Facebook, Twitter, Instagram, YouTube

From: Recht, Joel <jrecht@cpsc.gov>
Sent: Thursday, August 11, 2022 12:56 PM
To: Hampshire, Melissa <MHampshire@cpsc.gov>
Cc: Boniface(b)(6) Boniface@cpsc.gov>; McGarvey, Carla <CMcGarvey@cpsc.gov>; Ginsburg, Andrew <AGinsburg@cpsc.gov>; Crockett, David <DCrockett@cpsc.gov>; Mosheim, Abioye <AMosheim@cpsc.gov>
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Subject: RE: Letter for Mr. Alexander Hoehn-Saric, Chairman, Consumer Product Safety Commission

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Cc: Boniface, (b)(6) Boniface@cpsc.gov>; McGarvey, Carla <CMcGarvey@cpsc.gov>; Ginsburg, Andrew <AGinsburg@cpsc.gov>; Crockett, David <DCrockett@cpsc.gov>; Mosheim, Abioye <AMosheim@cpsc.gov>
Subject: RE: Letter for Mr. Alexander Hoehn-Saric, Chairman, Consumer Product Safety Commission

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Sent: Thursday, August 11, 2022 12:46 PM
To: Recht, Joel <jrecht@cpsc.gov>
Cc: Boniface, (b)(6) Boniface@cpsc.gov>; McGarvey, Carla <CMcGarvey@cpsc.gov>; Ginsburg, Andrew <AGinsburg@cpsc.gov>; Crockett, David <DCrockett@cpsc.gov>; Mosheim, Abioye <AMosheim@cpsc.gov>
Subject: RE: Letter for Mr. Alexander Hoehn-Saric, Chairman, Consumer Product Safety Commission

(b)(5)

From: Recht, Joel <jrecht@cpsc.gov>
Sent: Thursday, August 11, 2022 12:37 PM
To: Hampshire, Melissa <MHampshire@cpsc.gov>; Mosheim, Abioye <AMosheim@cpsc.gov>
Cc: Boniface (b)(6) Boniface@cpsc.gov>; McGarvey, Carla <CMcGarvey@cpsc.gov>; Ginsburg, Andrew <AGinsburg@cpsc.gov>; Crockett, David <DCrockett@cpsc.gov>
Subject: RE: Letter for Mr. Alexander Hoehn-Saric, Chairman, Consumer Product Safety Commission

Melissa and Abi –

(b)(5)

Thanks.

Joel

From: Heggs, Angela
Sent: Tue, 16 Aug 2022 20:22:37 +0000
To: Mosheim, Abioye
Subject: RE: (Urgent) 2022-08-01.RK to Hoehn-Saric-CPSC re Gas Stoves.pdf
Attachments: 20-F-00364 - Coded.pdf, FOIA Request 20-F-00364 - Folder 1.pdf, FOIA Request 20-F-00364 - Folder 2.pdf, FOIA Request 20-F-00364 - Folder 3.pdf, FOIA Request 20-F-00364 - Folder 4.pdf

Abi,

The only information that I could find that I deemed responsive, at this time, was for:

FOIA Request 14-F-00225 Meeting Log Attached Note: The case has been removed from FX (So no correspondence)
FOIA Request 20-F-00364 Material in the Review Log Attached Note: Still open, nothing released yet

Thanks~Angie

From: Mosheim, Abioye <AMosheim@cpsc.gov>
Sent: Monday, August 15, 2022 4:51 PM
To: Heggs, Angela <AHeggs@cpsc.gov>
Subject: RE: (Urgent) 2022-08-01.RK to Hoehn-Saric-CPSC re Gas Stoves.pdf

Ok, thanks, Angie. Please stop at 6 pm and resume tomorrow, I'll let them know.

From: Heggs, Angela <AHeggs@cpsc.gov>
Sent: Monday, August 15, 2022 4:32 PM
To: Mosheim, Abioye <AMosheim@cpsc.gov>
Subject: RE: (Urgent) 2022-08-01.RK to Hoehn-Saric-CPSC re Gas Stoves.pdf

Abi,

It is 4:30p and I still have about 60 documents to look over. I will stay until about 6:45p, but then I have to go.

Thanks~Angie

From: Mosheim, Abioye <AMosheim@cpsc.gov>
Sent: Monday, August 15, 2022 11:45 AM
To: Heggs, Angela <AHeggs@cpsc.gov>
Subject: RE: (Urgent) 2022-08-01.RK to Hoehn-Saric-CPSC re Gas Stoves.pdf

Thanks!

From: Heggs, Angela <AHeggs@cpsc.gov>
Sent: Monday, August 15, 2022 11:40 AM
To: Mosheim, Abioye <AMosheim@cpsc.gov>
Subject: RE: (Urgent) 2022-08-01.RK to Hoehn-Saric-CPSC re Gas Stoves.pdf

Hi Abi,

I will take a look…

Angie

From: Mosheim, Abioye <AMosheim@cpsc.gov>
Sent: Monday, August 15, 2022 10:41 AM
To: Heggs, Angela <AHeggs@cpsc.gov>
Subject: (Urgent) 2022-08-01.RK to Hoehn-Saric-CPSC re Gas Stoves.pdf
Importance: High

Hi Angie,

Hope you had a great weekend! Would you please search Document Management in FX to see if we ever released any of the documents listed on page 4 of this attachment? The request came in late Thursday but they need to know by COB today. If you do find any FOIA requests related to this, please send me the request numbers, the redacted records, and the final responses. Let me know if you have any questions. Thanks!

Abi

Abioye E. Mosheim
Assistant General Counsel, Division of Information Access
U.S. Consumer Product Safety Commission | Office of the General Counsel
4330 East West Highway | Bethesda, MD 20814
Office: (301) 504-7454 | **Mobile:** (b)(6)

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20-F-00364

Institute *for*
Policy Integrity
NEW YORK UNIVERSITY SCHOOL OF LAW

ROCKY MOUNTAIN INSTITUTE

Program Office: Hold
Requester Type: Educational
Product Codes: 0279
Track: Complex
RFD OK?: Not yet; penidng timeframe clarification
Assigned to: Korinne to clarify Timeframe for Bullet pts 3-8, and processing
Request #: Pending

Note: (b)(5)
(b)(5)

Abioye Mosheim
Chief FOIA Officer, Assistant General Counsel
U.S. Consumer Product Safety Commission
4330 East West Highway
Bethesda, MD 20814
Via foia.gov

July 6, 2020

Re: Freedom of Information Act Request

This is a request under the Freedom of Information Act. We request copies of the following records:

- All research, data, and correspondence between the Consumer Product Safety Commission (CPSC) and the Environmental Protection Agency (EPA) that informed the May 1986 publication entitled *Review Of The Consumer Product Safety Commission's Health Effects And Exposure Assessment Documents On Nitrogen Dioxide*, available on https://nepis.epa.gov/
- All research, data, and correspondence between CPSC and EPA that informed the April 1995 publication entitled *The Inside Story: A Guide to Indoor Air Quality*, located at https://www.cpsc.gov/Safety-Education/Safety-Guides/Home/The-Inside-Story-A-Guide-to-Indoor-Air-Quality
- Any record of communication between CPSC and gas stove manufacturers, gas companies, or related companies or trade associations regarding the health effects of indoor air pollution from gas stoves/ranges/ovens
- Any record of communication between CPSC and gas stove manufacturers, gas companies, or related companies or trade associations regarding potential regulatory or research initiatives related to gas stoves/ranges/ovens
- Any record of communication between CPSC and health and medical researchers, practitioners, doctors, medical associations, or related groups regarding health risks related to gas stoves/ranges/ovens
- Any documents containing information on the health effects of indoor air pollution from gas stoves/ranges/ovens
- Any documents containing information on potential regulatory or research initiatives related to the health effects of indoor air pollution from gas stoves/ranges/ovens
- Laboratory or field test reports of gas stove/range/oven emissions, including source-strength emissions

If access to any of the above information is delayed due to the impacts of COVID-19, please prioritize the documents that are readily accessible and send archived records as they become available.

In order to help to determine our status to assess fees, we are affiliated with the Institute for Policy Integrity at NYU School of Law, an educational institution, and Rocky Mountain Institute, a non-partisan, non-profit organization that provides information that is in the public interest. This request is made for a scholarly purpose and not for a commercial use.

Accordingly, we request a waiver of all fees for this request. Disclosure of the requested information is in the public interest as it is likely to contribute to public understanding of the health and environmental impacts of gas cooking stoves.

If you have any questions regarding this request, please contact us at jack.lienke@nyu.edu or dgrab@rmi.org.

Thank you for your consideration of this request.

Sincerely,

Jack Lienke
Regulatory Policy Director
Institute for Policy Integrity at NYU School of Law
139 MacDougal Street, Third Floor
New York, NY 10012
(212) 998-6222

Denise Grab
Building Electrification Program Manager
Rocky Mountain Institute
1111 Broadway
Oakland, CA 94607
(510) 501-6380

FOIA Request 142481

The following list contains the entire submission submitted July 08, 2020 01:10:03pm ET, and is formatted for ease of viewing and printing.

Contact information

First name	Jack
Last name	Lienke
Mailing Address	139 MacDougal Street, Third Floor
City	New York
State/Province	NY
Postal Code	10012
Country	United States
Phone	2129986222
Company/Organization	Institute for Policy Integrity at NYU School of Law
Email	jack.lienke@nyu.edu

Request

Request ID	142481
Confirmation ID	141956
Request description	We request copies of the following records: • All research, data, and correspondence between the Consumer Product Safety Commission (CPSC) and the Environmental Protection Agency (EPA) that informed the May 1986 publication entitled Review Of The Consumer Product Safety Commission's Health Effects And Exposure Assessment Documents On Nitrogen Dioxide, available on https://nepis.epa.gov/ • All research, data, and correspondence between CPSC and EPA that informed the April 1995 publication entitled The Inside Story: A Guide to Indoor Air Quality, located at https://www.cpsc.gov/Safety-Education/Safety-Guides/Home/The-Inside-Story-A-Guide-to-Indoor-Air-Quality • Any record of communication between CPSC and gas stove manufacturers, gas companies, or related companies or trade associations regarding the health effects of indoor air pollution from gas stoves/ranges/ovens • Any record of communication between CPSC and gas stove manufacturers, gas companies, or related companies or trade associations regarding potential regulatory or research initiatives related to gas stoves/ranges/ovens • Any record of communication between CPSC and health and medical researchers, practitioners, doctors, medical associations, or related groups regarding health risks related to gas stoves/ranges/ovens • Any documents containing information on the health effects of indoor air pollution from gas stoves/ranges/ovens • Any documents containing information on potential regulatory or research initiatives related to the health effects of indoor air pollution from gas stoves/ranges/ovens • Laboratory or field test reports of gas stove/range/oven emissions, including source-strength emissions

Supporting documentation

Additional Information	CPSC FOIA Request_2020.07.06.pdf

Fees

Request category ID	educational
Fee waiver	yes
Explanation	We are affiliated with the Institute for Policy Integrity at NYU School of Law, an educational institution, and Rocky Mountain Institute, a non-partisan, non-profit organization that provides information that is in the public interest. This request is made for a scholarly purpose and not for a commercial use.
Willing to pay	$25.00

Expedited processing

Expedited Processing	no

From:	National FOIA Portal@cpsc.gov on behalf of National FOIA Portal
To:	CPSC FOIA Request
Subject:	[Possible Spam] New FOIA request received for Consumer Product Safety Commission
Date:	Wednesday, July 08, 2020 1:30:11 PM
Attachments:	[illegible]
Importance:	Low

Hello,

A new FOIA request was submitted to your agency component.

The following list contains the entire submission submitted July 08, 2020 01:19:02pm ET, and is formatted for ease of viewing and printing.

Contact information

First name	Jack
Last name	Lienke
Mailing Address	139 MacDougal Street, Third Floor
City	New York
State/Province	NY
Postal Code	10012
Country	United States
Phone	2129982022
Company/Organization	Institute for Policy Integrity at NYU School of Law
Email	jack.lienke@nyu.edu

Request

Request ID	143491
Confirmation ID	141956
Request description	We request copies of the following records: • All research, data, and correspondence between the Consumer Product Safety Commission (CPSC) and the Environmental Protection Agency (EPA) that informed the May 1990 publication entitled Review Of The Consumer Product Safety Commission's Health Effects And Exposure Assessment Documents On Nitrogen Dioxide, available on https://nepis.epa.gov/ • All research, data, and correspondence between CPSC and EPA that informed the April 1995 publication entitled The Inside Story: A Guide to Indoor Air Quality, located at https://www.cpsc.gov/Safety-Education/Safety-Guides/Home/The-Inside-Story-A-Guide-to-Indoor-Air-Quality • Any record of communication between CPSC and gas stove manufacturers, gas companies, or related companies or trade associations regarding the health effects of indoor air pollution from gas stoves/ranges/ovens • Any record of communication between CPSC and gas stove manufacturers, gas companies, or related companies or trade associations regarding potential regulatory or research initiatives related to gas stoves/ranges/ovens • Any record of communication between CPSC and health and medical researchers, practitioners, doctors, medical associations, or related groups regarding health risks related to gas stoves/ranges/ovens • Any documents containing information on the health effects of indoor air pollution from gas stoves/ranges/ovens • Any documents containing information on potential regulatory or research initiatives related to the health effects of indoor air pollution from gas stoves/ranges/ovens • Laboratory or field test reports of gas stove/range/oven emissions, including source-strength emissions

Supporting documentation

Additional Information	CPSC FOIA Request_2020.07.08.pdf

Fees

Request category ID	educational
Fee waiver	yes
Explanation	We are affiliated with the Institute for Policy Integrity at NYU School of Law, an educational institution, and Rocky Mountain Institute, a non-partisan, non-profit organization that provides information that is in the public interest. This request is made for a scholarly purpose and not for a commercial use.
Willing to pay	$25.00

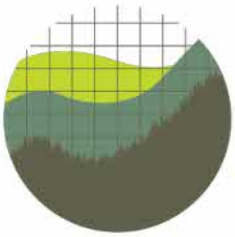
Expedited processing

Expedited Processing	no

The following table contains the entire submission, sent is formatted for ease of copy/pasting into a spreadsheet.

request_id	confirmation_id	address_city	address_country	address_line1	address_state_province	address_zip_postal_code	attachments_supporting_documentation	company_organization	email	expedited_processing	fee_amount_willing	fee_waiver	fee_waiver_explanation	name_first	name_last	phone_number	request_category	request_description
143491	141956	New York	United States	139 MacDougal Street, Third Floor	NY	10012	CPSC FOIA Request_2020.07.08.pdf	Institute for Policy Integrity at NYU School of Law	jack.lienke@nyu.edu	no	$25.00	yes	We are affiliated with the Institute for Policy Integrity at NYU School of Law, an educational institution, and Rocky Mountain Institute, a non-partisan, non-profit organization that provides information that is in the public interest. This request is made for a scholarly purpose and not for a commercial use.	Jack	Lienke	2129982022	educational	We request copies of the following records: • All research, data, and correspondence between the Consumer Product Safety Commission (CPSC) and the Environmental Protection Agency (EPA) that informed the May 1990 publication entitled Review Of The Consumer Product Safety Commission's Health Effects And Exposure Assessment Documents On Nitrogen Dioxide, available on https://nepis.epa.gov/ • All research, data, and correspondence between CPSC and EPA that informed the April 1995 publication entitled The Inside Story: A Guide to Indoor Air Quality, located at https://www.cpsc.gov/Safety-Education/Safety-Guides/Home/The-Inside-Story-A-Guide-to-Indoor-Air-Quality • Any record of communication between CPSC and gas stove manufacturers, gas companies, or related companies or trade associations regarding the health effects of indoor air pollution from gas stoves/ranges/ovens • Any record of communication between CPSC and health and medical researchers, practitioners, doctors, medical associations, or related groups regarding health risks related to gas stoves/ranges/ovens • Any documents containing information on the health effects of indoor air pollution from gas stoves/ranges/ovens • Any documents containing information on potential regulatory or research initiatives related to the health effects of indoor air pollution from gas stoves/ranges/ovens • Laboratory or field test reports of gas stove/range/oven emissions, including source-strength emissions

Email secured by Check Point



Institute *for*
Policy Integrity
NEW YORK UNIVERSITY SCHOOL OF LAW



Abioye Mosheim
Chief FOIA Officer, Assistant General Counsel
U.S. Consumer Product Safety Commission
4330 East West Highway
Bethesda, MD 20814
Via foia.gov

July 6, 2020

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Jack Lienke
Regulatory Policy Director
Institute for Policy Integrity at NYU School of Law
139 MacDougal Street, Third Floor
New York, NY 10012
(212) 998-6222

Denise Grab
Building Electrification Program Manager
Rocky Mountain Institute
1111 Broadway
Oakland, CA 94607
(510) 501-6380

FOIA Request 142481

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Last name	Lienke
Mailing Address	139 MacDougal Street, Third Floor
City	New York
State/Province	NY
Postal Code	10012
Country	United States
Phone	2129986222
Company/Organization	Institute for Policy Integrity at NYU School of Law
Email	jack.lienke@nyu.edu

Request

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Confirmation ID	141956
Request description	We request copies of the following records: • All research, data, and correspondence between the Consumer Product Safety Commission (CPSC) and the Environmental Protection Agency (EPA) that informed the May 1986 publication entitled Review Of The Consumer Product Safety Commission's Health Effects And Exposure Assessment Documents On Nitrogen Dioxide, available on https://nepis.epa.gov/ • All research, data, and correspondence between CPSC and EPA that informed the April 1995 publication entitled The Inside Story: A Guide to Indoor Air Quality, located at https://www.cpsc.gov/Safety-Education/Safety-Guides/Home/The-Inside-Story-A-Guide-to-Indoor-Air-Quality • Any record of communication between CPSC and gas stove manufacturers, gas companies, or related companies or trade associations regarding the health effects of indoor air pollution from gas stoves/ranges/ovens • Any record of communication between CPSC and gas stove manufacturers, gas companies, or related companies or trade associations regarding potential regulatory or research initiatives related to gas stoves/ranges/ovens • Any record of communication between CPSC and health and medical researchers, practitioners, doctors, medical associations, or related groups regarding health risks related to gas stoves/ranges/ovens • Any documents containing information on the health effects of indoor air pollution from gas stoves/ranges/ovens • Any documents containing information on potential regulatory or research initiatives related to the health effects of indoor air pollution from gas stoves/ranges/ovens • Laboratory or field test reports of gas stove/range/oven emissions, including source-strength emissions

Supporting documentation

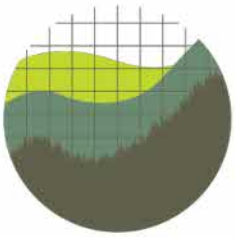
Additional Information	CPSC FOIA Request_2020.07.06.pdf

Fees

Request category ID	educational
Fee waiver	yes
Explanation	We are affiliated with the Institute for Policy Integrity at NYU School of Law, an educational institution, and Rocky Mountain Institute, a non-partisan, non-profit organization that provides information that is in the public interest. This request is made for a scholarly purpose and not for a commercial use.
Willing to pay	$25.00

Expedited processing

Expedited Processing	no



Institute *for*
Policy Integrity
NEW YORK UNIVERSITY SCHOOL OF LAW



Abioye Mosheim
Chief FOIA Officer, Assistant General Counsel
U.S. Consumer Product Safety Commission
4330 East West Highway
Bethesda, MD 20814
Via foia.gov

July 6, 2020

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- Any record of communication between CPSC and gas stove manufacturers, gas companies, or related companies or trade associations regarding the health effects of indoor air pollution from gas stoves/ranges/ovens
- Any record of communication between CPSC and gas stove manufacturers, gas companies, or related companies or trade associations regarding potential regulatory or research initiatives related to gas stoves/ranges/ovens
- Any record of communication between CPSC and health and medical researchers, practitioners, doctors, medical associations, or related groups regarding health risks related to gas stoves/ranges/ovens
- Any documents containing information on the health effects of indoor air pollution from gas stoves/ranges/ovens
- Any documents containing information on potential regulatory or research initiatives related to the health effects of indoor air pollution from gas stoves/ranges/ovens
- Laboratory or field test reports of gas stove/range/oven emissions, including source-strength emissions

If access to any of the above information is delayed due to the impacts of COVID-19, please prioritize the documents that are readily accessible and send archived records as they become available.

In order to help to determine our status to assess fees, we are affiliated with the Institute for Policy Integrity at NYU School of Law, an educational institution, and Rocky Mountain Institute, a non-partisan, non-profit organization that provides information that is in the public interest. This request is made for a scholarly purpose and not for a commercial use.

Accordingly, we request a waiver of all fees for this request. Disclosure of the requested information is in the public interest as it is likely to contribute to public understanding of the health and environmental impacts of gas cooking stoves.

If you have any questions regarding this request, please contact us at jack.lienke@nyu.edu or dgrab@rmi.org.

Thank you for your consideration of this request.

Sincerely,

Jack Lienke
Regulatory Policy Director
Institute for Policy Integrity at NYU School of Law
139 MacDougal Street, Third Floor
New York, NY 10012
(212) 998-6222

Denise Grab
Building Electrification Program Manager
Rocky Mountain Institute
1111 Broadway
Oakland, CA 94607
(510) 501-6380

FOIA Request 142481

The following list contains the entire submission submitted July 08, 2020 01:10:03pm ET, and is formatted for ease of viewing and printing.

Contact information

First name	Jack
Last name	Lienke
Mailing Address	139 MacDougal Street, Third Floor
City	New York
State/Province	NY
Postal Code	10012
Country	United States
Phone	2129986222
Company/Organization	Institute for Policy Integrity at NYU School of Law
Email	jack.lienke@nyu.edu

Request

Request ID	142481
Confirmation ID	141956
Request description	We request copies of the following records: • All research, data, and correspondence between the Consumer Product Safety Commission (CPSC) and the Environmental Protection Agency (EPA) that informed the May 1986 publication entitled Review Of The Consumer Product Safety Commission's Health Effects And Exposure Assessment Documents On Nitrogen Dioxide, available on https://nepis.epa.gov/ • All research, data, and correspondence between CPSC and EPA that informed the April 1995 publication entitled The Inside Story: A Guide to Indoor Air Quality, located at https://www.cpsc.gov/Safety-Education/Safety-Guides/Home/The-Inside-Story-A-Guide-to-Indoor-Air-Quality • Any record of communication between CPSC and gas stove manufacturers, gas companies, or related companies or trade associations regarding the health effects of indoor air pollution from gas stoves/ranges/ovens • Any record of communication between CPSC and gas stove manufacturers, gas companies, or related companies or trade associations regarding potential regulatory or research initiatives related to gas stoves/ranges/ovens • Any record of communication between CPSC and health and medical researchers, practitioners, doctors, medical associations, or related groups regarding health risks related to gas stoves/ranges/ovens • Any documents containing information on the health effects of indoor air pollution from gas stoves/ranges/ovens • Any documents containing information on potential regulatory or research initiatives related to the health effects of indoor air pollution from gas stoves/ranges/ovens • Laboratory or field test reports of gas stove/range/oven emissions, including source-strength emissions

Supporting documentation

Additional Information	CPSC FOIA Request_2020.07.06.pdf

Fees

Request category ID	educational
Fee waiver	yes
Explanation	We are affiliated with the Institute for Policy Integrity at NYU School of Law, an educational institution, and Rocky Mountain Institute, a non-partisan, non-profit organization that provides information that is in the public interest. This request is made for a scholarly purpose and not for a commercial use.
Willing to pay	$25.00

Expedited processing

Expedited Processing	no

CS# 5450

UNITED STATES GOVERNMENT

Memorandum

U.S. CONSUMER PRODUCT SAFETY COMMISSION
WASHINGTON, D.C. 20207

DATE: JUN 28 1984

TO: The Commission

THROUGH: Sadye Dunn, Secretary
THROUGH: Martin Howard Katz, General Counsel
THROUGH: Edgar Morgan, Executive Director

FROM: Peter W. Preuss, Associate Executive Director, Health Sciences

SUBJECT: Committee on Indoor Air Quality

The Committee on Indoor Air Quality (CIAQ) is an interagency group of some sixteen federal agencies which is addressing the area of indoor air quality. The group is chaired by the Environmental Protection Agency, and co-chaired by representatives of the Consumer Product Safety Commission, the Department of Energy, and the Department of Health and Human Services. There are seven subcommittees examining specific aspects of indoor air quality, two of which are chaired by CPSC staff. During the past six months, these subcommittees have prepared inventory reports outlining the various activities being conducted by the participating agencies, the extent of duplication of activities among the agencies (if any), and future research needs.

These reports, however, only cover federal activities. In order to achieve the major goal of the CIAQ, i.e., coordination of agencies in conducting future work on indoor air quality, it was decided at the May 25, 1984, meeting of the CIAQ to have the Environmental Protection Agency publish the subcommittee inventories in the Federal Register, as well as to distribute the reports to interested persons and organizations. The Federal Register notice will request public comment on the reports and request that people and organizations outside the federal government (including international organizations) conducting research on these topics provide the CIAQ with a summary of each project. This will help us to compile an inventory of research and studies on each topic, and will allow the CIAQ agencies to better identify the research that is needed in the future, and aid us in the coordination of such research.

The draft reports, which are substantially complete with respect to the information provided (but which may undergo some editing and format changes) are attached for your information.

Attachments

NOTE: This document has not been reviewed or accepted by the Commission.
Initial KD Date 6/28/84

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DRAFT

INVENTORY OF AGENCY RESEARCH ACTIVITIES
PROJECT SUMMARIES
RADON WORKING GROUP

A. EPA's Office of Radiation Programs

1. Butte study: Meteorological, House, and Occupant Effects on Indoor Radon Behavior

In July, 1983 an 18-month long period of data collection was completed in a study of the radon and radon decay product concentrations present in 68 homes in Butte, Montana. The data are associated with time periods ranging from instantaneous (grab samples) to yearly and were obtained by six different types of instruments. Three goals of the data analysis are the following:

A. Determine the precision and accuracy of measurement techniques over a range of concentrations and house and household characteristics.

B. Characterize the temporal variations of radon and radon decay products (WL) in houses including the effect of (a) daily cycles, (b) seasonal cycles, and (c) meteorological effects.

C. Characterize the spatial variations of radon and radon decay products (WL) in houses including variations with floor level and type of room.

2. Butte Study: Protocol Development

The general objective of this segment of the Butte study is to identify and evaluate sampling and measurement procedures which can be used to estimate long-term average radon and radon decay product concentrations in houses. The emphasis of this work is to develop reliable methods which can be used in two types of measurement/sampling procedures for radon and decay products. These are as follows:

A Survey Tool - for characterizing exposures in towns, cities, regions, or nationwide.

A Decision Tool - for determining whether the year-long average radon or decay product concentration of a given house exceeds some chosen action level.

3. Indoor Air Treatment Systems

Proposals are now being evaluated for a contract to develop and verify a modified Jacobi/Porstendorfer model for quantitatively

describing the behavior and fate of radon decay products in rooms with and without air treatment systems. Principal results are expected on the activity size distribution of decay products attached to particulates, dependence of unattached fractions on condensation nuclei concentration, removal efficiencies for various treatment systems, and a complete mass balance analysis (with and without treatment systems) for decay products.

4. Survey of Radon Concentrations in Ground Water Supplies

The Eastern Environmental Radiation Facility at Montgomery, Alabama has recently completed research for EPA's Office of Drinking Water. A draft report has been prepared on the results obtained from a three-year study of the radon levels found in public groundwater supplies serving 1000 or more people. Over 2500 groundwater systems were sampled in the nation-wide survey. The report also addresses the results from another 500 systems that were obtained in a previous pilot study. A final report is now being prepared.

5. Calibration/Intercomparison Studies of Radon and Decay Products.

A radon exposure chamber at EPA's Eastern Environmental Radiation Facility is used to investigate and compare the responses of instruments used to measure concentrations of radon and its decay products. Continuing research on the instrument responses provides data on measurement uncertainties and instrument reliability.

B. EPA Office of Research and Development

1. Flux Calibration Source for Radon-222

Rn-222 emanating from large sources (coal, gypsum, phosphate, slag, uranium mill tailings piles) is recognized as a potential source of indoor radon. Characterization of potential exposure requires measurement of the radon-222 release rate per unit area. Sources with a constant radon emanation rate suitable for calibration are not presently available. This research, planned for FY 1984, will develop the radon flux source and the necessary techniques for calibration of the measurements.

2. Mitigation Measures for Radon

Reduction of radon and/or radon decay product levels in structures can be achieved by prevention of radon infiltration into buildings or

cleaning/exchanging the air following infiltration. The choice of mitigation measure will be influenced by the type of structure (different construction, use, etc.) This project in the FY 84 budget proposal, will utilize representative structure types and the effectiveness of several control techniques will be assessed for each structure type.

3. Determination of Emission Factors for Radon

This project, included in the FY 84 budget proposal, provides for an assessment of the radon hazard potential of a building site. The primary source of radon in most enclosed structures is radium in the soil and near-surface bedrock, although radon from water and building materials may also be a significant source. This project's final product will be a method assessing the potential of lands to cause high radon levels in structures that are built upon them.

C. EPA's Office of Drinking Water

1. Dose Calculations for Ingestion And Inhalation of Radon

Contractors at Oak Ridge National Laboratory are collecting and analyzing dosimetry data for radon. The expected product is a background document on the distribution of dose to specific body parts that results from inhalation and/or ingestion of radon. The document is expected to be complete by September 1984. The approximate level of funding is $80,000.

2. Laboratory Study of the Ingestion Dynamics and Dosimetry of Radon

Contractors at Massachusetts General Hospital are using a short-lived xenon radioisotope to model the uptake and retention of radon for the ingestion pathway. This research will lead to better estimates of the dose that results form ingestion of radon dissolved on drinking water. The final report is expected by the end of 1984. The approximate level of funding is $75,000.

D. EPA's Industrial Environmental Research Laboratory (RTP)

1. Literature Review of Water-to-Air Transfer Coefficients

A report is in preparation on the results of a literature search of the data for water-to-air transfer coefficients of volatile organics

and radon. The schedule included a draft report for February 1984 and a final report in March 1984. Approximate funding level for the project is $25,000.

E. DOE's Office of Energy Research and Office of Health and Environmental Research

1. Radon Characterization (Lawrence Berkeley Laboratory (LBL))

This project focuses on characterizing indoor exposures to radon progeny. The multi-component program includes investigations of sources of indoor radon; transport processes and pathways by which radon enters buildings; deposition of progeny on indoor surfaces and their attachment to particulates; instrumentation development; and numerical modelling of air movement indoors.

Investigations of soil as a source of indoor radon include studies of the effects of temperature and moisture on radon release and transport; identification, through the use of aerial radiometric data, of areas with potentially high indoor radon levels; and the study of the driving forces and pathways by which radon enters a house. The roles of particulates and air movement on indoor concentrations of radon progeny will also be identified.

2. Assessment of Radon and Radon Decay Products in U.S. Single-Family Housing

This LBL project focuses on a determination of the concentrations of radon and its decay products (RDP) in the air of single-family houses in the midwestern U.S. The approach is (a) to estimate radon concentrations in many homes from two or more air samples; (b) to make continuous measurements of Rn and RDP in a small number of houses of different types; and (c) to determine, simultaneously with (b) those factors that effect behavior and levels of indoor radon and its decay products.

3. Direct Determination of Environmental Levels of Radon-222 Using the Electret to Remove Daughters at Formation (New York University Medical Center)

The purpose of this research is to develop a predictive model for indoor and outdoor radon-222 (Rn-222) concentration to estimate human exposure. A continous Rn-222 monitor has been developed and used successfully in monitoring daily fluctuations in Rn-222 concentrations inside and outside of single-family dwellings. Variables that affect Rn-222 concentrations (e. g., including temperature, heat flux, wind speed, barometric pressure, rainfall, and radium-226 (Ra-226) content of the underlying soil) will be monitored for 18 months in two different locations.

A predictive model based on a regression of the variables at the baseline locations will be used to estimate levels at two additional sites. Monitoring at those two sites will then be conducted to test prediction accuracy. A second part of the study will include use of duplicate commercially available integrating track-etch detectors at all test sites to assess the reliability of such equipment for future widespread use in evaluating Rn-222 exposures.

4. Studies of Radon in Buildings (University of Pittsburgh)

In this study techniques available for radon measurement are evaluated and compared. Where applicable, the techniques will be modified in order to increase sensitivity, increase effectiveness, provide automation, or reduce cost.

Work thus far has focused on two techniques: radon adsorption on charcoal and the use of large volume scintillation cells. The former is simple and inexpensive and has potential for use at multiple locations. The latter was developed in response to the need for high-sensitivity radon monitoring. Similarly, a study was begun to improve the properties of PERM-type detectors. Track-Etch detectors are also being used to monitor radon in 140 houses. House, wind and detector location data will be included in the analysis of the results.

5. Source Characterization and Transport Process Affecting Levels of Radon and Radon Decay Products in an Indoor Environment (New Mexico Institute of Mining and Technology)

This project involves critical studies of factors pertaining to source characterization of radon and its decay products in the indoor environment. There are four subtasks:

1. Experimental and theoretical investigations of the effects of atmospheric pressure variation and diffusion on the flux of radon and thoron from cracks and fissures are being undertaken. It is likely that a combination of model development and critical laboratory and field experiments to test the model will be required to improve our understanding of nondiffusive radon transport into inhabitied structures.

2. The effects of the magnitudes of the various radon sources and the air flow and exchange patterns in representative sites on campus are being studied. Emphasis is being placed on examination of the effects of air pressure variations, air exchange between the outdoor and the indoor atmospheres, and the radon input through cracks and fissures in the building structures.

3. The degree to which radon progeny exist as (1) radioactive atoms attached to aerosols, (2) high mobility ions, or (3) neutral "unattached" atoms are being investigated experimentally in underground and indoor environments. The phenomenon of plateout to the walls, which strongly influences the degree of equilibrum between radon and the various decay products, will also be studied.

4. New procedures are being developed for measuring radon, thoron, and their decay products including an improved electrostatic precipitation monitor to detect the decay products, and an x-ray spectrometer for measuring lead-210.

6. Study of the Atomspheric Chemistry of Polonium (Po-218) (University of Illinois)

Ths study focuses on the dynamics of radon and its decay products in indoor and outdoor atmospheres, primarily through investigation of Po-218. The charge state and particle characteristics (following attachment) are important to the deposition of radon progency in the lung or onto indoor surfaces. These dynamics are affected by environmental conditions (e.g., humidity, aerosol size distribution and concentration, and trace gas concentrations) and will be studied in this project.

Three parts of the proposed study are:

1. Measurement of the ionization potential of polonium dioxide (formed when postitively-charged Po-218 reacts with oxygen) by determing its diffusion coefficient in the presence of various trace species of known ionization potential.
2. Investigation of the gas-phase polonium mobility and the neutralization and reactions of the polonium ion.
3. Investigation of the significance of the proposed slow neutralization process due to the presence of water vapor.

7. Assessment of Radioactive and Chemically Active Air Contaminants (Environmental Measurements Laboratory)

The thrust of this task is research on the physical phenomena which govern human exposure to air contaminants arising from energy production or conservation. These phenomena include those related to sources, to transport through the air, and to sinks. Emphasis is given to small but ubiquitous sources which affect large numbers of people, rather than to large point sources.

Measurements at actual field locations are strongly stressed. Residental, occupational or outdoor locations are selected to be representative of a class of locations, so that the result can be generalized. Field measurements are supplemented with controlled laboratory experiments when the latter provide better understanding of underlying physical phenomena.

Natural background levels of air contaminants are measured in those cases where the energy-related contribution is only a small increment above background. In these cases, careful measurements are needed to meet scientific and legal needs and establish any long-term health implications.

Projects are launched when the necessary instruments, techniques, or strategies are not available through commercial channels, or when prompt response is needed to emerging issues. Once established, projects may be picked up by the private sector.

8. Indoor Air Pollution (Lawrence Berkeley Laboratory)

The objective of this prject is to develop strategies to make assessments of the health implications of indoor air pollution, emphasizing pollutants reaching higher levels than are commonly found outdoors. These include combustion products, radon and radon progeny, and organic compounds such as formaldehyde.

Each class of pollutants is characterized as to its dominant sources, the concentrations encountered in various environments, and the health and exposure implications of those concentrations.

Available information on exposures to radon progeny and on the associated added risk of lung cancer yields the estimate that indoor exposures cause 1,000 to 20,000 cases of lung cancer annually among the general population, with a significant number of individuals incurring an added individual lifetime risk of several percent or more.

The distribution of lung cancer risk from exposure to radon progeny will be examined with the intention of identifying population groups in the U.S. in which an epidemiological study can fruitfully be conducted.

9. Inhalation Hazards to Uranium Miners (Pacific Northwest Laboratory)

The objective of this program is to identify and quantify, in experimental animals, the agents responsible for the production of degenerative lung diseases similar to those observed in uranium miners.

Emphasis is on parametric lifespan studies in rodents, corroborated in beagle dogs. Exposure-response relationships are developed in animals exposed daily to various combinations and levels of radon daughters and other mine-air contaminants.

Results of these studies will identify the significant pathogenic agents in uranium mine air, quantify their relationship to observed effects, and clarify the role of background levels of radon in causing lung cancers.

These studies should provide experimental verification of suspected cause-and-effect relationships in the human epidemiology studies. They should also provide a basis for using epidemiology data to predict health effects attributable to current low radon level in mines and to increased radon levels in energy-efficient houses.

10. Internal Microdosimetry (Pacific Northwest Laboratory)

The principles of external microscopic dosimetry are applied to the correlation of radiogenic insult with the physical characteristics of inhaled and ingested nuclides.

Computer codes are developed to calculate probability densities in specific-energy deposition. Microscopic distributions describing the physical characteristics of animal tissues are measured and modeled. Dose distributions for radionuclides in the tissues of interest are prepared and applied to the interpretations of existing data on the biological effects of internally-deposited nuclides.

This application of microdosimetric concepts to internal emitters aids materially in understanding their radiobiological effects by facilitating the correlation and extrapolation of radiation effects between species and differing levels and types of exposure. These studies also help in judging which cases of internal deposition can be considered similar and which different from other radiation risks.

F. National Bureau of Standards

1. The Center for Radiation Research (CRR) is currently working on the development of a standard for radon in water. Completion of the work is expected within one year. The CRR is also collecting data in preparation for the development of a radon-in-air standard.

2. Although the Center for Building Technology (CBT) has no current research activities in radon it maintains an active interest in the effects of 'tight' homes and control measures on indoor radon levels, the radon contributed by solar rock beds, and the effect of heating, ventilation and air conditioning systems on levels of radon and radon decay products.

G. Department of Housing and Urban Development

Elevated indoor levels of radon and its decay products are of serious concern to HUD as a result of its mandate to insure that all HUD-assisted projects are located in a safe and healthful environment. The Department has actively participated in programs to develop measurement protocols, to incorporate construction features that will reduce radon infiltration and to develop appropriate remediation techniques ranging from removal of contaminated material to installation of a system for removing radon and its decay products from the indoor atmosphere. The Department's involvement has been particularly prominent in Colorado, Florida, and Wyoming.

Although HUD has no current research activities the possibility exists for funding of collaborative projects with other State and Federal agencies.

H. Tennessee Valley Authority

1. Survey of Radon and Radon Decay Product Concentrations in 60 Homes

A 60-home study of the background levels and variability in radon and decay product concentrations is expected to begin by April 1984. The frequency of measurements will allow the characterization of the seasonal variations seen in radon and decay product concentrations; measurements of the air-exchange rate in each house are also proposed. The choice of contractor and final details of the study are still under consideration.

TVA is interested in assessing the amount of indoor radon that is contributed by the radium found in building blocks made of phosphate slag from TVA's fertilizer industry. The geological characteristics of the phosphate ore include a radium concentration higher than that found in most soils or ores. The study of homes that do not incorporate phosphate slag blocks will provide the background information that is needed to assess the risk present in homes that do utilize the blocks.

I. Bonneville Power Administration

The Bonneville Power Administration (BPA) sponsors a conservation program in the states of Washington, Oregon, Idaho and Montana. A major portion of that program involves infiltration and ventilation

reduction measures which have the potential to increase indoor pollutant levels. BPA is conducting several indoor air quality studies to better understand these effects; three of those studies are surveys directed at specific building sectors. The surveys are expected to be complete by January 1986. Descriptions of the three surveys that involve radon are provided below:

1. New Homes: Approximately 50 energy efficient new homes will be compared to 50 ordinary new homes to determine if pollutant levels (e.g., radon, formaldehyde, particulates) vary between the two groups. Monitoring will be accomplished with Track-Etch passive detectors. Detectors will be deployed for a three-month period during the heating season and analyzed at the least expensive level ($18/monitor). Air infiltration rates will be measured concurrently using perfluorocarbon (PFT) and blower door tests. In addition to identifying existing levels of pollutants, mitigation strategies (e.g., air-to-air heat exchangers) will be tested.

2. Existing Homes: A portion of this study will be designed to characterize existing radon levels in two thousand homes. The sample will be stratified by house foundation type and geographic zone. Sampling will be performed for a three-month period in each home during the heating season using Track-Etch detectors ($18 analysis level). In a subset of twelve homes the effects of weatherization on radon levels will be assessed. In a subset of fifty homes the effectiveness of various mitigation strategies will be tested. Homes that will be weatherized and mitigated will have infiltration rates measured using PFT and blower doors. Also, site specific meteorological data will be collected.

3. Commercial/Institutional Buildings: This survey will involve thirty-five buildings of various uses (e.g., schools, offices), age, and size. Pollutant levels (including radon) will be monitored concurrently with infiltration rates. The objective of this survey is to establish a protocol for characterizing pollutant levels in relation to ventilation rates. Measurement methodology will be as in #1 above.

RADON WORKING GROUP

EPA Co-Chair: Rich Guimond
*
FTS 557-0704

DOE Co-Chair: Wayne Lowder
U.S. Department of Energy
376 Hudson Street
NY, NY 10014
353-5364 (Germantown)
FTS 660-3632 (NY)

HUD: Jim Christopulos
451 Seventh Street, S.W.
Washington, D.C. 20410
FTS 755-7225

BPA: Rob Rothman, KKT
Bonneville Power Admin.
Post Office Box 3621
Portland, Oregon 97219
(503) 230-5489

EPA: Stan Lichtman
*
FTS 557-8927

EPA: Tim Krohe
*
FTS 557-8224

EPA: Rick Cothern
Office of Drinking Water (WH-550)
U.S. Environmental Protection Agency
401 M Street, S.W.
Washington, D.C. 20460
FTS 382-7584

NBS: Sam Silberstein
NBS, Building 226
Washington, D.C. 20234
921-2758

TVA: Ron Maxwell
TVA Reservation
124 Multipurpose Bldg.
Muscle Shoals, Alabama 35660
FTS 872-8767
(205) 386-2767

*Office of Radiation Programs (ANR-460)
U.S. Environmental Protection Agency
401 M Street, S.W.
Washington, D.C. 20460

DRAFT

Report of the CIAQ Radon Subgroup

The Problem:

The naturally-occurring radioactive gas radon has been generally recognized to be one of the key pollutants in the indoor environment. It is produced by the radioactive decay of the radium present in all environmental media. In most structures the main input of radon is from the soil gas, which enters the indoor space through openings and cracks in the foundation. In some cases, however, the diffusion of radon out of building materials or its degassing from the water supply can be important source pathways.

The radon concentration in indoor air is determined by competition between the input rate and losses due to radioactive decay and air exchange with the outdoors. Radon concentrations indoors are typically 2 - 10 times higher than those outdoors, and thus are responsible for the bulk of human exposure, taking account of relative occupancy times.

The decay of radon produces a series of short-lived products, which readily attach to atmospheric particulates. When these particles are inhaled, they become deposited in the respiratory tract. Two of the short-lived product of radon are alpha particle emitters, and it is the irradiation of the target cells by these highly ionizing particles that is responsible for the incidence of excess lung cancers that have been observed in uranium miners.

The risk of lung cancer induction from exposure to radon and its decay products has been estimated from epidemiological studies of miner populations in various countries, from animal studies, and from models of radiation dose to lung tissues. At typical environmental exposure levels, a lifetime lung cancer risk on the order of 0.1% can be estimated, which accounts for a significant fraction of the spontaneous (non-smoking - related) incidence of lung cancer. If these risk estimates are borne out by further research, several thousand deaths per year in the U.S. population can be attributed to indoor radon exposure. The estimated upper range of health effects is 30,000 deaths per year.

Federal agency interests in this situation derive from two somewhat independent considerations. First, available data indicate that the

distribution of radon exposures in U.S. housing is broad, ranging from one-tenth of "typical" levels to a factor of 100 higher. The high-exposure tail of the distribution is thus a matter of concern to such agencies as EPA, HHS and HUD because the risk of lung cancer for individuals living in these structures can amount to the order of 10 percent over a lifetime. Second, radon exposure is approximately in inverse proportion to the indoor-outdoor air exchange rate, and thus a reduction in average exchange rate due to energy conservation practices and improvements in building technology would be expected to cause a proportionate increase in lung cancer deaths. This consideration is the basis for the interest of DOE, DOC (NBS), TVA and BPA.

Research in this country and elsewhere is providing the information needed to develop cost-effective control technology. Remedial actions on existing structures have been and will continue to be applied in special situations. However, because most of the lung cancers probably occur as a consequence of exposure to near-normal radon levels (because this is the situation experienced by most of the population) the greatest public health impact will be made by appropriate modifications in building construction practices.

Current Federal Activities:

Summaries of radon research programs being sponsored by Federal agencies are given in Attachment 1. Several aspects of these programs should be highlighted.

The substantial survey effort in Butte, Montana, sponsored by the EPA Office of Radiation Programs in cooperation with the Montana Department of Health and Environmental Sciences, is providing important information on the effectiveness of various field techniques for the measurement of radon and radon decay products. In addition, the DOE Environmental Measurements Laboratory (EML) is conducting a series of radon measurement intercomparison exercises, involving DOE, DOE-contractor and EPA laboratories as well as other U.S. and foreign institutions. EML is also one of the four reference laboratories in an international radon intercomparison program being sponsored by the OECD Nuclear Energy Agency. These activities are important steps in defining optimal survey strategies and in assuring the comparability of data from field studies performed throughout the world.

The DOE research programs are directed at improving our understanding of the phenomena of radon production and transport into structures, radon decay product attachment to atmospheric particulates, deposition on room surfaces and tissues of the respiratory tract, and the consequent dose to the critical cells. This information will be applied to the determination of environmental factors that influence radon exposure and the development of optimum control strategies.

Many of these studies are producing a limited data base on radon exposures in structures in various parts of the United States. It is becoming clear the high radon levels in inhabited structures may not be as unusual as once supposed, particularly in the Northeastern U.S., and that there may be a substantial number that exceed occupational limits.

The increased interest in indoor radon has resulted in the convening of six workshops or symposia on the subject in the last four years (May 1980 in Rome, June 1980 in New York, March 1981 in Berkeley, August 1981 in Montgomery, November 1982 in New York, and October 1983 in Capri). The report of the 1982 workshop is attached as Attachment 2. Another workshop in May of 1984 is being sponsored by the National Academy of Sciences to discuss future research priorities and to evaluate the role of federal agencies (including that of NAS) on this issue. Radon will also be an important subtopic at large conferences on indoor air quality to be held in Stockholm in August and Knoxville in October.

Future Research Priorities

The attached 1982 workshop report provides an indication of the large-scale international effort being focussed on the indoor radon problem. At the 1983 Capri meeting, 85 papers were presented, mostly on radon and mostly from Europe. It is clear that U.S. agencies and scientists must maintain continuing communication with the international community in order to form appropriate judgments on research gaps and priorities. The cooperative arrangements between DOE and the Commission of the European Communities on overall radiation research provide a useful mechanism for assuring this communication.

Part I of the attached workshop report gives the recommendations of the participating scientists on research priorities. Though the specific wording could be updated, the essential aspects remain fully valid. The research

programs described in Attachment 1 are responsive to many of these recommendations, and the Capri meeting demonstrated that there is very little research that could be considered to be an undesirable duplication of effort with respect to programs in other countries. A detailed reconsideration of these priorities would be valuable, and is likely to take place at the planned NAS workshop.

The importance of the recommendations for a large-scale assesment of radon in U.S. housing has been enhanced by recent developments. The nature of the indoor radon problem is becoming clear, but its extent can not be defined with the available data. The ultimate difficulty is that occupational standards for radon exposure, determined on the basis of health risks comparable to other occupational situations, are only a factor of 20 above normal indoor exposures. Thus, some fraction of the U.S. population is being exposed to radon levels that would not be tolerated occupationally. This fraction will increase as energy conservation practices are introduced on a large scale. A national assessment of representative U.S. structures, appropriately distributed in terms of climate and geology, would serve to identify those areas and conditions where high indoor radon exposures are likely to permit a quantitative estimate of the effect on radon exposure of the introduction of advanced energy conservation technology.

Plans for a national radon assessment are presently in the discussion stage. If and when detailed plans are developed, the option will be available to incorporate its essential element into a multi-pollutant survey.

Planned Subgroup Activities

Prior to the next CIAQ meeting, presumably in the Fall, the subgroup will update the research project abstracts, including those new programs that are expected to be funded in FY 1985. More precise statments of agency interests and concerns will be prepared. Finally, overall research priorities will be reassessed, taking account of the NAS-sponsored workshop. An important element of this will be a progress report on the development of a national assessment plan and the appropriate Federal role in conducting this large-scale project.

Wayne Lowder, DOE/ER
Richard Guimond, EPA/ORP

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FIRST REPORT OF THE CIAO* WORKGROUP

ON FORMALDEHYDE

FEBRUARY 3, 1984

*Committee on Indoor Air Quality

INTRODUCTION

On January 12 and 15, 1984, the Formaldehyde Workgroup (representatives of Federal agencies and departments) met to exchange and compile information on various activities related to formaldehyde such as, monitoring surveys, epidemiological studies, materials research, health studies, etc. The Formaldehyde Workgroup is a function of the Committee on Indoor Air Quality which has as one of its purposes the collection and dissemination of data related to indoor air quality topics to Federal, state, and local governments and the public. Because of certain time constraints, the report may not be complete as it could have been. However, the co-chairs believe it represents the significant formaldehyde related projects of the Federal government.

a. FORMALDEHYDE RELATED ACTIVITIES

I. MONITORING STUDIES

1. The primary focus of the Department of Housing and Urban Development's indoor air quality activities involve radon and its daughter-nucleides and urea-formaldehyde as used as an insulation source and as a resin in wood products.

The greatest amount of the Department's indoor air quality activity is investigations of urea-formaldehyde based wood products, specifically those used in the construction of manufactured homes.

In 1979, the Department sponsored a two part study entitled "An Evaluation of Formaldehyde Problems in Mobile Homes". The lead contractor was Technology & Economics, Inc. (T & E), of Cambridge, MA.

- Volume I of this study was primarily an examination of the state of the art of formaldehyde related research. The contractor also explored the size and magnitude of the problem nationally.
- Volume II (unpublished) concentrated on assessing the magnitude of the problem based on three states which possessed substantive consumer complaint data. Due to perceived under-reporting, T & E was not able to provide a specific estimate of the problem's magnitude. Nor did the contractor feel it could recommend a standard for concentration levels.
- Anticipating possible regulatory action, the Department consulted at length with scientific and engineering personnel in other government agencies and with industry experts regarding the development of an objective,

accurate and eocnomical testing procedure for measuring formaldehyde in manufactured homes. The Department neeced a test procedure appropriate for enforcement purposes. Since no large scale testing had previously been performed on manufactured homes, the Department contracted with Clayton Environmental Consultants, Inc. of Southfield, Michigan, in 1980, to perform two studies.

(a) The first study was entitled, "Evaluation of Formaldehyde Problems in Mobile Homes - Testing and Evaluation." Using the pararosaniline test method, optimized by Lawrence Berkeley Laboratory as the primary monitoring method, this study involved three primary tasks:

1. Evaluating formaldehyde field monitoring procedures
2. Validating and using the adopted monitoring procedure in a representative sample of 280 new and occupied manufactured homes
3. Evaluating the relationship between values obtained from in-home testing of manufactured homes and product testing of wood used to manufacture the homes.

(b) The second study, "Evaluation of the Relationship Between Formaldehyde Emissions from Particleboard Mobile Home Decking and Hardwood Plywood Wall Paneling in Experiemental Mobile Homes" assessed the impact on formaldehyde concentrations of using reduced formaldehyde-emitting hardwood plywood paneling and particleboard decking. In this study:

1. Four (4) test homes were specifically constructed for this purpose.
2. Both the small scale dessicator test and the large scale chamber test were used in product analyses (HUD).

2. Below is a list of industrial hygiene surveys conducted by NIOSH which have included sampling for formaldehyde. The primary objective of these activities has been to identify industries suitable for our epidemiologic study, and to characterize in detail formaldehyde exposures in the garmet plants selected for the study. As a secondary objective, our industrial hygiene group has planned to document formaldehyde exposures in other industries. Finally, as a part of our Health Hazard Evaluation (HHE) program NIOSH has conducted environmental measurements for formaldehyde at four office buildings (NIOSH).

INDUSTRIAL HYGIENE SURVEYS CONDUCTED

Garmet Manufacturers - 7 Walk Throughs
3 In-Depths

Mushroom Growing Operations - 1 Walk Through

Textile Plant - 2 Walk Throughs

Chemical Manufacturer - 2 Walk Throughs

Paraformaldehyde Packaging - 1 Walk Through

Hospital Dialysis - 1 Walk Through

UFFI Insulation Manufacturer - 1 Walk Through

Pathology Laboratory - 1 Walk Through

Indoor (Office) Air - 4 Walk Throughs (HHEs)

3. The Bonneville Power Administration (BPA) sponsors a regionwide conservation program. A major portion of that program involves infiltration and ventilation reduction measures, all of which potentially increase indoor pollutant levels. Accordingly, to better understand these effects BPA is conducting several indoor air qualtiy studies. Three of these studies are surveys directed towards three specific building sectors: (1) New Homes, (2) Existing Homes, and (3) Commerical/Institutional Buildings. In each of those surveys, formaldehyde will be monitored in the following manner.

(a) New Homes: Approximately 50 energy efficient new homes will be compared to 50 ordinary new homes to determine if pollutant levels (e.g. radon, formaldehyde, particulates) vary between the two groups. Monitoring will be accomplished with a passive (one week exposure) monitor developed by LBL. Also, mitigation devices (e.g. air to air heat exchangers, de-humidifiers) will be tested for their effectiveness. Monitoring will be accompanied with infiltration tests (i.e. PFT and blower door).

(b) Exisiting homes: A portion of this study will be directed towards characterizing existing formaldehyde levels in 400 homes. (Homes will be selected by age categories to demonstrate decay characteristics). This study will also include before and after retrofit IAQ measurements as well as mitigation testing.
Another portion of this study is expected to include 100 mobile homes. Formaldehyde levels will be characterized

concurrently with infiltration rates. Measurement methodology will be as described in (a) above.

(c) Commercial/Institutional Buildings: This survey will include approximately 35 buildings of various uses (e.g. schools, offices), age, and size. Pollutant levels (including formaldehyde) will be measured concurrently with infiltration rates. The objective of this survey is to establish a protocol for characterizing pollutant levels in relation to ventilation rates. Measurement methodology will be as in (a) above (BPA).

4. The Oak Ridge National Laboratory (ORNL) under contract to CPSC, has measured formaldehyde levels over several seasons in 30 residences in the Oak Ridge, Tennessee area. Other pollutants were monitored as well. Some homes were insulated with urea-formaldehyde foam insulation, some contained pressed wood products, and some had no obvious source of formaldehyde. Preliminarily, it has been found that in some of these homes, the formaldehyde levels can vary as much as 3-fold in a single day and as much as 10-fold from season to season. The data that has been collected is currently being analyzed, and a final report should be available in the latter part of 1984 (CPSC).

5. As a general study of indoor air pollutants, three office buildings, homes for the elderly, and schools plus two hospitals are the subject for monitoring for VOCs, formaldehyde, respirable particulates, and metals (EPA).

6. The objectives of this research are to study the relationship between building design and operation and indoor air quality. Central to this effort is the study of the effects of energy conservation strategies in buildings on indoor pollutant concentrations. Thus, the main objective of the work may be considered to be the establishment of criteria that permit the improvement of energy efficiency in buildings while avoiding excessive exposures indoors.

This research has several components. These include measuring the concentrations of pollutants found within buildings, identifying the sources of these pollutants, studying the emission characteristics of the sources, and developing techniques to control the pollutant concentrations with a minimal energy cost. The pollutant classes of concern include emissions from combustion appliances, radon and its daughters (from building materials and natural soil), and formaldehyde and other organics (from building materials and other sources).

The activities that support this research include field measurement of pollutant concentrations in buildings in many areas of the United States, intensive laboratory studies of source and pollutant behavior, and laboratory and field study of pollutant control techniques (DOE/LBL).

II. EPIDEMIOLOGICAL STUDIES

1. Embalmers use numerous chemicals in the course of thier work, e.g. formaldehyde, sodium nitrate and various dyes. Recent inhalation studies have shown a marked excess of squamous cell carcinoma of the nasal cavity among rats exposed to 15 ppm formaldehyde vapor. In humans, formaldehyde is known to have acute effects on the respiratory system and skin, but the extent of its chronic effects has not been established. This study utilized data on deceased embalmers identified from licensure records of the Bureau of Funeral Directors and Embalmers in California. The study group includes 1041 men and 63 women who died between 1925-1980. Among white men, proportionate mortality ratios were significantly elevated for cancer, arteriosclerotic heart disease, and suicide. Significant deficits of deaths were noted for respiratory diseases and diseases of the genitourinary system. Among cancers, mortality was significantly elevated for cancers of the colon, brain, and prostate, and for leukemia. Mortality was analyzed by length of licensure and age at death. Results are compared with the proportionate mortality experience of a group of embalmers from New York State (NCI).

2. In view of recent findings of nasal cancer in rats exposed to formaldehyde vapors, we investigated the proportinate mortality experience of embalmers licensed to practice in New York State. Mortality was significantly elevated for cancers of the skin and colon and for arteriosclerotic heart disease, whereas significant deficits were seen in mortality from respiratory diseases and accidents. Respiratory cancer mortality was not excessive and no

deaths were attributed to nasal cancer. Mortality was significantly elevated for cancers of the skin, kidney, and brain among those licensed only as embalmers, whereas mortality patterns were unremarkable among those licensed also as funeral directors (and presumably less exposed to formaldehyde). These preliminary results indicate the need for occupational cohort studies to clarify the carcinogenic potential of formaldehyde (NCI).

3. Formaldehyde is one of the principal compounds used by anatomists to preserve and fix biologic tissues. Other compounds include phenol, methyl alcohol, solvents, dyes, and stains.

The purpose of this study is to investigate whether there are differences in the risks of death from various causes between a group of anatomists and others with little or no exposure to these compounds. Included in the study are 2321 men who joined the American Assocation of Anatomists between 1888 and 1970. Vital status as of December, 1979 will be ascertained. About one third of the individuals in the study are known to have died. Cause-specific mortality rates for anatomists will be compared with rates for the general population using modified life table techniques.

Information on academic subspecialities and membership in other professional assocations will be used to identify a subgroup of anatomists who were probably exposed to high concentrations of formaldehyde. The potential for exposure to other chemicals will also be considered (NCI).

4. To evalute the risk of cancer among industrial populations a cohort mortality study of workers employed in formaldehyde producing and using industries has been intitiated. Approximately 28,000 workers with exposure to formaldehyde before 1966 from industries such as formaldehyde production, resin making, photographic products, decorative laminates, heat-resistant plastic products, and plywood are being assembled. Cause specific mortality rates will be compared to expected rates based on the general U.S. population. Work histories, plant production levels, and industrial hygiene measurements will be used to categorize formaldehyde exposures to provide information on exposure-response gradients (NCI).

5. A population-based case-control study of cancers of the sinus and nasal cavity and oro- and nasopharynx is underway to investigate the role of formaldehyde in the etiology of these neoplasms. Cancers of these sites are primarily squamous cell carcinomas and likely have several risk factors in common. The plan is to use a single control group, equal in size to the overall case group, and a single protocol and set of study instruments to investigate all neoplasms under study. This will result in increased efficiency of data acquisition and a ratio of controls to cases of slightly greater than 1 (1.3:1) for the pharyngeal sites and approximately 4:1 for the sinus and nasal cavity sites, thus resulting in an increase of statistical power over a 1:1 ratio.

The investigation is being conducted in a 13-county region of Northwestern Washington State served by a population-based cancer registry. All incident cases of the study sites in this population for a defined period of time will be identified who were between 15 and 74 years of age at diagnosis. A control group will be selected at random from the same population using a sampling technique referred to as random digit dialing. Telephone interviews will be conducted with cases or their next-of-kin and controls to ascertain prior exposure to formaldehyde in both occupational and non-occupational settings. Analyses of these data will help to clarify the role of formaldehyde inhalation in the etiology of these neoplasms, taking into account other known or suspected risk factors (EPA).

6. In response to the findings of nasal cancer in rats, NIOSH in 1980 began searching for a group of workers exposed to formaldehyde that would be suitable for an epidemiologic study. After investigating a number of potential groups, garment workers exposed to formaldehyde were chosen for this study. This group was chosen because it is relatively large, has reasonably continuous exposures to formaldehyde, and is not exposed to other potentially carcinogenic agents.

This study has two phases: an initial phase consisting of a proportionate mortality study (PMR), followed by a retrospective cohort mortality study (SMR). The first phase (PMR) has been completed, and a report is currently undergoing agency review. The second phase (SMR) is in progress and should be completed in 1985 (NIOSH).

7. Under the TSCA section 28 cooperative agreement with Wisconsin, three epidemiologic sampling methodologies available to the Division of Health were evaluated to select the most feasible method of enrolling mobile home residents for studies of formaldehyde (HCHO) concentration measurements and construction, environmental factors and health status. Utilizing property tax records, an age (home) stratified, random sample cohort of 100 homes less than 3 years old and 37 homes of more than 3 years of age (mean 67 months) were enrolled and followed prospectively at monthly intervals. A total of 976 monthly home visits were made (77% of homes had 6 or more monthly visits).

A total of 1874 air samples, 730 blanks and 573 impinger blanks were analyzed. Formaldehyde air measurements ranged from below detection (0.1 ppm by the method used) to 2.84 ppm. The average monthly concentration from homes less than three years old was 0.54 ppm and 0.19 from homes older than three years. Regression modeling found that the log of home age was the best environmental parameter for predicting formaldehyde levels.

A total of 2,423 monthly self-administered symptom questionnaires were completed by the 288 persons in the homes which remained under study for 6 or more months. Also fifty-three adults participated in clinical examination (EPA).

III. MATERIALS RESEARCH

1. USDA and ORNL, under contract to CPSC and the CPSC laboratory have completed or are currently performing a number of activities. These are:

(a) Availability of formaldehyde from DMDHEU (dimethyloldihydroxyethyleneurea) treated textiles. Work (USDA) is complete, report available. It was found that even after 25 launderings, formaldehyde can still be released from treated textiles, although the release is much less than that from a new, unwashed product.

(b) Penetration of formaldehyde radiolabel through rabbit skin in vivo. Work (CPSC Laboratory) is complete, report available. It was found that ^{14}C label from ^{14}C labeled formaldehyde, or cloth treated with DMDHEU synthesized from ^{14}C formaldehyde, can be absorbed through the skin of rabbits in vivo (0.1 - 10% of the dose applied penetrates). Most of the applied radiolabel is retained by the skin itself.

(c) Penetration of intact formaldehyde through rabbit skin in vitro. Work (CPSC Laboratory) is complete, report available. It was found that although ^{14}C from ^{14}C formaldehyde could penetrate freshly prepared rabbit skin (placed in a diffusion cell), the radiolabel was not detectable as formaldehyde itself (perhaps the label was in the form of reaction products such as formate).

(d) Textile survey. Work (ORNL) in progress. Textiles of various types are being collected from large and small domestic manufacturers and importers. Fabrics will be

analyzed for formaldehyde content by three different tests.

(e) Correlation experimentation. Work (ORNL) in progress. Hazard (skin sensitization reaction) is determined by using patch tests, where known concentrations of formaldehyde are placed on subject's skin. If a fabric is rated at a certain ppm by a certain test, its equivalency in terms of a solution of formaldehyde must be determined. Tests will therefore be performed looking at how much formaldehyde is released from a fabric, rated as to ppm by a standard test, under damp conditions, as compared to patch (e.g. piece of cloth) dampened with a known formaldehyde solution (CPSC).

2. The Inhalation Toxicology Research Institute/Lovelace Biomedical and Environmental Research Institute (Lovelace) and the ORNL, under contract to CPSC, have completed the following formaldehyde-release investigation. Various consumer products suspected of emitting formaldehyde were tested for such release using a dessicater methodology. Other than pressed wood products, the highest emitters that would likely have a high loading in residences were fiber wools glass products. The report (Lovelace) is available.

3. The following work on pressed wood products is ongoing at CPSC & ORNL:

(a) A survey of currently manufactured particleboard, paneling, and medium density fiberboard samples for formaldehyde emission.

(b) A decay study (how emission changes as products age).

(c) Characterization: How emission varies with changes in temperature, relative humidity, and background formaldehyde concentration.

(d) Diffusion barriers: Effect of barriers such as rugs, tile, etc. which overlay a formaldehyde source.

(e) Sinks: Effect of formaldehyde absorbers such as gypsum board on indoor formaldehyde levels.

(f) Modeling: Impact of formaldehyde sources under given conditions on indoor formaldehyde levels will be modeled using the data gathered in the other aspects of this study. Two models, an emission model and an indoor air model (which considers loading, room site, air exchange & mixing, etc.) are combined to obtain this prediction.

(g) Model verification: The modeled levels will be compared with actual levels in well characterized homes and a large chamber.

(h) Development of an emission monitoring device. This device will determine emission rates when placed on pressed wood product formaldehyde sources.

Work is ongoing; expected completion end of 1984 (CPSC).

IV. SURVEY DESIGN/SURVEYS

1. Conducted most recently from 1976 to 1980, the National Health and Nutrition Examination Survey (NHANES) is tentatively scheduled to be conducted again in 1987. Planning is beginning now. The Survey collects prevalence data for specifically defined diseases or conditions, as well as data on health related parameters such as blood pressure, visual acuity, or serum cholesterol. Data are gathered by (1) a home visit in which an interview is obtained and by (2) a complete physical exam conducted in a mobile trailer. About 20,000 persons were examined. The NHANES I Augmentation Survey, 1974-1975, included a Water Sample Collaborative Study with NHLBI and EPA in which a tap water sample was taken at the time of the home visit. This precedent suggests that other kinds of samples could also be taken during the home visit, e.g. formaldehyde (NCHS).

2. The National Health Interview Survey is conducted annually in approximately 40,000 households including about 100,000 persons and provides National data on the incidence of acute illness, accidental injuries, the prevalence of chronic conditions and impairments, utilization of health care services, and other health related topics. The data are presented by selected demographic and socioeconomic characteristics of the U.S. civilian, non-institutionalized population. Data are gathered by an interview questionnaire (NCHS).

3. The National Death Index is a system by which researchers who are following a cohort of individuals may more easily search

death certificate data to learn whether any of their "lost to follow-up" subjects have died, and if so, which state the researcher should probably contact for a copy of the death certificate. Researchers submit key, identifying data such as name, social security number, and birthdate. NCHS can return a list of possible matches among recorded death data (NCHS).

4. Data on State health department programs for hazard evaluations of nonoccupational indoor air pollution (NIAP) were acquired through the support of the U.S. Department of Health and Human Services Regional Directors and the assistance of National Institute for Occupatinal Safety and Health (NIOSH) Regional Consultants. A questionnaire was distributed to each Regional Office on July 6, 1982, and the summary was prepared after the last State response was received at the end of Decmeber 1982. Responses were obtained from each of the 50 States, the District of Columbia, the City of New York, Puerto Rico, and the U.S. Virgin Inslands.

Principal findings of the survey are that state NIAP activities usually do not provide air sampling analyses, guidelines for exposure standards, comprehensive evaluations of health complaint data, comprehensive field services and research related to formaldehyde (communicated by NAS).

5. The staff of the Federal Trade Commission is gathering data concerning consumer awareness of information about allegedly adverse health effects of formaldehyde emissions in the ambient

air of mobile homes. In January 1983, the Consumer Federation of America petitioned the Commissior to issue an industry-wide rule to require formaldehyde warnings in mobile homes. The Commssion voted to deny this petition, but directed the staff to monitor closely developments concerning formaldehyde emissions in mobile homes, such as medical and scientific data pertaining to alleged health risks, technological developments to reduce formaldehyde emissions in mobile homes, voluntary industry initiatives to inform buyers of the alleged health risks, and federal and state regulatory activities.

The staff is currently gathering information on disclosure of mobile home formaldehyde emissions from various private and governmental sources. The staff is aware that the Deparment of Housing and Urban Development (HUD) has specifically requested comments on the need for, and the form of pre-sale disclosure of formaldehyde emissions to mobile home purchasers (48 C.F.P. 37136, at 37151, August 16, 1983). The Commission may submit comments to HUD on this subject based on the information compiled during the staff's investigation (FTC).

V. OTHER

1. Foliage Plants for Removing Indoor Air Pollutants from Energy Efficient Homes--A sealed, Plexiglas® chamber with temperature and humidity control and illuminated externally with wide spectrum grow lights was used to evaluate the ability of golden pothos (Scindapsus aureus), nephthytis (Syngonium podophyllum), and spider plant (Chlorophytum elatum var. vittatum) to effect the removal of formaldehyde from contaminated air at initial concentrations of 15-37 ppm. Under the conditions of this study, the spider plant proved most efficient by sorbing and/or effecting the removal of up to 2.27 mg formaldehyde per cm^2 leaf surface area in 6 h of exposure. The immediate application of this new botanical air-purification system should be in energy-efficient homes that have a high risk of this organic concentrating in the air, due to outgassing of urea-formaldehyde foam insulation, particleboard, fabrics and various other synthetic materials (NASA).

2. The Federal Trade Commission recently intitiated formal law enforcement actions against two national advertisers of portable household indoor air cleaning equipment. Rush-Hampton Industries, Inc., manufacturer of the Ecologizer CA/90 air cleaner, and Associated Mills, Inc., manufacturer of the Pollenex air cleaner, have both been charged with violation of Section Five of the Federal Trade Commission Act. The charges, which will be ruled upon after formal hearings, are the result of advertising claims concerning the performance capabilities and effectiveness of these shousehold air cleaners. In 1982 the

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relatively new portable indoor air cleaning industry as a whole sold more than six million units to consumers, at prices ranging from $20-$40.

3. At the Lovelace ITRI, a small Office of Health and Environmental Research sponsored research effort on formaldehyde toxicology has been in progress for about two years. In this work, patterns of formaldehyde deposition in various parts of the respiratory tract after inhalation (as vapor) are being determined in nose-breathing animals and in species that breathe like humans. This information is correlated with data on formaldehyde metabolism in different regions of the respiratory tract. Such data are needed to define health risks under different exposure conditions. It is of particular interest to study the binding of formaldehyde to common indoor particulates and to determine the extent to which particle-bound formaldehyde may be retained in the deep lung. It is important to know if particle-bound formaldehyde present in indoor air presents a significant cancer risk in the deep lung; this requires detailed knowledge concerning the disposition of such particles and the bioavailability of the formaldehyde that they carry to the deep lung. Research on this problem is in progress and will increase in FY 1984. Future work will also include a study of the potential for formladehyde or fibers coated with formaldehyde to induce hypersensitivity reactions (allergic responses) in the upper airways.

In FY 1984, it is planned to initiate at the University of Maryland a controlled study to determine acute and delayed effects of inhaled formaldehyde in healthy nonsmoking human subjects exposed at levels of 0.5, 1.0, and 1.5 ppm. Objective measurements of pulmonary function and other procedures will be utilized to assess biological responses (DOE).

4. Elevated concentrations of indoor air contaminants can be present in buildings with low ventilation rates and/or high contaminant source strengths. Adverse impacts on the health or comfort of occupants may result from increased exposure to these contaminants. The number of buildings with elevated contaminant concentrations may be increasing because energy conservation measures that reduce the rate of ventilation are becoming increasingly popular. Effective and economical techniques for controlling the concentrations of contaminants in the indoor air need to be identified and developed so that adverse health effects are avoided and energy conservation measures can be implemented.

The performance of various control techniques will be assessed in detailed laboratory experiments. Based on the measured performance, models will be developed and/or utilized to predict the impact of the control technique on indoor pollutant concentrations and energy consumption. Control techniques may be evaluated further in field studies. Specific efforts for FY'84 are discussed below.

(a) Formaldehyde Control

i. Absorporation of formaldehyde by water (air washing)

Formaldehyde reacts readily with water to form primarily methylene glycol (for dilute solutions). Because of this reaction, formaldehyde can be considered highly soluble in water and it may be practical to employ formaldehyde absorption in water (or aqueous solutions) as a control measure. It is often desirable to combine this absorption process with dehumidification, because the rate of emission of formaldehyde from building materials is substantially decreased with a decrease in indoor humidity. An investigation of this control technique was initiated in FY'83. Theoretical calculations were performed that indicated the feasibility of the technique. A unique test system was developed that produces airstreams (up to 250 ft^3 min flow rate) with a controlled temperature, humidity, and formaldehyde concentration. The test system includes a multipoint formaldehyde measurement system. A control device was designed and fabricated that permits the placement of various systems for air-water contact between the evaporator and condenser of a small refrigeration (dehumidification) system. To prevent saturation of the water with formaldehyde, a small amount of the water is continuously replaced with fresh water. In tests with only a relatively small amount of surface area for contact between the air and the water, the formaldehyde removal efficiency has averaged approximately 50 percent. This removal efficiency was maintained down to low inlet formaldehyde concentrations.

Evaluation of this technique will continue in FY'84 using different media for air-water contact. The impact of variables (e.g., water replacement rate and temperature) on formaldehyde removal efficiency will be determined. Consideration will be given to the use of aqueous salt solutions and chemical additives that combine in solution with formaldehyde. Such an approach might eliminate the need for a refrigeration system and for continuous water replacement.

ii. Photodecomposition of formaldehyde

Formladehyde is decomposed into hydrogen and carbon monoxide upon absorption of ultraviolet light with a wavelength between approximately 3380 and 3580 angstroms. (Absorption of shorter wavelength light results in a different and less desirable decomposition). This decomposition process could be the basis for an energy-efficient and cost-effective control technique. The amounts of carbon monoxide and hydrogen produced would be negligible (acceptable concentrations for carbon monoxide are one or two orders of magnitude greater than the amounts of carbon monoxide that would be produced). Commercially available ultraviolet lamps with filters could be used as the light source. A control device based on this principle could be designed for integration into a forced air heating or cooling system or as a portable unit. For FY'84 one or more control devices based on photodecomposition will be designed, fabricated, and evaluated experimentally (DOE).

B. DUPLICATION

Duplication

The formaldehyde IAO group did not find that agencies are duplicating each others efforts with regard to research activities. In fact, most of the work being done will be useful to more than one agency. There was considerable discussion about how the work being done by one agency could benefit another. One major bonus is that as a result of the group's meetings, discussion has been intiated on how one agency's proposed efforts can be modified to benefit another without major cost implications. For example, Bonneville Power Administration is going to do a mobile home survey, and HUD requested that when homes are chosen, homes built to HUD's standards be included. The two agencies are, in separate discussion, going to explore this possibility. CPSC's research regarding pressed wood products and a formaldehyde surface emission monitor, as another example, is of interest to HUD. The epidemiological studies being done by NCI, EPA and NIOSH will be of great use to all agencies. Methodologies were explored as to how agencies could cooperate to launch ventures; for example, including formaldehyde related monitoring, questions and/or medical examinations (e.g. patch testing) during NCHS surveys.

The only area that seems to be investigated by multiple agencies is the determination of levels of formaldehyde in mobile homes. Although the group feels all accumulated information to date is useful, since many geographical locations and climates are included, it is felt that the utility of data of this nature to be collected in the future is diminishing--and that possibly other research areas should be considered as an alternative.

C. RESEARCH NEEDS

Research Needs

1. If any National survey is to be done (such as using the National Health and Nutrition Examination Survey) which would describe the distribution of exposures to formaldehyde in U.S. homes, there needs to be a suitable, effective, and reliable monitor, i.e. to measure the level of formaldehyde in a given home.

Although various measurement devices are available, their reliability may be questionable. Also, if a national level survey is needed, the type of monitor needs to be: (a) inexpensive, (b) unobtrusive, (c) convenient, (d) easy to use by unskilled interviewer or by the subject himself, and (e) reliable.

2. Information is needed regarding the general consumer concern or awareness of indoor air pollution generally, specifically formaldehyde, and/or the perceived effectiveness of a portable indoor air cleaners. Information is needed regarding this products ability to clean household rooms and offices (as large as 14' by 18') of harmful or irritating pollutants, e.g. formaldehyde.

3. Information is desired about products, other than the portable household air cleaning equipment described in 2 above, which are specifically represented to eliminate the odor or the alleged harmfull effects of formaldehyde emissions in mobile homes. These products may be sprays or solutions which can be

applied directly to mobilie home components from which emissions are likely to occur, such as plywood paneling or particleboard shelving. Information concerning the marketing and performance of any consumer product represented as eliminating the odor or the alleged irritant effects of formaldehyde in mobile homes is also desired.

4. Need testing of air change rates (ventilation rates) and air infiltration rates in manufactured homes (HUD - Code homes).

The above are needed for present Federal standard and for energy Efficient thermal envelopes. Also needed is a simplified surface monitoring testing device and proven procedure for measuring low levels for correlating with ambient and large chamber tests (1,000 cubic feet).

5. As noted at the recent EPA sponsored Consensus Workshop on Formaldehyde, there are significant gaps in our information on the levels of formaldehyde exposure in many American industries. This information is crucial for making risk assessments and for interpreting some of the epidemiologic studies that are being performed. Additional surveys in the fertilizer production, furniture manufacture, fungicide production, wood, paper, and particleboard production industries are needed.

6. Information is needed on the relationship between ambient levels of formaldehyde and the "effective" concentration at a

target site, and the role of cytotoxicity/cellular turnover in formaldehyde carcinogenesis.

Ways of identifying human exposure with biologic tests are needed (e.g., in urine or blood). Results of exposure tests would identify persons exposed and be useful in assessing dose-response relationships and establish correlations with the exposure and nonspecific symptoms (headaches, tiredness, thirst, drowsiness, etc.). Studies should address magnitude and duration of exposures and confounding factors, such as cigarette-smoking habits, presence of other environmental pollutants, and identify any unique source generators that contribute to the burden of exposure. The development and application of simple and reliable passive monitors would be essential in helping to characterize exposure of individuals.

A practical means for identifying susceptible persons is needed to develop quantitative information on the proportion of the general population that is susceptible and the variability in responses to formaldehyde. It is not known what fractions of persons with asthma, atopic or nonatopic persons, or those with chronic obstructive lung disease constitute susceptible persons--or whether children, infants, pregnant women, older persons, or persons with specific medical conditoins (e.g., heart disease) are also susceptible.

Data on human irritation or sensitization responses to formaldehyde concentrations especially below 1 ppm are few or lacking. More information is needed to more precisely determine ranges of concentrations to which humans are exposed and to assess the risk.

Irritation Responses - most of the information on irritation has been derived from subjective complaints and a more objective means is needed for determining responses of the human eye, nose and throat to formaldehyde exposure.

Defense Mechanisms - more information showing the relationship of formaldehyde exposure and resulting effects on nasal and bronchial ciliary motility, alveolar macrophage function and other defense mechanisms are needed.

Respiratory Effects - Substantial controversy surrounds the question of whether exposure to formaldehyde is associated wiht acute or chronic changes in respiratory function. Therefore, there is a clear need for well conducted pulmonary function studies to address these questions. NIOSH's Division of Respiratory Disease Studies in Morgantown, West Virginia is considering performing a pulmonary function study of anatomy students at a local medical school. The primary objective of this study will be to assess acute changes in pulmonary function before and after formaldehyde exposure. Since these students are in anatomy class for one year, this study would also be able to assess changes in pulmonary function over this period of time. Other studies of workers with more prolonged exposures to formaldehyde are needed. Controlled inhalation studies with histamine or methacholine challenge tests are needed for assessing how formaldehyde sensitizes the airways and whether there is an immunologic or nonimmunologic basis causing the airway responses.

Dermatitis - studies are needed to evaluate risk and nature of skin reactions, whether airborne formaldehyde can cause skin reactions, and to produce better data for evaluating the risks for this route of exposure.

Long-term Effects - the effects caused by exposure to formaldehyde over continuous or long terms has not been studied to determine the mutagenic, embryotoxic, or teratogenic potential through human epidemiologic or laboratory animal studies. The observation of the mutagenic potential in a wide variety of organisms in short-term tests points to the need for new work to better ascertain the mutagenic and carcinogenic responses in mammalian germinal or somatic cells.

MEMBERS OF THE FORMALDEHYDE WORKGROUP

Name	Organization	Telephone No.
Murray S. Cohn*	CPCC	492-6994
Richard Hefter*	EPA	382-3844
Marty Kaplan	DOJ	633-1442
Jim McCollom	HUD	755-2690
Rick Menelson	HUD	755-5798
Robert M. Doyle	FTC	523-3911
Leslie Stayner	NIOSH	513/684-4481
Judy Walrath	NCI	496-9093
Myrna Aavedal	NCHS	436-7037
Rob Rothman	FRA-Portland, Or.	503-230-5489
George Ozga	OSHA	523-7081
James A. Frazier	NAS/NRC	224-2242
Stephen Byrne	TVA	615-494-9800
David Bussard	EPA	382-3988

*Co-Chair

ACRONYMS

1. BPA = Bonneville Power Administration
2. CPSC = Consumer Product Safety Commission
3. DOE = Department of Energy
4. EPA = Environmental Protection Agency
5. FTC = Federal Trade Commission
6. HHE = Health Hazard Evaluation
7. HUD = Housing and Urban Development
8. ITRI = Inhalation Toxicology Research Institute
9. LBL = Lawrence Berkeley Laboratory
10. NASA = National Aeronautics and Space Administration
11. NAS = National Acacemy of Sciences
12. NCHS = National Center for Health Statistics
13. NCI = National Cancer Institute
14. NIOSH = National Institute for Occupational Safety and Health
15. ORNL = Oak Ridge National Laboratory
16. TSCA = Toxic Substances Control Act
17. UFFI = Urea-Formaldehyde Foam Insulation
18. USDA = United States Department of Agriculture

DRAFT

ORGANICS WORKING GROUP

BRIEFING FOR
COMMITTEE ON
INDOOR AIR QUALITY

MAY 25, 1984

STATEMENT OF PURVIEW

- Included
  - Volatiles (Benzene, Chloroform)
  - Pesticides
  - PCB's
  - PAH's (Benzo-a-pyrene)
  - Nitrosamines

- Excluded
  - Formaldehyde

ORGANICS: DIMENSIONS OF THE PROBLEM

- HEALTH EFFECTS
  - ACUTE ("SICK" BUILDINGS?)
  - CHRONIC (CANCER RISK?)
- SOURCE ASSESSMENT
  - MULTITUDINOUS; UNCATALOGUED
  - EMISSIONS UNSTUDIED
- MONITORING
  - METHODS DEVELOPMENT
  - FIELD STUDIES

TABLE 1

MAJOR RECENT OR ONGOING FEDERAL STUDIES OF ORGANICS

STUDY	SPONSOR	$K
1. TOTAL (24 HOUR) EXPOSURE IN RESIDENTS OF CHEMICAL INDUSTRIAL CITIES (RTI)	EPA	3000 (3 YRS.)
2. CHARACTERIZATION OF VOCs AND PESTICIDES IN SCHOOLS, HOMES FOR THE ELDERLY AND OFFICE BUILDINGS (RTI)	EPA	850 (2 YRS.)
3. CHARACTERIZATION OF ORGANICS IN 40 HOMES (ORNL)	CPSC	
4. CHLOROCARBONS FROM CONSUMER PRODUCTS (LBL)	CPSC	
5. EMISSIONS FROM BUILDING MATERIALS, CHAMBER STUDY (LBL)	DOE	200

TABLE I (CONTINUED)

MAJOR RECENT OR ONGOING FEDERAL STUDIES OF ORGANICS

STUDY		SPONSOR	$K
6.	TERMITICIDES IN MILITARY HOUSING (VARIOUS)	DOD	1000
7.	WOODBURNING EMISSIONS IN TEST HOME	TVA	
8.	ETHYLENE OXIDE IN HOSPITALS	NIOSH	174
9.	METHODS FOR AMINES, ALDEHYDES, ISOCYANATES	NIOSH	180
10.	METHOD FOR BREATH ANALYSIS OF SOLVENTS	NIOSH	39
11.	RETROFIT STUDY OF HOMES	BPA	

RESEARCH NEEDS

- METHODS DEVELOPMENT
  - Quiet Hi-Volume Samplers
  - Microanalytical Techniques
- SOURCE ASSESSMENT
  - Cataloguing Products and Materials
  - Chamber Studies of Emissions
- MONITORING
  - Broad-Spectrum Identification
  - Quantification of Target Chemicals
- HEALTH ASSESSMENT
  - Toxicology
  - Potency
  - Mutagenicity

Membership
Organics Working Group
Committee on Indoor Air Quality

Chairman

Dr. Lance Wallace
Office of Monitoring Systems
and Quality Assurance (RD-680)
Office of Research and Development
U.S. Environmental Protection Agency
401 M. Street, S.W.,
Washington, D. C. 20460
(202) 382-5798

Co-Chair

Dr. Kailash Gupta
Health Sciences
Consumer Product Safety Commission
Room 714
5401 Westbard Avenue
Bethesda, MD 20207
(301) 492-6994

Members

Mr. Robert Rothman
Department of Energy
Bonneville Power Administration
P.O. Box 3611 - KTT
Portland, OR 97208
(503) 230-5489, FTS# 8-429-5489
FTS# 8-429-5489

Justice Manning
Tennessee Valley Authority
Knoxville, TN 37902

Members

Mr. Robert Oliver
Department of Energy -
Mail Station GH068CE-111
Room GH068
1000 Independence Avenue, S.W.
Washington, D.C. 25285
(202) 252-9819

Dr. Robert Bielguski (112B1)
Naval Facilities Engineer Command
200 Stovall Street
Alexandria, VA 22332
(202) 325-8175

(For Internal Review Only - DRAFT)

COMMITTEE ON INDOOR AIR QUALITY

WORKING GROUP ON ORGANICS

Statement of Purview

This Working Group will concern itself with monitoring methods, source assessment and health effects of organic compounds (excluding formaldehyde). Major groups of organics covered will be volatile organic compounds (VOC's); pesticides; PCB's; polyaromatic hydrocarbons (PAA's); and nitrosamines.

Recent and Ongoing Studies

The working group co-chairs have identified very few Federal research efforts on organics other than formaldehyde (Table 1). These are concentrated in two areas: (1) characterizing VOC's in homes (CPSC) and public access buildings (EPA); (2) determining termiticide levels in military housing (DOD). Several small chamber studies of emissions from building materials are supported by EPA and DOE. Two EPA exposure studies have documented high indoor levels of about a dozen VOC's.

Major Gaps

With so little ongoing work, it might be most accurate to say that the entire area of indoor organics is one large gap. However, one can distinguish subareas where nothing is known from subareas where knowledge is miniscule. In the former category, we may include:

1) Characterization of airborne insecticides and herbicides in private homes and commercial shops (especially restaurants) resulting from spraying or from storage.

2) Determination of the indoor sources and source strengths of the following VOC's, all found at higher levels indoors than out: benzene, styrene, xylenes, dichlorobenzenes, trichloroethylene, tetrachloroethylene, chloroform, 1,1,1-trichloroethane.

3) Characterization of very volatile organic vapors (i.e., vinyl chloride, methylene chloride).

4) Characterization of organics adsorbed to particulated (PAH's especially).

5) Characterization of other organic groupings, such as nitrosamines, phenols (esp. pentachlorophenol), aldehydes (other than formaldehyde).

6) Determination of partioning between vapor-phase and particle-adsorbed phase for higher molecular weight organics.

In the latter category (i.e., some knowledge exists), we may include the following areas (agencies that have done some work on the problem are indicated in parentheses):

1) Determination of possible irritants, lachrymators, etc. in office environments. (CDC).

2) Knowledge of ingredients of consumer products, building materials (CPSC)

3) Determination of temporal behavior of organics in residences and buildings from the time of construction. (EPA)

4) Characterization of termiticide levels in homes and commercial buildings.(DOD)

Recommendations:

The following recommendations from the co-chairs are put forward in the sense of inviting comment and suggestions from all members of the CIAQ.

1) Methods to collect particulates indoors in sufficient amounts to allow chemical analysis for organics, especially polyaromatic hydrocarbons, are urgently needed. Samplers should be quiet and inconspicuous so that they can be used in residential and commercial locations. Microanalytical methods should be developed that are capable of great sensitivity for very small amounts of material.

2) The recent peer review of EPA's indoor air program has recommended two major efforts to be undertaken. One is an intensive survey of a small number of "high-use" homes and commercial buildings for pesticides and VOC's to determine sources and source strengths. Such a study is the appropriate next step for selecting the important sources from a myriad of possibilities. The second is a study of source emissions, source strengths, and emission rates for the most important indoor sources. Headspace analysis and chamber testing under controlled conditions are envisioned.

3) Certain organics identified as important indoor pollutants may have inadequate toxicological information. A good example of this may be styrene, a mutagen that is decomposed rapidly outdoors but is frequently found indoors. When such chemicals are identified, toxicological studies should be carried out.

DRAFT

UNITED STATES GOVERNMENT

Memorandum

U.S. CONSUMER PRODUCT SAFETY COMMISSION

TO : David Berg, EPA

DATE: 5/1?/84

FROM : W.K. Porter, Jr., Chairman, Combustion Source Work Group

SUBJECT: Indoor Air Quality - Combustion Related Membership

The combustion work group consists of personnel from DOE, EPA, TVA, BPA, and HHS. The representatives are:

CPSC	Warren K. Porter
DOE	Dan Lin, Robert Oliver
EPA	Gene Tucker
TVA	Karen Knight
BPA	Robert Rothman
HHS	James Melius

Purview of the Combustion Sources Work Group

The work group was established to create a matrix of recent, ongoing, and future initiatives concerning combustion related sources of pollutants in the indoor environment. The matrix was envisioned to identify specific pollutants, where source strengths have been determined and identified with specific appliance sources. The sources would further be categorized in the matrix to identify exposure determinations resulting from field studies of occupied (or unoccupied) buildings. The health effects section of the matrix was envisioned to address general sources of information used for development of existing standards, both controlled human and animal laboratory studies, epidemiologic studies, and predictions from modeled projections of exposure to predicted health effects.

The matrix developed by the combustion sources work group of recent studies on combustion sources of pollutants is presented in Table I. Generally, pollutants which have been addressed are carbon monoxide (CO), carbon dioxide (CO_2), nitrogen oxides (NO_x), sulfur dioxide (SO_2), formaldehyde (HCHO), oxygen depletion, particulates, and polyaromatic hydrocarbons (PAH). A more detailed presentation of the recent efforts is given in the subsequent discussion.

The work group has identified the following areas where additional studies would be of value:

Epidemiology

- Correlation of health effects of NO_2 with respiratory infections - general population and sensitive groups.

Sources

- Source strengths and potential exposure to particulates, PAH's and nitrosated PAH's from gas appliances, and kerosene heaters.

Field Studies

- Exposure to combustion pollutants (particularly NO_2) from new technology, kerosene heaters, and gas appliances.

Health Effects

- Further controlled studies of NO_2 effects on respiratory system.
- Further definition of CO effects on cardiopulmonary system.

Use Patterns

- Defining typical, maximum, and minimum use scenarios for wood stoves.

New Initiatives

- Studies on effects of passive smoking indicate a health effects association. Whether passive smoking is under the purview of the combustion sources or should be addressed as a separate pollutant source and unique control options, require guidance from the Committee on Indoor Qir Quality Chairs.

Discussion

The attached inventory of projects for various agencies covers studies, to the present where source strengths, field monitoring, and modeling or a combination of the three have been undertaken. The inventory includes past projects as well as current projects. The inventory also includes studies known to us undertaken by industry.

Attached to the inventory is a matrix of indoor air vs. combustion source. The matrix lays out what studies provide information on health effects, use, source strengths, field studies, and new technology applicances. Blanks indicate an absence of data.

Health Effects

The potential health effects from combustion sources for kerosene heaters and unvented gas space heaters have been based on the controlled laboratory studies and epidemiologic studies, largely those used in developing the National Ambient Air Quality Standard (NAQS) of 0.05 ppm (100 ug/m^3). As a portion of the Pierce Foundation study (NIEHS), correlation of health effects with NO_2 concentrations and kerosene heater use will be made during 1984. Generally, industrial organizations have criticized the studies upon which the NAQS is based (Report to Gas Research Institute, Contract # 5080-351-0353, Roth, H.D., et al., October 1982 NO_2 and Health Effects: Analysis of Selected Studies; and Elkins, R.H. et al., November 1978, Technical Assessment of the Report "Health Effect for Short-Term Exposures to Nitrogen Dioxide" - U.S. EPA). The Roth et al. analysis addresses NO_2 exposure and health effects associated with gas ranges and ovens and not criteria for either long-term or short-term concentration standards. Elkins et al. evaluated various animal studies, epidemiologic studies, and the NAS/NRC Nitrogen Oxides Report and concluded a short-term standard of 0.60 to 1.2 ppm for one hour three to four times a year, was appropriate. It should be noted that although the SoCal gas cooking-NO_2 concentration study had proposed a health effects element, this was dropped from the final proposal. This deletion resulted, we believe, because SoCal's reviewers did not believe the proper indicies of effect were well identified. Thus, the IAQ combustion committee should consider what additional exposure-health

effects data may be appropriate, either field studies or controlled laboratory studies, to identify appropriate health indicies and to define NO_2 related health effects.

User Surveys

Projected use of unvented combustion sources has been addressed by the National Kerosene Heater Association (NKHA) and KeroSun. These independent surveys addressed hours of use, heater location, number of heaters, ventillation of the heater room, and housing construction characteristics. Range and over use have been predicted on cooking patterns projected from cooking a "typical" USDA meal. The SoCal Study should provide information on NO_2 concentration and use patterns in southern California. Use patterns for wood and coal stoves, cabinet heaters, unvented gas space heaters, and catalytic heaters have not been defined. It should be noted that cabinet heaters and catalytic heaters have not been certified for U.S. residential use.

Source Strengths

Source strengths for CO, CO_2, NO, NO_2, NO_x, SO_2 and oxygen depletion have been investigated for unvented gas space heaters (LBL 1982-1984; AGAL 1979; and GRI 1984), kerosene heaters (LBL 1983; Pierce Foundation 1982-1983; CPSC 1982-1983; Yamanaka (NO_x) 1979; and Coceres (NO_2) 1983), ranges and ovens (LBL; GRI; AGAL 1974), wood stoves (TVA/BPA 1983-1984; LBL 1983-1984), and cabinet heaters (LBL 1983-1984). It should be noted that the 1974 study by AGAL is probably the most in-depth study for gas stoves, having tested 18 ranges and ovens under both blue and yellow tipped flame conditions. However, industry indicates these data are outdated since most range/oven manufacturers ceased production of their own burners and currently buy the burners from a limited number of burner manufacturers. Other recent studies (LBL, GRI) have only screened one or two stoves.

Field Studies Other than Epidemiologic

Field studies on kerosene heaters have been performed by the Pierce Foundation (120 heater homes, 144 non-heater homes). Ritchie performed monitoring (NO_2, CO, temperature) in an unoccupied dwelling. Weston Associates, under contract to KeroSun, monitored a single family occupied residence for NO_2, NO, NO_x, CO, CO_2, and oxygen for about 1-2 months using four different kerosene heaters. The SoCal study should provide NO_2 data associated with gas cooking and a limited amount of data on CO, formaldehyde, and O_2 concentrations. The BPA study (3 yrs.) should provide data on pollutant concentrations associated with a variety of combustion sources including wood stoves (NO_2, NO, NO_x, CO, humidity and air exchange).

New Technologies

New technology kerosene and unvented gas space heaters will be investigated by CPSC (1984), LBL (1983-1984), and GRI (1984). The

extent of the testing will depend on market conditions for supplemental zone heaters and general industry success in developing low-emitting appliances. Wood stoves of air-tight construction with and without catalytic combustors have been under investigation by TVA/BPA (1983-1984).

Attachment

Combustion Related IAQ Studies:

Study (laboratory)	Pollutants	Sources	Agency
Pollutant emissions from unvented combustion sources, occupancy and tobacco odor, chamber study (Pierce Found).	CO, NO_2, SO_2, O_2 depletion, particulates, odor, VOCs, and trace elements.	All combustion appliances	NIEHS
Exposure assessment and health effects associated with supplementary heat sources, field study (Pierce Found).	CO, CO_2, NO_2, SO_2, formaldehyde, oxygen depletion.	Kerosene heaters.	NIEHS and CPSC
Pollutant emissions from unvented gas stoves, wood stoves, kerosene heaters; (LBL).	CO, NO_x, SO_2.	Unvented gas stoves, wood stoves, and kerosene heaters.	DOE
Pollutant emissions from unvented gas appliances, wood stoves, kerosene heaters, cooking and cigarette smoking, chamber study (ITRI).	CO, CO_2, NO_x, formaldehyde and others.	Unvented gas appliances, wood stoves, kerosene heaters, cooking, and cigarette smoking.	GRI
Total exposure to pollutants from unvented gas appliances, Field study (Harvard).	CO, NO_2	Unvented gas appliances	GRI
Pollutant generation from from combustion appliances (LBL).	All	Gas appliances, kerosine heaters, unvented gas space heaters.	DOE
Emissions from kerosene heater, chamber study (CPSC).	CO, CO_2, NO_2, SO_2	Kerosene heaters.	CPSC
Emissions from unvented gas space heaters, field study	CO, CO_2, NO_2, HCHO	Unvented gas	CPSC
Emissions from wood burning appliances, test homes (TVA)	CO, CO_2, NO_2, particulates	Wood burning appliances	TVA and BPB
Emissions from wood and coal burning stoves, exposure and health effects	CO, CO_2, NO_2, PAH, particulates	Wood and coal burning stoves	CPSC
Control of indoor pollutants from wood stoves (TVA)	CO, CO_2, NO_2, particulates	Wood burning stoves	TVA

	Poll	Sources	Agency
Combustion Sources Handbook	All	All	DOE
Pollutant Emmissions from unvented gas appliances, primarily space heaters (IGT). Test stand/ chamber/home correlations. (1984)	CO, NO_x, CO_2, Formaldehyde	Unvented gas space heaters	GRI
Use survey addressed to unvented gas space heaters (unclear if this will be completed in FY 84	N/A	N/A	GRI
Pollutant Emissions from unvented combustion devices (contract undecided) Identify organics/ particulates and interrelation-ship to combustion parameters. Although Bioassay was originally proposed, this will be held in abayance this year. Will include some instrument development (1984-1985)	organics CO, NO_x, SO_x, RSP	kerosene & unvented gas heaters	EPA
Exposure to NO_2, air in filtration passive monitoring. Other Other pollutants, real time monitoring on selected houses. Passive monitoring spring 1985, Real time spring 1986.	NO_2, CO, HCHO,	Gas ranges and ovens	SOCAL Gas Co.

TABLE I

MATRIX OF COMBUSTION RELATED INDOOR AIR POLLUTANTS

SOURCE	POLLUTANTS	SOURCE STRENGTHS	FIELD STUDIES*	USE PATTERNS	HEALTH EFFECTS**
Kerosene Heaters (unvented)	CO, CO_2, NO, NO_2, NO_x, SO_2, O_2 depletion.	LBL, Pierce Foundation, Weston Associates, Yamanaka, CPSC, Coceres, Ritchie.	Pierce Foundation, approx 270 homes; Ritchie (unoccupied Townhouse); Weston Associates (one occupied home)	Industries Telephone Surveys, Pierce Foundation	Health effects have been estimated from predicted pollutant concentrations from lab chamber data, and field studies applied to animal and epidemiologic data used in setting the Primary Air Quality Standard (PAQS).
Gas Space Heaters (unvented)	CO, CO_2, NO, NO_2, NO_x, O_2 depletion, HCHO, limited particulates	LBL, AGAL	LBL one unoccupied House. CPSC-1984	Gas Res. Inst. 1984.	See Kerosene Heaters
Ranges and Ovens	See Gas Space Heaters	LBL, IITRI, AGAL (1974) (LBL, IITRI data are on few ranges; AGAL tested 16 ranges but burners are probably not of current design)	CPSC-1984 May fund pilot study: Southern Calif. Gas company (SOCAL) 1984-1985.	Socal 1984-1985 and estimates based on USDA typical meal and Inst. of Gas/Technology (IGT) cooking times.	See kerosene heaters; also GRI and IGT critiques of data used in developing PAQS.
Wood Stoves	CO, CO_2, NO, NO_2, NO_x, SO_2, O_2 depletion, RSP, TSP, metals in particulates, PAH, infiltration.	TVA - Airtight catalytic and non-catalytic. TVA Non-Airtight (1984) LBL 1983-1984	LBL - 1983-1984 one house. BPA 1984-1987.	None Current	CPSC 1984 None Current.
New Technologies					
Kerosene Heaters (unvented)	See Kerosene Heaters	CPSC 1984	None	See Kerosene Heaters	Update 1984
Gas Space Heaters	See Gas Space Heaters	CPSC 1984-1985 IGT 1984	CPSC 1984-1985	See Gas Space Heaters	See Kerosene Heaters (Update 1984-1985)

* Field studies are either controlled unoccupied dwellings or in larger settings occupied dwellings. Relatively little real time monitoring is available on occupied dwellings.

** Health effects of NO_2, NO_x are limited in controlled lab testing, field studies are complicated by effects of other pollutants. CO data have been weakened to a degree with the questions raised recently on some of the work.

DRAFT

Report From Working Group on

Allergens and Pathogens

Chaired by Phil Morey, Ph.D., NIOSH

March 1, 1984

Committee on Indoor Air Quality

I. Dimensions of Problems with Allergens and Pathogens in the Indoor Environment

The extent of health problems caused by allergens and pathogens in the indoor environment is difficult to estimate. This is partly due to the broad array of arthropods, algae, protozoans, fungi, and bacteria that evoke human responses, and also to the broad range of environments in residential, commercial, and public buildings where exposure occurs. Despite uncertainty about the magnitude of the health effects caused by exposure, the impact of some allergens and pathogens is appreciable.

Outbreaks of hypersensitivity pneumonitis in both large and small office buildings are well documented. Attack rates vary from less than 1% to over 50%. Remedial measures have ranged from simply cleaning a component of the ventilation system to the total replacement of the heating and air-conditioning system together with all building furnishings (cost of over 10 million dollars in one office complex). Cases of hypersensitivity pneumonitis in residences are also well documented with the most common causes being contaminated forced air heating systems, humidifiers, and flooding disasters. Humidifiers with reservoirs containing stagnant water may be important sources of allergens in both residential and public buildings. Hypersensitivity penumonitis has also been caused by inhalation of microbial aerosols from saunas, from home tap water, and even from automobile air conditioners.

Asthma may be caused by pollens, acarids (eg., house dust mite), animal dander, fungi, and insect eminations. While the indoor environment offers some protection from pollen, the growth and proliferation of other allergens especially molds and acarids is encouraged by moisture and high relative humidity. Cold mist vaporizers may produce fungal aerosols that are potential exposure hazards to individuals with asthma. A few studies in residences have been made on the factors that are associated both with the elevation and the reduction of fungal levels in the indoor environment.

The occurrence of Legionnaire's disease both in commercial buildings and in hospitals has been widely publicized. For the most part Legionnaire's disease has been associated with the inflitration into the built environment of aerosols containing Legionella pneumophila from external sources such as cooling towers. Recently, nosocomial Legionnaire's disease has been found to be caused by aerosolization of microorganisms from shower heads. The extent of this problem in hospitals and in residences is unknown. Whether recent energy conservation measures directed at lowering the temperature of hot water services enhances the survival of pathogenic microorganisms in potable water is unknown. Pathogenic microorganisms may also be disseminated into the intramural environment by use of hot tubs (dermatitis caused by Pseudomonas spp.), whirlpools, and cold mist vaporizers and nebulizers.

See Appendix A for documentation.

II. Brief Summary of Agency Concerns or Projects Dealing With Allergens and Pathogens

(1) USA-CERL

The U.S. Army - Construction Engineering Research Laboratory (USA-CERL) in Champaign, Illinois has cooperated with NIOSH recently in preliminary experiments designed to trace pathways of movement of fungal aerosols in occupied space and in a ventilation system simulating a modern office building. Although USA-CERL has no active program on allergens and pathogens, this facility is ideal for future studies on the interaction between ventilation systems and allergens and pathogens in the built environment.

(2) CPSC

CPSC has no active programs dealing with allergens and pathogens. However CPSC does have interest in potential problems associated with the use of humidifiers in residential heating systems. Specifically, do humidifying devices act as sources of allergens in the residential environment?

(3) DOE

DOE has no formal projects in the area of allergens and pathogens. However, there is interest in learning of moisture problems caused by some energy conservation measures are associated with the build-up of allergens in the indoor environment.

(4) EPA

EPA has no formal projects dealing with problems of microbial aerosols in the indoor environment. However, the Office of Drinking Water is concerned about possible contamination of the indoor environment by Legionella aerosols originating from hot water services, and also about microbial aerosols from saunas, hot tubs, and whirlpools.

(5) HRSA, DHHS

Although the Health Resources and Services Administration (HRSA) is not extensively involved in indoor air quality research, two of its bureaus have ongoing activities relevant to the concerns of the CIAQ. The Bureau of Health Maintenance Organizations and Resources Development (BHMORD) is involved with establishing health and safety codes for maintenance of air quality in hospitals. An important component of BHMORD's activity deals with the development of ventilation standards (air change rates, filter bank efficiencies, direction of air flow, etc.) for protection of patient care areas from airborne contagion. BHMORD does not maintain any research programs in the area of indoor air quality/allergens and pathogens.

The Division of Federal Employee Occupational Health (DFEOH) of HRSA is responsible for providing health care assistance to approximately 200,000 federal employees from various agencies. DFEOH maintains a small (20K) technical assistance program, primarily for agencies in the Washington, D.C. area dealing with problems of indoor air quality in the office environment. A significant number of technical assistance consultations carried out by DFEOH have dealt with mold in the indoor environment.

(6) NHLBI, NIH, DHHS

The National Heat, Lung and Blood Institute (NHLBI) of NIH through its grant program administered by the Division of Lung Diseases supports research on the etiology, immunoregulation, and clinical evaluation of hypersensitivity pneumonitis. FY83 support was about 150K (3 grants; see appendix B).

(7) NIAID, NIH, DHHS

The National Institute of Allergy and Infectious Diseases (NIAID) supports a considerable amount of research dealing with building associated allergens and pathogens. Twelve projects dealing with studies on molds, actinomycetes, algae, animal dander, house dust mites, etc., are supported (FY83=916K) by NIAID's Allergy and Clinical Immunology Branch. NIAID also maintains a substantial grant program (FY83, 8 projects, 820K) on Legionella pneumophila, the pathogenic agent causing Legionnaire's disease. Abstracts of NIAID projects on allergens and Legionella are found in Appendix C.

(8) NIOSH, CDC, DHHS

The Division of Respiratory Disease Studies (DRDS) of NIOSH carries out several activities that relate to allergens/pathogens and indoor air quality. These include research projects, health hazard investigations in office buildings, and internal documents.

In FY82 NIOSH and USA-CERL collaborated on preliminary studies dealing with the effect of ventilation system operational parameters on airborne fungal levels in simulated occupied space (see Appendix D). DRDS has been particularly active in carrying out health hazard evaluations in office buildings where allergens are likely sources of complaints. During the past 3 years approximately 10 hazard evaluations have been undertaken where collection of airborne microorganisms have been an important part of the environmental sampling protocol (see Appendix D). Each building hazard evaluation conservatively requires 0.2 person years of time, and costs approximately 5K.

Three internal documents pertinent to the interests of the Working Group are: a draft Morbidity Mortality Weekly Report publication, "Hypersensitivity Pneumonitis in Office Buildings: Engineering Preventive Measures," the draft report, "Interim Report on Environmental Techniques Needed to Sample for the Variety of Biological Agents that may Cause Hypersensitivity Pneumonitis," and the transcript of a talk on "NIOSH Industrial Hygiene Studies in Moldy Office Buildings" (see Appendix D).

III. Areas for Research, Cooperation, and Coordination

It was the consensus of the Working Group, that while there is considerable interest in the presence of allergens and pathogens in the built environment, current research efforts are minimal and, as such, no overlap exists. Areas that are not being addressed and where cooperation and coordination could be beneficial are listed as follows (no priority intended):

(1) Standards and Engineering Preventive Measures

The American Society of Heating, Refrigerating, and Air-Conditioning Engineers (ASHRAE) Standard 62-1981 recommends ventilation rates and defines contaminant levels in conditioned air that will be acceptable to human occupants and will not impair health. Although microorganisms are recognized as air contaminants, ASHRAE makes no recommendations on measurement techniques, acceptable levels, and means that can be employed to remove contaminants. A document similar to ASHRAE 62-1981 should be developed specifically for allergens/pathogens with emphasis on remedial measures needed to prevent contamination in the built environment.

Studies need to be conducted to quantify the dispersal and dissemination of allergens and pathogens within building ventilation systems. The effect of filter efficiency, air flow rate, temperature, humidity, and other operating parameters on biological agents in the built environment needs to be quantified. The USA-CERL facility provides an excellent field laboratory to address these needs. Testing performed in this facility does not require occupant disruption and all relevant ventilation parameters can be carefully controlled and monitored. The results of this study will help develop guidelines for standards and engineering preventive measures for dealing with allergens and pathogens in the built environment (see Appendix E).

Agencies involved: EPA, USA-CERL, NIOSH, National Bureau of Standards

(2) Laboratory Studies

There exists a need to develop laboratory methodologies to aid in the study of allergens and pathogens in indoor air/environmental problems. Techniques to quantitatively sample and identify microorganisms in air and to identify the source of those organisms are needed. Such techniques are needed to monitor changes in microbial populations during the course of the day, during changes in the operation of heating and ventilation systems, and during seasonal or environmental changes. In particular, data is needed to establish dose-response relationships, and technologies need to be developed with this goal in mind. In addition, laboratory techniques are needed that can aid in the diagnosis of building associated illness, as well as in definition of the mechanism of disease (see Appendix E). Agencies that could be involved include NIAID, CDC, USA-CERL, NIOSH, the National Institute of Environmental Health Sciences, and the Food and Drug Administration.

(3) Basic Studies on Moisture and Microorganisms

The survival and growth of microorganisms (bacteria and fungi) in indoor environments require water to be present. Thus, devices which intentionally or unintentionally are used to increase the humidity of air in an enclosed environment can be a potential source of microorganisms which can cause infection (e.g., Legionella pneumophilia) or more commonly allergic reactions (e.g., molds). In addition, microbial products (e.g., endotoxin) can produce an inflammatory reaction when inhaled. Some examples of equipment used to increase the moisture content of air are obvious, such as humidifiers ranging in size from cool mist vaporizers used in a room to units that are an integral part of the ventilation system of large buildings. Less obvious sources of moisture include shower heads, indoor saunas and swimming pools, fountains and stagnant pools of water in poorly maintained ventilation systems. Research is needed to define the prevalence of microorganisms in these sources as well as corrective measures that can be used to prevent microbial growth accumulation. Agencies that might be affected or could contribute to these studies include EPA, CDC, NIOSH, CPSC, FDA, NIAID, NHLBI, and USA-CERL.

(4) Environmental Epidemiology in Non-Problem and Problem Buildings

Normal buildings and residences where occupants have no complaints need to be surveyed to characterize the levels and types of allergens (pollen, house dust mites, viable and non-viable molds, actinomycetes, etc.) found in these environments. In order to allow for statistically valid conclusions and for control of different modes of ventilation system operation, these surveys need to be conducted in a large number of buildings and residences, during both the winter and summer, and for several years. Similar studies need to be carried out in problem buildings and residences with documentation of the effectiveness of control efforts. Such information would greatly assist in the development of public health policy (including research directions) regarding biological agents in the built environment.

Agencies involved: CDC, NIOSH, CPSC EPA, NIAID, HRSA.

(5) Medical Epidemiology and Diagnostic Testing

A questionnaire needs to be developed that can distinguish between the mixture of complaints present in the indoor environment. The questionnaire should at a minimum distinguish between complaints arising from the following: temperature and humidity problems; insufficient outdoor air; cigarette smoke in the environment; problems associated with the use of video-display terminals; and airborne allergens. Computer program software to expedite the analysis and interpretation of the questionnaire is needed.

Medical diagnostic techniques need to be developed to better sort out the etiology of building-associated complaints. Some complaints such as sneezing, eye irritation, sinus congestion, and lingering colds are not sufficiently severe so as to cause the building occupant to consult a physician. It is presently difficult to decide if complaints are work-related or are due to some other cause.

Much of the acute morbidity relating to airborne allergens deals with subtle health effects. To better understand this morbidity and its relation to dose, responses of human volunteers should be studied under controlled situations. Such a study requires sophisticated laboratory facilities, careful selection of subjects, careful selection of allergens (e.g., agents that may cause hypersensitivity pneumonitis), proper exposure durations and concentrations, and careful analysis of response parameters. The resulting information can be very relevant to the standards setting processing.

Agencies involed: NIAID, NHLBI, FDA.

(6) Energy Conservation and Biological Agents

The national requirement for energy conservation in buildings has preceeded the evaluation of effects of conservation on building occupants. Concern is now only being raised regarding the somewhat sporadic reports of serious allergen/pathogen exposures in these energy efficient structures. Objective and prospective research into the effects of allergens and pathogens must be done before increasing numbers of newer structures are built.

Agencies involved: DOE, USA-CERL, HRSA, EPA, General Services Administration.

(7) LIAISON MECHANISM

On a national level, the need for a liaison mechanism to link those with allergen/pathogen concerns and those with allergen/pathogen solutions and research expertise is urgent. Inquiries from the state and local level must be properly directed, documented, and addressed. Concurrently, private sector research into allergen/pathogen problems must be identified, disseminated, and exploited. The best mechanism to achieve this is intentionally not recommended here. It may be that professional societies and contacts as elsewhere in the building industry (ASHRAE, National Fire Protection Association) could best perform this task.

IV. Allergens/Pathogens Working Group

*Richard E. Rundus — FTS 958-7571
U.S. Army - CERL - DOD
Box 4005
Champaign, IL 61820

Kailash Gupta — FTS 492-6994
CPSC
5401 West Bard Ave.
Bethesda, MD 20207

*Susan Rose — FTS 233-4731
US - DOE
Office of Environmental Research
ER 73
Washington, DC 20545

Paul Berger — FTS 382-7585
US - EPA
Office of Drinking Water WH550
Headquarters Bldg.
401 M Street, SW
Washington, DC 20460

Barry Davis — FTS 236-3408
Center for Infectious Diseases, CDC
Bldg. #1, Room 5044
Atlanta, GA 30333

Victor Avitto — FTS 443-5405
HRSA, Div. Fed. Emp. Occ. Health
Room 7829
5600 Fishers Lane
Rockville, MD 20857

*Grady Smith — FTS 443-6846
HRSA, BHMORD, Div. Conversion & Utilization
11A-19 Parklawn Bldg.
5600 Fishers Lane
Rockville, MD 20857

John Nutter — FTS 496-6752
National Institute of Allergy and Infectious Diseases
Bldg. 31, Room 7A19, NIH
Bethesda, MD 20205

William Sanslone — FTS 496-7208
Westwood Bldg., Room 6A16,
Division of Lung Diseases
National Heart, Lung, & Blood Institute, NIH
Bethesda, MD 20205

*Phil Morey — FTS 923-4421 (301-291-4421)
NIOSH - CDC
Environmental Investigations Branch
944 Chestnut Ridge Rd.
Morgantown, WV 26505

*Tom Richards — FTS 923-4755
NIOSH - CDC
Clinical Investigations Branch
944 Chestnut Ridge Rd.
Morgantown, WV 26505

*Dan Lewis — FTS 923-4256
NIOSH - CDC
Laboratory Investigations Branch
944 Chestnut Ridge Rd.
Morgantown, WV 26505

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*Participants in February 15 meeting.

DRAFT

STRUCTURE CHARACTERISTICS

WORKING GROUP

CIAQ
STRUCTURE CHARACTERISTICS
WORKING GROUP

ROBERT OLIVER, CHAIRPERSON

APRIL 11, 1984

STRUCTURE CHARACTERISTICS

WORKING GROUP PURVIEW

THE PURVIEW OF THIS WORKING GROUP ENCOMPASSES THOSE ASPECTS OF STRUCTURES IN THE INDOOR AIR QUALITY IMPACTS OF WHICH ARE NOT POLLUTANT SPECIFIC. THESE ASPECTS INCLUDE AIR INFILTRATION, VENTILATION, DISTRIBUTION, MOVEMENT, AND MIXING. NON-SPECIFIC MITIGATION MEASURES SUCH AS AIR-TO-AIR HEAT EXCHANGERS AND AIR WASHING DEVICES ARE ALSO INCLUDED. RESEARCH ACTIVITIES OF INTEREST WITHIN THIS PURVIEW INCLUDE MATHEMATICAL MODELING AND COMPUTER SIMULATION DEVELOPMENT, AND DEVELOPMENT OF INSTRUMENTATION AND MEASUREMENT TECHNIQUES.

STRUCTURE CHARACTERISTICS
IMPACTS ON INDOOR AIR QUALITY

- DIMENSIONS OF PROBLEM
- CURRENT FEDERAL ACTIVITY
- APPROPRIATE AREAS FOR FURTHER RESEARCH, COORDINATION, AND COOPERATION

BUILDINGS

- RESIDENTIAL - 80 MILLION UNITS - 100 BILLION SQ-FT
- NON-RESIDENTIAL - 4 MILLION UNITS - 50 BILLION SQ-FT

VEHICLES

- LAND
  - SURFACE
  - SUBSURFACE
- WATER
  - SURFACE
  - SUBSURFACE
- AIR
- SPACE
  - EARTH ORBITAL
  - INTERPLANETARY

OTHER STRUCTURE TYPES

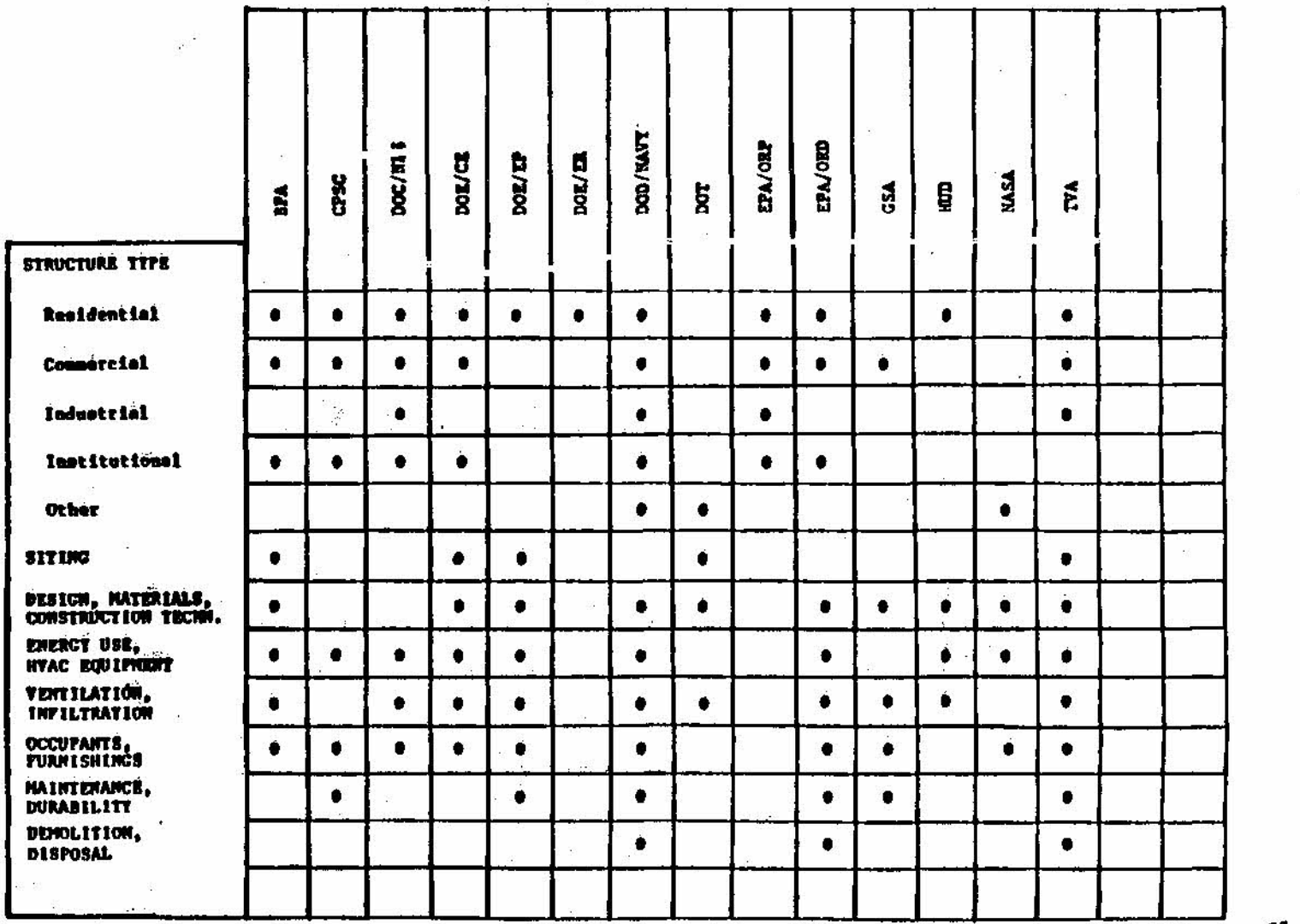
- TUNNELS
- MISSILE SILO FACILITIES
- OTHER

BUILDING LIFE CYCLE

- SITING
- DESIGN
- CONSTRUCTION
- OCCUPANCY AND USE
- REHABILITATION
- DEMOLITION AND DISPOSAL

OCCUPANCY AND USE

- HEAT SOURCES
- EQUIPMENT OPERATION
- VENTILATION
- INFILTRATION
- FURNISHINGS
- OCCUPANT ACTIVITIES
- MAINTENANCE



STRUCTURE TYPE	EPA	CPSC	DOC/NIST	DOE/CE	DOE/EP	DOE/ER	DOD/NAVY	DOT	EPA/ORP	EPA/ORD	GSA	HUD	NASA	TVA		
Residential	•	•	•	•	•	•	•		•	•		•		•		
Commercial	•	•	•	•			•		•	•	•			•		
Industrial			•				•		•					•		
Institutional	•	•	•	•			•		•	•						
Other							•	•					•			
SITING	•			•	•			•						•		
DESIGN, MATERIALS, CONSTRUCTION TECHN.	•			•	•		•	•		•	•	•	•	•		
ENERGY USE, HVAC EQUIPMENT	•	•	•	•	•		•			•		•	•	•		
VENTILATION, INFILTRATION	•		•	•	•		•	•		•	•	•		•		
OCCUPANTS, FURNISHINGS	•	•	•	•	•		•			•	•		•	•		
MAINTENANCE, DURABILITY		•			•		•			•	•			•		
DEMOLITION, DISPOSAL							•			•				•		

1-20-89

CURRENT RESEARCH ACTIVITY AREAS

FURTHER RESEARCH NEEDS

- VENTILATION EFFICIENCY
- ZONAL BALANCE
- INFILTRATION IN COMMERCIAL BUILDINGS
- SITING EFFECTS
- INDOOR/OUTDOOR AIR QUALITY RELATIONSHIPS
- COMFORT ASPECTS OF AIR QUALITY
- CHARACTERIZATION
- MEASUREMENT/MONITORING
  - METHODS
  - INSTRUMENTATION
- MODELING, COMPUTER SIMULATION
- STANDARDS, GUIDELINES

Federal Research Activities

Projects	Pollutants or Variables	Sources	Agency & Contact
IAQ Data Analysis	Radon, Formaldehyde, Particulates, Organic Pollutants, Carbon Monoxide Ventilation, Infiltration Occupant Activities, Pollutant Concentrations	Materials Emissions Furnishings, Combustion Devices, Ground TBD	EPA/ORD EMSL/RTP Steve Bromberg
IAQ in Public Access Buildings	RN, HCHO, CO, Particulates, Organic Compounds in New and Old Hospital, Home for Elderly, Schools, to Office Building	Furnishings, Materials Emissions, Combustion Devices, Ground TBD	EPA/ORD EMSL/RTP Steve Bromberg
Pilot IAQ Monitoring	RN, HCHO CO, Particulates, Organic Compounds, Ventilation, Infiltration, Occupant Activities, in Residences	Materials Emissions Combustion Devices, Ground	EPA/ORD EMS/RTP Steve Bromberg
Source Characterization	HCHO, Pesticides, Other Organics, NO_2 Chamber Studies Temperature, Humidity, Ventilation, Air Change Rates, UV Light, Interactive Pollutants	Building Materials, Consumer Products, Furnishings, Maintenance	EPA/ORD IERL/RTP Steve Bromberg
Source Characterization	CO, O_2 Particulates, Organics Laboratory Tests	Unvented Kerosene Space Heaters	EPA/ORD IERL/RTP Steve Bromberg
Rn from Domestic Water	Rn, Weather, Ventilation, Infiltration	Domestic Water	EPA/ORD EMSL/LV Steve Bromberg
Wood Heating Studies	Combustion Products, Particulates (TSP, RSP), Organics	Residential Wood Heating Devices	TVA Myron Iwanski
IAQ Booklet	Rn, HCHO, Combustion Products Organics, Molds, Construction Techniques Ventialation, Infiltration Occupants, Furnishings Demolition, Disposal	Residential Materials, Groundwater	TVA Myron Iwanski

Projects	Pollutants or Variables	Sources	Agency & Contact
Evaluation of Study of 40 Residences in Tennessee	Rn, HCHO, Particulates, Combustion Products, Organics, Meterology, House Characteristics, Weatherization	Materials Emissions, Groundwater, Combustion Devices	TVA Myron Iwanski
Environmental Information Handbook	Combustion Products in Residences	Indoor Combustion Devices	DOE/EP Dave Moses
IAQ Hanbook (Completed in 1982)	Combustion Products, HCHO, Rn, Organics, Siting, Design, Materials, Construction Techniques, HVAC Equipment, Ventilation, Infiltration	Furnishings, Combustion Devices, Groundwater, Materials Emissions	DOE/EP Dave Moses
Retrofit Effects on IAQ	CO, Rn, HCHO, Particulates, Water Vapor, Nitrogen Oxides, Infiltration	Materials Emissions, Combustion Devices, Soil and Groundwater, TBD	BPA Robert Rothman
Commercial/Institutional Buildings IAQ	HVAC Retrofit Measures in Offices, Schools, Restaurants, Libraries Rn, HCHO, Particulates, Water Vapor, Carbon Dioxide, CO, Ventilation Rates	Materials Emissions, Combustion Devices, Soil and Groundwater, TBD	BPA Robert Rothman
Fan Mitigation Study	Fan Power, Infiltration, Intermittent Controls, Meteorology Effects in Residences CO, Rn, HCHO, Particulates, Water Vapor, Nitrogen Oxides	Materials Emissions, Particulates, Soil and Groundwater, TBD	BPA Robert Rothman
Ordinary/Energy Efficient Homes Comparison	CO, Rn, HCHO, Particulates, Water Vapor, Nitrogen Oxides, Infiltration	Combustion Devices, Soil and Groundwater, Materials Emissions, TBD	BPA Robert Rothman
Tracer Gas Techniques	Computer Modeling and Field Measurements of Ventilation and Infiltration in Multi-Zone Residential and Commercial Buildings	Various	NBS Richard Grot
Measurement Equipment and Techniques	Standardization of Techniques and Equipment for IAQ Measurements	Various	NBS Preston McNall

April 11, 1984

INTERAGENCY COMMITTEE ON INDOOR AIR QUALITY
STRUCTURE CHARACTERISTICS WORKING GROUP

1. David Eakin
   Government Services Administration
   18th and F Street, N.W.
   Room 3046
   Washington, DC 20405
   (202) 566-1726

2. Pat Gerry
   EP-332, MS 3F-056
   US Department of Energy
   1000 Independence Avenue, S.W.
   Washington, DC 20585
   (202) 252-2061

3. Ray Gilbert
   Code LGT
   NASA Headquarters
   Washington, DC 20546
   (202) 453-8415

4. Howard Jongedyk
   U.S. Department of Transportation
   Construction, Maintenance & Environmental Design
   HNR-30, Rm. 115 TFHRS
   400 7th Street, SW
   Washington, DC 20590

5. Jerome P. Harper
   Assistant to the Chief
   Planning and Communication Staff
   Tennessee Valley Authority
   403 Power Broad Building
   Chattanooga, TN 37401

7. Tim Krohe
Office of Radiation Programs
ANR-460
U.S. Environmental Protection Agency
401 M Street, S.W.
Washington, DC 20460
(202) 557-8224

8. Ernest C. Lory
Materials Science Division, Code L52
Naval Civil Engineering Laboratory
Port Hueneme, CA 93043
(805) 982-5555 FTS 799-5555

9. Dr. Preston McNall
U.S. National Bureau of Standards
Building 226, Room 218
Washington, DC 20234
(301) 921-3447

10. David Moses
EP-332, MS 3F-056
US Department of Energy
1000 Independence Avenue, SW
Washington, DC 20585
(202) 252-2061

11. Robert Oliver
CE-111.1, MS GF-253
US Department of Energy
1000 Independence Avenue, SW
Washington, DC 20585
(202) 252-9187

12. David Pellish
CE-312, MS 5H-047
US Department of Energy
1000 Independence Avenue, SW
Washington, DC 20585
(202) 252-8110

13. Rob Rothman
Bonneville Power Administration
P.O. Box 3621
Portland, OR 97208
FTS 429-5189

14. David C. Sanchez
IERL
US Environmental Protection Agency
Environmental Research Center
Research Triangle Park, NC 27711
FTS 629-2979

15. Hosham Tejuja
US Department of Housing
and Urban Development
Room 3238
451 7th Street, S.W.
Washington, DC 20410
(202) 775-5798

16. Paul White
US Consumer Products Safety Commission
5401 West Bard Avenue
Washington, DC 20207
(202) 492-5957

17. Dr. B. C. Wolverton
Chief, Environmental Assurance Branch
US National Aeronautics and Space Administration
National Space Technology Laboratories
NSTL, Mississippi 39529
FTS 494-3155

FIELD STUDIES SUBCOMMITTEE

DRAFT

Chaired by James M. Melius, M.D., NIOSH

This subcommittee of the CIAQ is mainly concerned with the design and coordination (where possible) of large scale field studies of indoor air quality. Initially, the subcommittee will collect information on past and current indoor air quality field studies. Information on both governmental and non-governmental studies will be collected. These studies will be reviewed focusing on the scope of the studies, the methodologies used, overlap with other studies, and compatibility for merger as a large data source. This review will be presented to the full CIAQ in April. This will also require coordination with other CIAQ subcommittees who may have recommendations relevent to the field studies subcommittee.

Possible areas for interagency cooperation have already been identified:

1. Both Department of Energy and EPA have FY'84 funds for initiating large field studies on indoor air pollution. This funding is chiefly for planning such studies. This provides an obvious opportunity for coordination through the CIAQ and this subcommittee.

2. The National Center for Health Statistics which conducts several types of national health surveys (Health Interview Survey, Health and Nutrition Examination Survey) is interested in utilizing their surveys for assessing indoor air quality related problems. Their large field medical survey

(HANES) is currently in the planning stages. An initial meeting was held on January 27 with NCHS, EPA, and the subcommittee chairman to discuss possible collaborative efforts on an indoor air quality survey. Further meetings involving other CIAQ participants are planned.

The lack of a national data base indicating the scope distribution, and impact of indoor air quality problems is clearly a major research gap. Developing such a data base will require substantial effort over several years. Interagency field survey coordination and collaboration through the CIAQ could make a major contribution to developing this data base.

Initial Report of the CIAQ Subcommittee on Field Studies

May 1984

Introduction:

This subcommittee of the CIAQ is mainly concerned with the design and coordination (where possible) of large scale field studies of indoor air quality. This initial report includes information on past and current indoor air quality field studies and recommendations for further efforts in this area. This report and recommendations need to be reviewed in the context of other subcommittee reports in regard to overlapping recommendations and for coordination of field studies efforts.

Current and Past Studies:

The following is a list of indoor air quality field studies looking at multiple pollutants. Large studies focusing on a single exposure have not been included. Most of the studies listed are governmental although some non-governmental studies have been included.

1. Forty Home Monitoring Study (CPSC): Forty homes in the Oak Ridge, Tennessee area were monitored over a 9-month period, for CO, NO_2, formaldehyde, particulates, and volatile organic vapors, along with air exchange rates and meteorological data. Ten of the homes utilized combustion devices as a supplementary heat source. A draft report on this study has recently been released.

2. Building Materials Emissions (DOE/LBL): Although not a field study, this study of organic emissions from building materials will be important in identifying important emissions and appropriate analytical methodology.

3. Indoor Air Quality Data Base (DOE/LBL): This study is designed to compile existing field measurement studies data for the development of a data base which will eventually be accessible to outside groups.

4. Bonneville Power Authority Studies (BPA): BPA has conducted a number of field studies examining exposures to various pollutants in relation to "energy efficiency" and weatherization.

5. Analytical Methods Development (NIOSH): NIOSH is currently working on the development of a number of analytical methods relevant to indoor air quality including: formaldehyde, isocyanates, ozone, and amines. In addition, some work is being done on diffusive samplers and on direct-reading instruments.

6. Study of the effects of sulfur dioxide and respirable particulates on health (NIEHS/Harvard): This large study funded by NIEHS is examining the relationship between respiratory disease and air pollution in six cities. This includes indoor and outdoor monitoring of sulfur dioxide, nitrogen oxides, and respirable particulates.

7. TVA Studies (TVA): TVA is conducting studies focusing mainly on the effects of weatherization and the use of wood stoves in residential buildings.

8. Monitoring Study (EPA/RTI): This study is monitoring office buildings, homes for the elderly, schools, and hospitals for several pollutants.

9. Residential and Commercial IAQ Study (Niagara Mohawk): This study is monitoring buildings, including 40 homes for multiple pollutants.

10. Weatherization and IAQ (U. of Wisconsin): This study is examining 50 homes for mulliple pollutants in relation to weatherization.

11. Development and field evaluation of passive samples (DOE/LBL): This project is developing and evaluating passive samplers for CO, formaldehyde, and particulates.

12. Residential pollutants and ventialtions (Batelle/Pacific Power and Light): Three pairs of homes are being evaluated for multiple pollutants.

13. Total Exposure Study (EPA/RTS): This study is evaluating 24-hour exposure to organics for residents of chemical-industrial cities. A report has recently been released.

Summary of Current Research:

A number of the current research projects outlined above will contribute to our understanding of indoor air exposures. However, very few of these studies have any health assessment component. In addition, the ability to generalize from these studies may be limited by difference in sampling methodology and by the type of building included in the studies.

Recommendations:

Both the 1980 GAO report and the 1980 draft Strategy Plan of the Interagency Research Group on Indoor Air Quality recognized the need for a more comprehensive approach to indoor air quality including studies of multiple indoor air exposures and accompanying health studies.

This can be accomplished in three ways:

1. Better coordination and collaboration on current and planned IAQ field studies. This includes coordination or field methodologies and sampling and analytical methods. Where possible, collaborative efforts may expand studies focusing on a single pollutant to include other pollutants and/or appropriate health studies.

2. Compilation of a data base on indoor air exposures. The current DOE/LBL effort should continue. This data base will be quite valuable in assessing exposures and related parameters and in planning further studies.

3. Development of a national IAQ field study including an assessment of health effects. Both DOE and EPA have initiated the planning for such a study. Coordination with planned NCHS studies (either the Health and Nutrition Examination Survey and/or the Health Interview Survey) is already underway for a possible joint DOE/EPA/NCHS study.

Further Activities of this Subcommittee:

1. Involvement in the planning of the NCHS/DOE/EPA Study: This subcommittee will function as part of the planning process for this study including the review of the study design, methodologies, etc. This will allow input from the various CIAQ agencies into this process.

2. Coordination of other field studies. Although considerable interagency review and assistance has already taken place concerning planning studies, this subcommittee could help to involve groups not already included in these reviews and help to coordinate these studies to make optional use of planned field studies.

RECEIVED
OFFICE OF THE
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JUL -8 1 17 PM '86
CONSUMER PRODUCT
SAFETY COMMISSION

UNITED STATES GOVERNMENT

Memorandum

U.S. CONSUMER PRODUCT
SAFETY COMMISSION
WASHINGTON, D.C. 20207

TO: The Commission
THROUGH: Sadye Dunn, Secretary
THROUGH: Daniel Levinson, General Counsel
THROUGH: Leonard DeFiore, Executive Director
THROUGH: Harry I. Cohen, Director, OPM

JUL - 3 1986

FROM: Sandra Eberle, Program Manager
Chemical Hazards Program

SUBJECT: Report of the Clean Air Scientific Advisory Committee

In September, 1985, the Clean Air Scientific Advisory Committee of the U.S. Environmental Protection Agency met, and, at the request of the CPSC, reviewed the existing scientific information on the potential health hazards associated with exposure to nitrogen dioxide from combustion appliances.

The peer review was requested because voluntary standards efforts were stalemated by disagreements between CPSC staff and industry over what level of nitrogen dioxide exposure the voluntary standards should seek to prevent.

The attached report provides both a peer review of the CPSC health hazard evaluation and the requested scientific advice on what levels of NO_2 exposure present a public health concern.

The CASAC has concluded that:

- repeated peak exposures at concentrations of 0.3 ppm of nitrogen dioxide may cause health effects in some individuals and there is a possibility that such effects may occur at concentrations as low as 0.1 ppm. It is noted, however, that both the epidemiological and chamber studies at or near this range of concentrations have produced inconsistent evidence regarding the health effects of such exposures.

- The population groups that appear most sensitive to nitrogen dioxide exposure include children, chronic bronchitics, asthmatics, and individuals with emphysema; and

- the most direct evidence regarding lung damage associated with nitrogen dioxide is obtained from animal studies - such studies conclude that a number of effects occur in a variety of animal species, many of which can be considered serious and irreversible. The relevance of these studies to human exposure at concentrations found indoors is uncertain.

NOTE: This document has not been reviewed or accepted by the Commission.
Initial _____ Date 7/2/86

CPSA 6 (b)(1) Cleared
7/2/86
No Mfrs/Prvtlblrs or Products Identified
Excepted by
Firms Notified

I believe the support and advice of these highly respected scientists will assist all parties to the voluntary standards process in achieving consensus on emissions standards for unvented appliances. I will be distributing copies of the report to the community interested in our activities on combustion appliances. Progress on the voluntary standards will be discussed in the quarterly reports from Doug Noble.

Attachment

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

May 9, 1986

OFFICE OF
THE ADMINISTRATOR

Honorable Carol Dawson
Acting Chairman
U.S. Consumer Product Safety Commission
Washington, DC 20207

Dear Ms. Dawson:

The Clean Air Scientific Advisory Committee (CASAC) of the U.S. Environmental Protection Agency has completed its review of the health effects and exposure assessment documents on nitrogen dioxide provided by the U.S. Consumer Product Safety Commission. This review was conducted at the Commission's request in order to obtain independent outside scientific advice on the potential health hazards associated with exposure to 0.1 to 1.0 ppm nitrogen dioxide generated by unvented indoor combustion sources. We are pleased to transmit to you the enclosed report which represents the Committee's analysis and recommendations concerning the documents and the specific questions that you raised.

The Committee has concluded that: 1) repeated peak exposures at concentrations of 0.3 ppm of nitrogen dioxide may cause health effects in some individuals and there is a possibility that such effects may occur at concentrations as low as 0.1 ppm. We note, however, that both the epidemiological and chamber studies at or near this range of concentrations have produced inconsistent evidence regarding the health effects of such exposures; 2) the population groups that appear most sensitive to nitrogen dioxide exposure include children, chronic bronchitics, asthmatics, and individuals with emphysema; and 3) the most direct evidence regarding lung damage associated with nitrogen dioxide is obtained from animal studies - such studies conclude that a number of effects occur in a variety of animal species, many of which can be considered serious and irreversible. The relevance of these studies to human exposure at concentrations found indoors is uncertain.

The Committee also addressed the adequacy of the CPSC documents as a basis for assessing the risks of exposure to nitrogen dioxide emissions, and provided guidance regarding further efforts to assess the risks associated with indoor use of appliances producing nitrogen dioxide emissions. We found that the CPSC documents addressed the appropriate issues, but that they were repetitive and not well integrated. Perhaps this was reflective of their being prepared by various authors at different times for different purposes. We recommend that the CPSC utilize more fully the EPA Criteria Document and Staff Paper on Nitrogen Dioxide as primary resources in developing an assessment of the health risks of indoor combustion sources.

The Committee appreciates this unique opportunity to interact with the Commission and to provide scientific advice on an issue of current interest and great importance to us all.

Sincerely,

James H. Ware, Chairman
Review Panel on Nitrogen Dioxide
Science Advisory Board

Morton Lippmann, Chairman
Clean Air Scientific Advisory Committee
Science Advisory Board

cc: Lee Thomas
A. James Barnes
Don Ehreth
Craig Potter
Peter Preuss
Terry Yosie

A REVIEW OF THE CONSUMER PRODUCT SAFETY COMMISSION'S HEALTH EFFECTS AND EXPOSURE ASSESSMENT DOCUMENTS ON NITROGEN DIOXIDE

A Report by the Clean Air Scientific Advisory Committee of the U.S. Environmental Protection Agency

Science Advisory Board
U.S. Environmental Protection Agency
Washington, DC

May 1986

NOTICE

This report has been written as part of the activities of the Environmental Protection Agency's Congressionally established Science Advisory Board, a public group providing advice on scientific issues. The Board is structured to provide a balanced, independent, expert assessment of the scientific issues it reviews. The contents of this report do not necessarily represent the views and policies of the U.S. Environmental Protection Agency, the U.S. Consumer Product Safety Commission nor of other agencies in the Executive Branch of the Federal government.

U.S. Environmental Protection Agency
Science Advisory Board
Clean Air Scientific Advisory Committee

Panel for the Review of Nitrogen Dioxide Health Effects and Exposure Assessment Documents for the Consumer Product Safety Commission

Chairman

Dr. James Ware, Department of Biostatistics, Harvard School of Public Health, Boston, Massachusetts

Panel Members

Dr. Edward Crandall, Division of Pulmonary Medicine, Cornell Medical College, New York, New York

Dr. Robert Frank, Department of Environmental and Health Sciences, School of Hygiene and Public Health, Johns Hopkins University, Baltimore, Maryland

Dr. Jack Hackney, Environmental Health Laboratories, Rancho Los Amigos Hospital, University of Southern California, Downey, California

Dr. Ian T.T. Higgins, American Health Foundation, New York, New York

Dr. Timothy Larson, Department of Civil Engineering, University of Washington, Seattle, Washington

Dr. Brian Leaderer, Pierce Laboratory, Yale University, New Haven, Connecticut

Dr. Mark Utell, Pulmonary and Critical Care Unit, University of Rochester Medical Center, Rochester, New York

Dr. James Whittenberger, Southern Occupational Health Center, University of California, Irvine, California

Executive Secretary

Mr. A. Robert Flaak, Science Advisory Board, U.S. Environmental Protection Agency, Washington, DC

U.S. Environmental Protection Agency
Science Advisory Board

Clean Air Scientific Advisory Committee

Chairman

Dr. Morton Lippmann, Professor, Department of Environmental Medicine, New York University Medical Center, Tuxedo, New York

Members

Dr. Robert Frank, Professor of Environmental Health Sciences, Johns Hopkins School of Hygiene and Public Health, Baltimore, Maryland

Dr. Warren B. Johnson, Director, Atmospheric Science Center, SRI International, Menlo Park, California

Dr. Paul Kotin, Adjunct Professor of Pathology, University of Colorado Medical School, Denver, Colorado

Dr. James H. Ware, Associate Professor, Department of Biostatistics, Harvard School of Public Health, Boston, Massachusetts

Dr. Jerry Wesolowski, Chief, Air and Industrial Hygiene Lab, California Department of Health, Berkeley, California

Executive Secretary

Mr. A. Robert Flaak, Environmental Scientist, Science Advisory Board, U.S. Environmental Protection Agency, Washington, DC

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1. EXECUTIVE SUMMARY

This is the report of the U.S. Environmental Protection Agency's (EPA) Congressionally established Clean Air Scientific Advisory Committee (CASAC) concerning its review of the Nitrogen Dioxide (NO_2) Health Effects and Exposure Assessment Documents of the U.S. Consumer Product Safety Commission (CPSC). Under the provisions of an interagency agreement between the two agencies, the CASAC reviewed the CPSC documents at a public meeting on September 26-27, 1985, in Bethesda, Maryland.

Following its review of the documents prepared by the CPSC, the CASAC reached the following major conclusions:

- Preliminary evidence from epidemiologic and related indoor air pollution monitoring studies suggest that repeated peak exposures at concentrations of 0.3 ppm of NO_2 may cause health effects in some individuals and raises the possibility that such effects may occur at concentrations as low as 0.1 ppm. However, the epidemiologic and controlled human exposure studies provide somewhat inconsistent evidence which makes it difficult to be more definitive.

- Population groups that appear to be most sensitive to NO_2 exposures include children, chronic bronchitics, asthmatics, and individuals with emphysema.

- Human epidemiologic studies suggest that exposure to NO_2 may lead to increased respiratory illness rates among children. However, the most direct evidence regarding lung damage associated with NO_2 is obtained from animal studies. Such studies conclude that a number of effects occur in a variety of animal species, many of which can be considered serious and irreversible.

- The work undertaken by the CPSC to quantify the indoor NO_2 concentrations produced by kerosene space heaters is innovative and important and provides information that is essential to assess human health risks from these and other appliances producing NO_2 emissions.

- The EPA Air Quality Criteria Document and Staff Paper for NO_2 provide peer reviewed information and assessments directly relevant to questions facing the CPSC. The CASAC recommends that CPSC use these documents more fully as a primary resource in developing an assessment of the health risks of indoor combustion sources.

- The documents submitted by CPSC for CASAC's review were prepared at different times by various authors, for different CPSC purposes; therefore, they were sometimes repetitive and not well integrated. Although the documents generally identified the appropriate issues, they were not sufficiently developed to provide a primary resource for risk assessment without further revision. However, in light of the availability of the EPA Nitrogen Dioxide Criteria Document and Staff Paper, such revision may not be needed.

2. INTRODUCTION

A. Background

The Consumer Product Safety Commission (CPSC) has been concerned about exposure to nitrogen dioxide associated with the use of gas cooking stoves and a variety of home combustion heaters. Various studies, including several conducted for the CPSC, have shown that the levels of nitrogen dioxide exposure associated with the use of these appliances significantly exceed the national ambient air quality standard (NAAQS) as well as the short-term standard for nitrogen dioxide recommended by staff of the U.S. Enviromental Protection Agency (EPA).

On March 29, 1985, Commission Chairman Terrence Scanlon requested the assistance of the EPA's Congressionally established Clean Air Scientific Advisory Committee (CASAC) in conducting the CPSC's review of the potential health hazards associated with exposure to 0.1 to 1.0 plus parts per million (ppm) nitrogen dioxide generated by the unvented combustion sources used in the home (see Appendix C). In this request, the CPSC requested guidance on issues such as:

- the levels of nitrogen dioxide for which there are data indicating adverse health effects;
- the identity of subsets of the population more sensitive to nitrogen dioxide than others; and
- whether exposure to nitrogen dioxide leads to irreversible lung damage.

On May 1, 1985, EPA Administrator Lee M. Thomas agreed to this request, noting that the CASAC, which has reviewed the scientific basis of EPA's NAAQS for nitrogen dioxide, is well qualified to address the issues raised by the CPSC (see Appendix D). Staff of both agencies then developed an interagency agreement which was signed in August 1985.

Under the provisions of this interagency agreement, the CASAC reviewed the CPSC documents at a public meeting on September 26-27, 1985, in Bethesda, Maryland. At this meeting, the Committee heard presentations from CPSC staff on exposure assessment, controlled human exposure, animal toxicology and epidemiology relating to nitrogen dioxide, as well as comments from the interested public. The focus of this review was the September 1985 report of the Commission entitled Review of Nitrogen Dioxide: Health Effects and Exposure from Consumer Products, a six-part document discussing health effects of nitrogen dioxide and presenting information on kerosene heaters and unvented gas space heaters.

B. Report Format

This report has been divided into an Executive Summary, Introduction, three major Sections and an Appendix. Of the three major sections, Section 3 discusses the similarities between the assessment needs of the EPA and

the CPSC as well as the CASAC's view of its role in the CPSC review, and, in particular, the Committee's views regarding use of information generated by the EPA to simplify CPSC's assessment process. Section 4 contains the conclusions and recommendations of the Committee concerning Chairman Scanlon's three questions. Section 5 addresses additional issues that go beyond the information requested by Chairman Scanlon. Appendix A addresses in more detailed fashion some of the CASAC's comments on the documents supplied by the CPSC. Appendix B is a copy of the October 18, 1985 CASAC report to EPA detailing its findings and recommendations concerning EPA's National Ambient Air Quality Standard for nitrogen dioxide. Appendix C contains Chairman Scanlon's March 29, 1985 letter requesting the review. Appendix D presents Administrator Thomas' May 1, 1985 response to the request. Appendix E contains full citations to the literature referenced in this report.

3. COMMENTS ON THE GOALS OF THE REVIEW

During the course of its meetings on September 26-27, the CASAC sought to clarify the goals of the review. The Committee concluded that it had three tasks:

- To comment on the three questions posed by Chairman Scanlon regarding the health effects of NO_2.
- To assess the adequacy of the documents prepared by CPSC staff as a basis for assessing the risks of exposure to NO_2 emissions.
- To provide guidance to CPSC regarding further efforts to assess the risks associated with indoor use of space heaters and other appliances producing NO_2 emissions.

The questions posed by Chairman Scanlon in his letter to the EPA are both difficult and highly relevant to CPSC concerns about the potential health effects of kerosene space heaters. Fortunately, the EPA Criteria Document and Staff Paper on NO_2 provide peer-reviewed information directly relevant to these questions. We encourage CPSC staff to use these documents as a primary resource in future efforts to assess the health risks of indoor combustion sources.

The second and third tasks are similiar to those that the CASAC ordinarily performs in advising EPA on the adequacy of air quality criteria documents and staff papers. Given the availability of these EPA documents, we believe that further CPSC efforts should focus on quantification of the peak and average NO_2 concentrations produced in residences by unvented combustion sources and on systematic reevaluation of the evidence summarized in the EPA Criteria Document and Staff Paper with a focus on the higher indoor NO_2 concentrations produced by unvented combustion sources relative to the typical ambient concentrations of NO_2 implicitly addressed in the EPA documents.

4. MAJOR CONCLUSIONS AND RECOMMENDATIONS ON ISSUES POSED BY CPSC

In its evaluation of the materials provided by the CPSC, the CASAC drew a distinction between the material on exposure assessment and the documents reviewing the health effects literature. The Committee noted that CPSC staff and contractors have undertaken important and innovative work to quantify the indoor NO_2 concentrations produced by different, but typical uses of kerosene space heaters. The documents summarizing this work provide new and important information directly relevant to CPSC concerns. This information is central to assessing the health risk of indoor use of these and other applicances producing NO_2 emissions. Moreover, this material is unlikely to be assembled by other government or private groups. Thus, the CASAC urges the CPSC to continue this work and to further investigate the implications of these data for the impact of space heaters and other indoor sources on the population distribution of exposures to NO_2. Specifically, further efforts by CPSC staff to assess the health risks associated with indoor use of kerosene space heaters and other sources of nitrogen dioxide emissions should focus on efforts to quantify the nitrogen dioxide concentrations produced by these sources. We urge the CPSC to avoid duplication of EPA's effort to develop a comprehensive review of the literature on health effects of NO_2.

The following paragraphs respond to the questions posed by CPSC Chairman Scanlon.

- For what levels of nitrogen dioxide are there data indicating adverse health effects?

The CASAC has concurred with EPA's recommendation to retain the current Annual Primary National Ambient Air Quality Standard of 0.053 ppm (Appendix B). Evidence suggests that this average annual concentration should provide adequate protection against the adverse health effects associated with long-term exposure and protect to a lesser degree against short-term effects related to peaking of outdoor concentrations. Among the adverse effects related to chronic exposure in animals are a reduction in resistance to respiratory infection, accelerated aging of the lung manifested as a loss of elastic recoil, fibrotic and emphysematous-like structural changes in the lung, and impairment of function.

The lowest concentration(s) associated with acute adverse health effects can be expressed in a range of estimates. Preliminary epidemiologic findings and related indoor air pollution monitoring studies assessing the variation of NO_2 levels in gas stove homes suggest that repeated peaks in the range of 0.15 to 0.30 ppm may be of concern for children (USEPA, 1982). The limited number of controlled laboratory studies on human subjects, both healthy and with underlying lung disease, have produced conflicting results. For example, increased bronchial reactivity to a provocative aerosol has been reported after exposure to 0.1 ppm NO_2 in asthmatics (Orehek et al., 1976). This finding was not confirmed in a second study at the same concentration involving asthmatics and healthy subjects (Hazucha et al., 1983), while a third study found a "...variable effect..." on bronchial reactivity (Ahmed et al., 1982). The effect

of 0.2 ppm NO_2 on bronchial reactivity in asthmatics has been equivocal (Kleinman et al., 1983). Although both bronchoconstriction (Bauer et al., 1984; Rogers et al., 1985) and increased airway reactivity (Bauer et al., 1984) were found in response to 0.3 ppm NO_2, another study could find little or no effects at 4.0 ppm (Linn et al., in press). Therefore, the data base remains too sparse and the between-studies variance too great to project definitive dose-response relationships and complicates the task of identifying "safe levels" for the general population. Moreover, the mechanisms underlying these effects, and consequently their potential for contributing to chronic lung damage, are unknown. Such information may be vital in judging which effects are adverse, but much of this latest generation of studies has not yet appeared in the peer reviewed literature. Nonetheless, this preliminary evidence suggests that repeated peak exposures at concentrations of 0.3 ppm of NO_2 may cause health effects in some individuals and raises the possibility that such effects may occur at concentrations as low as 0.1 ppm, encouraging a cautionary approach in matters of policy.

As discussed in Appendix A, both the epidemiologic studies of children exposed to gas stove emissions and the controlled exposure studies of adults exposed to NO_2 have reported inconsistent findings regarding the health effects of these exposures. Such large uncertainties in clinical and epidemiological data are troubling to policy makers but are a reality in interpreting the currently available evidence on the health effects of NO_2.

- Which subsets of the population are most sensitive to nitrogen dioxide?

The EPA Staff Paper on NO_2 states that:

> ...the groups that appear to be most sensitive to exposures to NO_2 include children, chronic bronchitics, asthmatics, and individuals with emphysema....Health effects data from epidemiological studies in gas stove homes suggest that young children are at increased risk of respiratory symptoms and infection from exposures to elevated concentrations of NO_2....Other groups at risk to NO_2 exposures are asthmatics and bronchitics. Human clinical study data have provided evidence that some of these individuals suffer mild symptomatic effects (nasal discharge, headaches, dizziness, and labored breathing) after light to moderate exercise during an exposure to 0.5 ppm NO_2 for two hours.[1]

CASAC concurs with this statement.

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[1] Review of the National Ambient Air Quality Standards for Nitrogen Oxides: Assessment of the Scientific and Technical Information. U.S. EPA, Office of Air Quality Planning and Standards, Research Triangle Park, NC, EPA 450/5-82-002, Page 41, August 1982.

- Does exposure to nitrogen dioxide lead to irreversible lung damage?

As noted in Appendix A, controlled exposure studies provide little information about this question. Human epidemiologic studies suggest that exposure to nitrogen dioxide may lead to increased respiratory illness rates among children. Although a history of respiratory illness in childhood may be predictive of respiratory disorders in adult life, relatively little is known about this relationship at the present time. Thus, the most direct evidence regarding lung damage resulting from exposure to nitrogen dioxide is obtained from animal studies. These studies are reviewed in EPA's Criteria Document and Staff Paper. The Staff Paper provides the following summary of this complex and extensive data base:

> In critically assessing animal studies involving short-term exposure to NO_2, it is obvious that numerous effects have been observed for a variety of animal species (dogs, rabbits, guinea pigs, monkeys, rats, and mice). There is presently no reliable way to relate human and animal dose-response data. Many of the effects associated with short-term exposures appear to result not from a single exposure, but from multiple exposures in the range of 0.2 ppm to 0.5 ppm for several hours. Of particular interest is that exposure of animals to concentrations slightly above those currently being experienced in the ambient air appears to cause a decrease in resistance to bacterial infection.... this same type of effect has also been reported to occur in humans.
>
> ... effects which have been associated with animals exposed to NO_2 over relatively long periods (1 day to several years) ... include: (1) significantly increased susceptibility to infection resulting in increased mortality for continuous and intermittent exposure to $\geq$ 0.5 ppm NO_2; (2) decreased immunological response resulting in increased respiratory infection for exposures of 0.5-1.0 ppm NO_2, continuous and intermittent; (3) increased lung protein content suggesting edema and cell death for 3-6 week exposures to 0.5 or 1.0 ppm NO_2 in Vitamin C deficient animals; (4) hematological disturbances (e.g. increased cholinesterase lysozyme levels) suggestive of liver and heart damage at 0.5 ppm NO_2 for 1 week; (5) increased RBC 2,3-diphosphoglycerate, indicating tissue deoxygenation after 1 week exposure to 0.36 ppm NO_2; (6) emphysematous alterations resulting from a six month exposure to 0.1 ppm NO_2 with daily spikes of 1.0 ppm NO_2 or 68 months exposure to 0.64 ppm NO_2 and 0.25 ppm NO followed by a 2 year period in clean air.

> A critical assessment of the available animal toxicological data for long-term exposure to NO_2 reveals that many of the above effects occur in a variety of animal species, and that many of the effects can be considered serious and irreversible. For example, the emphysematous alterations in dogs associated with long-term exposure to NO_2 are of major concern since the occurrence of this type of effect in humans would clearly be adverse.
>
> While most of the chronic studies were conducted at exposures considerably higher than those encountered in the ambient air, it should be noted that one study did observe emphysematous alterations in mice when exposed to NO_2 levels about twice the current annual standard. However, in this study, the chronic exposure was supplemented with daily spikes of 1.0 ppm and it is not possible to determine if the cause of the effect was chronic exposure, short-term spikes or a combination of these two.
>
> Currently there is no means available to extrapolate the results of the animal studies (either short-term or chronic) directly to humans. Nevertheless, the animal toxicology studies do indicate that NO_2 exposure causes serious biological damage to a number of animals. These studies clearly raise a "warning flag" for potential effects in humans.[2]

Thus, while the animal studies do provide evidence that both short-term and long-term exposure to nitrogen dioxide can lead to irreversible health effects in a variety of animal species, it is difficult at the present time to determine whether these effects are of concern at concentrations associated with use of kerosene heaters, gas stoves, or other indoor sources of NO_2.

5. ADDITIONAL ISSUES

The documents summarizing the evidence regarding the health effects of NO_2 exposure correctly identify many of the important issues and studies, but the Committee believes that the materials have some important deficiencies. In particular, we note that the various documents have not been integrated, and we also have numerous questions about the studies chosen for emphasis and about the interpretation of some of the evidence. An informal compilation of CASAC's comments regarding the CPSC's review of the health effects literature is included as Appendix A.

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[2] Review of the National Ambient Air Quality Standards for Nitrogen Oxides: Assessment of the Scientific and Technical Information. U.S. EPA, Office of Air Quality Planning and Standards, Research Triangle Park, NC, EPA 450/5-82-002, Pages 10-11, August 1982.

The EPA's experience in preparing Criteria Documents and Staff Papers for the periodic assessment of criteria pollutants has shown that preparation of such reviews is enormously difficult and time consuming. Moreover, EPA staff routinely submit several revisions of each of these documents to CASAC in their efforts to develop a consensus on the relevant literature and its implications. In our view, the CPSC materials are comparable to the first draft of such integrated documents. From that perspective, the documents could provide a basis for developing an integrated review of the relevant literature. Given the effort involved in developing an integrated risk assessment, however, the Committee urges CPSC not to duplicate the resource-consuming effort required of the EPA in its periodic assessment of the health effects of NO_2. Rather, CPSC should make extensive use of the EPA Criteria Document and Staff Paper on Nitrogen Dioxide in its assessment of the health risks associated with indoor sources of nitrogen dioxide. In particular, the CPSC should utilize EPA's Staff Paper to the extent practicable. Every effort should be made to avoid duplication of reviews of the health effects literature carried out by EPA and reviewed by CASAC under the requirements of the Clean Air Act.

APPENDIX A

Document Review - More Detailed Comments

DOCUMENT REVIEW

The CPSC provided the following six documents to the Committee as background for the review:

- Update on Health Effects Associated with NO_2
- Health Effects of Nitrogen Dioxide
- Status Report on Kerosene Heaters (includes update: Health Effects of NO_2; Nitrogen Dioxide Health Assessment 1984)
- Status Report: Pollutants Generated by Unvented Gas Space Heaters
- Kerosene Heater Emissions: Estimation of Exposure
- Indoor Air Quality Kerosene Heater Testing Documentation

A. Overall Comments

Because these documents were prepared at different times by various authors for different CPSC purposes, they were sometimes repetitive and not well integrated. An introduction for the documents presented by CPSC delineating the purpose of the review would have been helpful. In addition, the CASAC's decision not to recommend a short term standard for ambient NO_2 concentrations in its review for EPA should not be interpreted as implying that no adverse effects are associated with the higher concentrations produced by indoor sources. Thus, an introduction discussing the potentially high NO_2 concentrations caused by space heaters and other sources and the need to re-evaluate the health effects literature with these concentrations in mind would have provided a sharper focus for the assessment.

The CPSC should consider organizing the material according to major topics and issues. This has already - and commendably - been done in part, i.e., Biochemistry, Pulmonary Functional Effects, and Extra Pulmonary Effects. In dealing with any category of effects, emphasis should be given to what is known about the mechanisms of the effect - this being a particularly valuable contribution of animal and *in vitro* toxicology - and whether similar mechanisms have been demonstrated or are plausible in humans. An important way of organizing and classifying effects is by duration, i.e., acute and chronic. Virtually the only explicit information available on the chronic effects of NO_2 (short of industrial accidents affecting individual workers) results from animal studies.

The CPSC should attempt to define the actual exposure levels of the indoor environment and to focus the discussion of health effects on those levels, insofar as possible. This effort should include any information on the frequency of occurance of such levels as well as the relationship between pollutant level and averaging time. Such an approach would help distinguish this document from the EPA documents which were concerned with outdoor effects.

There is a tendancy in the CPSC documents to overstate findings which may actually be more attributable to chance and which are not really statistically significant. Normal variation is a reasonable expectation,

but more caution is needed in interpreting statistical significance. The Committee was advised by the CPSC that their normal practice is to state when a finding is not statistically significant and may have been attributable to change and that caveats have been added when necessary.

For animal toxicology studies, the CASAC recognizes that one of the major problems, beyond the issue of whether or not the animal's health is adversely affected, is how to extrapolate animal data to humans. Relative dosing is one of the major issues in these extrapolations. This document need not exhaustively review dosimetry, but giving some perspective relative to the problems with animal studies would be helpful.

B. Exposure

Nitrogen dioxide is produced by a variety of combustion sources within the home, including space heaters and gas stoves. Although the CPSC document carefully describes a limited number of studies concerning emissions from certain space heater devices, additional discussion is needed of the relative NO_2 contribution of these devices compared to gas stoves.

The current protocols used by the CPSC to characterize emissions from space heaters are carefully conceived and well executed. Their approach focuses on the contributions to steady state levels represented by a 4 to 6-hour average concentration. The extrapolation of this information on steady state levels in confined spaces to actual homes is obviously more difficult. To the extent that additional information on the potential for shorter term (one-hour) peaks and their spatial distribution within the home can be generated from current data, this information would be very useful.

The chamber studies and modeling work carried out by CPSC and its contractors have been informative. In particular, these studies have identified a range of steady state concentrations associated with continuous operation of space heaters in chambers, and have characterized the relative emission rates of white and blue flame heaters, as well as the reductions in emissions achieved by catalytic converters and dual chamber designs. Such modeling activities are insufficient, however, to characterize distribution and temporal pattern of concentrations experienced by persons using space heaters in their homes. Factors that could affect the distribution of exposure include model preferences, age of heating units, mode of operation, and home characteristics. Given the potential health and regulatory significance of issues concerning kerosene space heaters, additional direct measurements of indoor concentrations are urgently needed. Such data should ideally be gathered in the context of a well designed sampling frame, with attention to temporal and home-to-home variability and to such covariates as wind speed and indoor and outdoor temperature. Given the paucity of direct measurement data available, a well-defined set of measurements in 50 to 100 homes, for example, would be of great value. If the CPSC is unable to undertake such a study,

interested scientific and industry groups should be encouraged to collect NO_2 measurements in homes using kerosene heaters. To the extent that long-term exposures of NO_2 are relevant to health, the present passive collector technology can be directly applied to this large home to home survey. If short-term (less than 6-hour) peak concentrations are more relevant, then the passive sampler technology must be supplemented with continuous NO_2 analyzers.

C. Animal Studies

The section on in vitro and animal toxicology is concise, and, for the most part, clearly stated. However, at times it is too selective and superficial in its attempt to reduce some complex problems to simple judgments.

If the CPSC chooses to rewrite the document, it should consider organizing the material more effectively. Effects might be organized under Pulmonary (biochemical, functional, immunological, resistance to infection, morphological) and Extra-pulmonary. Wherever possible, the document should distinguish between acute, subacute and chronic effects. Virtually the only empirical information available on the subacute and chronic effects of NO_2 (short of occupational accidents) comes from animal studies and, thus, this information is particularly important.

A topic not treated anywhere in the document is the uptake of NO_2 within the respiratory system, an issue which has implications for regional dose and for identifying target tissues. NO_2 uptake has been measured in some animal species; it has also been modeled in a preliminary way for the human lung. A critical issue in any attempt to extrapolate from animals to humans is the extent to which, for a specified ambient concentration, both total dose (corrected for differences in size) and regional dose are comparable across species.

An important issue that warrants separate, integrated treatment is a discussion of factors influencing susceptibility. These might include age, sex, nutritional status, and any animal models of underlying lung disease or extrapulmonary disease that may have been tested. This is another area of research in which animal toxicology can contribute significantly to insight into human risk. Whatever information is available on mechanisms of effect should also be added.

Summary tables for all three sections (animal, clinical, epidemiological) providing details on selected critical studies are useful and could be organized to show effects as functions of increasing concentrations (separate tables for acute and chronic exposures). Such tables could also include whatever information is available on the reversibility of effects.

The animal toxicology discussion includes a variety of biochemical changes that occur in response to single or repeated exposures to NO_2. In general, these changes are reversible; even dead epithelial cells may be replaced through regenerative processes. However, some of the changes may

become part of a process that culminates in irreversible tissue damage and deformity. This process may be sustained if the exposure to NO_2 is sufficiently protracted or severe, if other forms of environmental stress are also present, or if the defensive and reparative responses of the body are compromised. The studies of Gillespie and co-workers suggest that the evolution of changes that are "emphysematous-like" in character may continue even after cessation of exposure to NO_2; however, this work has defects in experimental design and should be redesigned. How reversible the effect of NO_2 may be following protracted exposure must be determined empirically by allowing the animals to survive after ending the exposure; data of this sort are presently scarce.

D. Controlled Human Exposure

This section of the CPSC Report gives a useful review of the key scientific literature. The Committee identified no substantial errors or omissions in this material. Nevertheless, a reader wishing to compare and contrast the various studies needs a tabular summary similar to Tables 2 and 6 in the EPA Staff Paper on Nitrogen Dioxide (USEPA, 1982).

Committee members offered many suggestions for changes in content, emphasis, or language. The specific comments below highlight some issues about responsiveness of population sub-groups, interpretation of pulmonary function measurements, and interpretation of conflicting results from different controlled human exposure studies.

Specific Suggestions:

Comments in the CPSC document that imply definitive knowledge about the relative NO_2 responsiveness of population sub-groups who have lung disease should be revised (see pages 19 and 20)[3]. Because available results are conflicting, they should be cautiously interpreted and statements about them referenced whenever possible.

The discussion of confounding issues that arise in the interpretation of pulmonary function measurements (see page 20)[3] should be changed to indicate: 1) that intra-subject variability in clinical studies is mitigated by using repeated measures with subjects serving as their own control, 2) that with due care, subjects with decreased lung function reserve from respiratory disease can and are being studied to collect evidence about how their responsiveness to NO_2 exposure varies with disease severity, and 3) that the use of the term "significance" has statistical support; if not, another word should be used.

As pointed out in the CPSC document (see page 21),[3] results of the presently available studies on NO_2 effects are inconsistent. The CPSC should expand its discussion of the reasons for such inconsistency to include

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[3] Page references are to the CPSC Report Update on Health Effects Associated with Nitrogen Dioxide, 1985 by Lori Saltzman

other factors that are also likely (if not more likely) for this development - such as differences in exposure methods as well as differences in subject populations. For example, the "positive" studies have probably involved more sensitive asthmatics than the "negative" studies, but judging this will be very difficult until detailed results have been released and critically reviewed.

From short-term (1-hour) controlled studies of adults, one could estimate the lowest-observable-effect level to be below 0.2 ppm at one extreme, or above 4.0 ppm at the other extreme depending on the response being tested. This large discrepancy between different findings is troubling to scientists as well as to policymakers. With ozone and sulfur dioxide, the discrepancies between different studies and different laboratories are much smaller. However, two recent reports (Bauer et al., 1984; Rogers et al., 1985) have observed changes in lung function in exercising asthmatics exposed to 0.3 ppm NO_2. Despite the failure of other studies to demonstrate such changes, these levels may provoke responses in some asthmatic individuals. There is no good explanation for the widely divergent results on NO_2.

The controlled human studies which have suggested unfavorable effects at low concentrations (0.2 - 0.3 ppm or even lower) all have employed adult asthmatics. Asthmatics thus are the best candidates for the "more sensitive" designation. However, other studies have concluded that many mild asthmatics experience a detectable effect at concentrations an order of magnitude higher. This has led to the suggestion (not explicitly tested as yet) that NO_2 sensitivity is correlated with an index of severity of asthma, perhaps the degree of airway obstruction or baseline airway hyperreactivity.

The demonstration of unfavorable short-term reversible effects is sometimes thought to imply a possible risk of long-term irreversible effects, but any direct relationship between short-and long-term effects remains unclear. As indicated above, even short-term effects have not been demonstrated unequivocally for NO_2.

E. Epidemiology

The most relevant studies for the CPSC's needs are the British and U.S. studies of the health status of children and adults living in homes where gas is used for cooking or heating. The CPSC review document is an accurate evaluation of these studies. The Staff Paper (USEPA, 1982) represents a more extensive evaluation of all NO_2-related epidemiologic studies, with conclusions that are consistent with the CPSC evaluation. In brief, the extensive information available is suggestive but not conclusive that unvented gas combustion devices in homes are associated with slight excesses of respiratory illnesses, especially in children. The inconsistent findings among investigators and among different studies by the same investigators, some of which are comparisons of cross-sectional

and longitudinal observations, suggest that the many confounding factors which inevitably occur have not been adequately accounted for (and, in some instances possibly "over-corrected").

It is evident that use of gas for cooking or heating is not a risk factor of great magnitude in comparison with a factor such as cigarette smoking. It is also not certain that NO_2 is the causal factor for whatever risk may be associated with gas stoves or heaters. Unfortunately the majority of epidemiologic studies include no information on NO_2, and among those that do have actual measurements, the number of homes and characterization of concentrations are very limited. This suggests that better quantification of exposure is a major need in future studies.

The epidemiological studies provide some information relevant to the three questions posed by the CPSC. Regarding the concentrations at which adverse health effects are seen, these studies suggest that repeated exposures to peak NO_2 concentrations in the range of 0.15 to 0.30 ppm may be associated with increased risks of respiratory illness in children. No relevant epidemiologic research has been directed at other particularly susceptible groups such as asthmatics, bronchitics, heart disease patients, or the elderly. Epidemiological studies to-date have not addressed the question of the reversibility of effects.

APPENDIX B

Nitrogen Dioxide Closure Letter of the Clean Air Scientific Advisory Committee - October 18, 1984

NEW YORK UNIVERSITY MEDICAL CENTER
A private university in the public service

Institute of Environmental Medicine
550 FIRST AVENUE, NEW YORK, N.Y. 10016
AREA 212 340-7300, Ext. 835-

ANTHONY J. LANZA RESEARCH LABORATORIES AT UNIVERSITY VALLEY
LONG MEADOW ROAD, STERLING FOREST, TUXEDO, N.Y. 10987
MAIL AND TELEPHONE ADDRESS: 550 FIRST AVENUE, NEW YORK, N.Y. 10016

October 18, 1984

Honorable William Ruckelshaus
Administrator
U.S. Environmental Protection Agency
401 M Street, S.W.
Washington, DC 20460

Dear Mr. Ruckelshaus:

On July 19-20, 1984, the Clean Air Scientific Advisory Committee (CASAC) met to consider the Agency's proposal regarding revisions to the National Ambient Air Quality Standards (NAAQS) for Nitrogen Dioxide. Included in this proposal is the reaffirmation of the existing annual average standards for nitrogen dioxide at 0.053 ppm (100 ug/m^3), and solicitation of public comments on both the need to set a separate short-term standard and the need to use an alternative form of the standard (statistical instead of deterministic). The Committee has prepared this closure letter to advise you of its major conclusions and recommendations concerning the scientific and technical aspects of these and other issues associated with the Agency's proposal for the revision of the NAAQS for nitrogen dioxide.

Through previous closure letters dated June 19, 1981 and July 6, 1982, respectively, the CASAC advised that the revised Air Quality Criteria Document for Nitrogen Oxides was scientifically adequate for standard setting and that the Office of Air Quality Planning and Standards (OAQPS) Staff Paper represented a balanced and thorough interpretation of the scientific evidence contained in the criteria document. The Committee has reviewed relevant research which has been published since those documents were prepared, and concludes that the scientific conclusions reached in those documents are still satisfactory.

The CASAC has concluded that the existing annual average primary standard for nitrogen dioxide adequately protects against adverse health effects associated with long-term exposure and provides some measure of protection against short-term health effects. Therefore, the Committee concurs with the Agency's recommendation to retain the current annual average primary standard of 0.053 ppm. The Committee further concludes that, while short-term effects from nitrogen dioxide are documented in the scientific literature, the available information was insufficient

SESQUICENTENNIAL 1831 CELEBRATION

to provide an adequate scientific basis for establishing any specific short-term standard, or for determining an acceptable number of exceedances, a concentration limit, and an averaging time for such a standard. Indeed, the scientific basis for setting a separate short-term standard appears to be less firm than it was at the time of the Committee's previous review. We recommend that the Agency vigorously pursue a research program designed to address and resolve the issues related to short-term effects of nitrogen dioxide.

The Committee reaffirms its conclusion from two years ago that a secondary standard set at a level equivalent to the annual primary standard would offer sufficient protection against the identified welfare effects of nitrogen dioxide.

Members of the Committee who held a view on the issue of the form of the standard favored retaining the present deterministic form rather than adopting a statistical form for the annual standard.

A more extended analysis of the factors leading to the Committee's recommendations is contained in the enclosed report. Thank you for the opportunity to present the Committee's views on this important public health issue.

Sincerely,

Morton Lippmann

Morton Lippmann, Ph.D.
Chairman, Clean Air Scientific
Advisory Committee

Enclosure

cc: Mr. Alvin Alm
Mr. Joseph Cannon
Dr. Bernard Goldstein
Dr. Terry Yosie

CASAC Findings and Recommendations on the Scientific Basis for a Revised NAAQS for Nitrogen Dioxide

Options Presented by the Agency

Agency staff presented for CASAC review and comment three options that the Agency could pursue in concluding its current review of the NAAQS for nitrogen dioxide. These are as follows:

1. Reaffirm the annual standard at the current level and propose a short-term standard, or

2. Reaffirm the annual standard at the current level and conclude that a short-term standard is not needed, or

3. Reaffirm the annual standard at the current level, defer a decision on a short-term standard, and perform high priority research on short-term effects of nitrogen dioxide.

Based on the Committee's review of the scientific issues associated with the reaffirmation of the annual standard and the possible short-term standard as discussed below, the Committee believes: 1) that there is an insufficient scientific basis for action on option 1; and, 2) that options 2 and 3 are functionally equivalent, i.e. a vigorous program of research into the short-term effects of nitrogen dioxide is needed and can be accomplished under either option.

Scientific Issues in Revising the Standards

In CASAC's closure letter of July 6, 1982, the Committee discussed its review of the nitrogen oxides staff paper, noting that no single study provided the scientific basis for a decision on revising the primary standard for nitrogen dioxide. Rather, it could be based on a "weight of evidence" approach, using animal studies, controlled human exposure studies and epidemiology studies to provide both quantitative (i.e. exposure/effect) and qualitative (mechanistic) support for such a decision. Since that time new studies have been completed and, along with previously discussed studies, form the basis for the Committee's conclusions and recommendations concerning the critical issues associated with reaffirming the annual standard and evaluating a short-term standard for nitrogen dioxide.

1. Animal Toxicology Studies.

The results from recent animal studies provide further substantiation of the effects of nitrogen dioxide exposure on immune functions and increased susceptibility to infection. Some of these studies also examine patterns of exposure to nitrogen dioxide that are closer simulations of what may be actually occurring in, for example, gas stove homes. An example of this

is superimposing repeated short-term higher levels of exposure to nitrogen dioxide (e.g. 0.4 to 5.0 ppm, or more) on relatively low background levels of nitrogen dioxide, such as found in gas stove homes.

2. Controlled Human Exposure Studies.

The more recent controlled human exposure studies (mostly unpublished) present rather mixed and often contradictory results concerning respiratory effects in asthmatic and normal subjects exposed to concentrations in the range of 0.1 to 4.0 ppm nitrogen dioxide. Kagawa and Tsuru (1979) reported results possibly suggestive of short-term nitrogen dioxide effects on pulmonary function in normal subjects without combined provocative challenge by other agents (such as carbachol). Although they reported no significant differences for mean pulmonary function changes for a group of six subjects exposed to 0.15 ppm nitrogen dioxide, there were small significant decreases in airway conductance in three of the six subjects. However, the smallness of these decrements and questions regarding the statistical analyses used suggest caution in accepting the reported findings as demonstrating nitrogen dioxide effects on pulmonary function at 0.15 ppm. More recently, Bauer et al. (1984 - abstract) exposed asthmatics to 0.3 ppm nitrogen dioxide and observed effects on both pulmonary function after exercise and airway reactivity following cold air challenge.

3. Epidemiological Studies.

The most recent epidemiological studies indicate less conclusive findings of an association between nitrogen dioxide and respiratory effects than previously reported. The first report of the Harvard Six Cities Study, published several years ago, noted one positive result -- an association between both lung function changes and respiratory illnesses in children under age two and exposure to gas stoves -- among a number of associated variables. More recent analyses, published in Ferris et al. (1983) and Ware et al. (1984) made adjustment for the socio-economic status of the children under age two and reported that the association between their living in homes with gas stoves and their incidence of respiratory illness is no longer statistically significant. From these results, as well as those reported by other investigators studying people living in homes with gas stoves, CASAC concludes that the scientific evidence supporting an association between living in homes with gas stoves and increases in respiratory illnesses and symptoms is insufficient to support specific limits for either short-term or long-term standards for nitrogen dioxide.

Annual Standards

1. Primary Standard.

The CASAC reviewed the results of animal, controlled human exposure, and epidemiological studies to determine if such evidence provided a scientific basis for retention of the annual standard and scientific support

for establishment of a short-term standard. The most serious effects associated with nitrogen dioxide exposures that are reported in the scientific literature result from animal studies conducted at concentrations well above those permitted by the current annual standard. Although there are large uncertainties in extrapolating these results directly to humans, the seriousness of these effects coupled with the biological similarities between animals and humans suggests that there is risk to human health from long-term exposure to nitrogen dioxide. This set of factors, widely accepted within the scientific community, leads the Committee to conclude that there is a continuing need for a long-term nitrogen dioxide standard.

The results from recent studies showing some evidence of detectable health effects due to short-term nitrogen dioxide exposures do not provide sufficient evidence to develop a concentration level, an averaging time, or a number of exceedences for a short-term standard. For example, the gas stove studies were originally used in support of the rationale for a short-term standard; however, recent reassessments by the authors of these studies led them to reduce the level of statistical significance of their reported results. Moreover, the results of the recent clinical studies have been inconsistent. As a result, the overall scientific support for a short-term standard is more equivocal than previously thought. If the CASAC were to make a recommendation favoring a short-term standard, the Committee would also have to take into account the need to determine the number of allowable exceedances, the establishment of a concentration level, and the identification of an averaging time. At the present time, the Committee is unable to make such recommendations due to the absence of a sufficient body of information on such factors.

2. Secondary Standard.

The CASAC has not identified any further information to change its conclusion from two years ago that a secondary standard set equivalent to the annual primary standard would offer sufficient protection against the identified welfare effects of exposures to nitrogen dioxide. Although the issue of visibility impairment was raised, several members noted that, given the present state of knowledge, it is difficult to identify the degree to which nitrogen dioxide concentrations may contribute to this phenomenon. The Agency indicated that further work on this complex, multi-pollutant issue has been assigned a high priority in relation to the task force on visibility and that the issue will be addressed further at subsequent CASAC meetings. The Committee is looking forward to reviewing the results of the Agency's progress on this important issue.

Form of the Standard

The Committee did not reach a consensus on the desirability of changing the form of the standard from the present deterministic form to a statistical form which uses the available arithmetic averages from

the last three-years to determine compliance. Although most members of the Committee took no position, one member suggested that there is a stronger argument for a statistical approach to short-term standards than for annual standards. Two others favored the retention of the current deterministic form for the annual standard.

Research Efforts

The CASAC was encouraged to learn that the Agency is currently pursuing research which addresses some of the issues raised in our December 30, 1983 report to you on Research Needed to Support the Development of NAAQS. We look forward to continued reports from the Agency on the progress of this important research. The Committee feels compelled to reiterate that without an adequately funded research program aimed at assessing the significance of the health effects associated with short-term nitrogen dioxide exposures, the Agency cannot make scientifically informed decisions concerning the need for a short-term standard, its concentration level, averaging time or an acceptable number of exceedances.

Summary of CASAC Recommendations

For the reasons stated, the Committee recommends that you reaffirm the annual standard at the current level, and that you defer a decision on the short-term standard while pursuing an aggressive research program on short-term effects of nitrogen dioxide.

APPENDIX C

Letter from CPSC Commissioner Scanlon
March 29, 1985

UNITED STATES
CONSUMER PRODUCT SAFETY COMMISSION
WASHINGTON, D.C. 20207

The Chairman

March 29, 1985

Honorable Lee M. Thomas
Administrator
U.S. Environmental Protection Agency
401 M Street S.W.
Washington, DC 20460

Dear Mr. Thomas:

I am requesting the assistance of the Environmental Protection Agency's Clean Air Science Advisory Committee (CASAC) in our review of the potential health hazards associated with exposure to 0.1 to 1.0 plus ppm nitrogen dioxide generated by the unvented combustion sources used in the home.

The Commission has been concerned about consumer exposure to nitrogen dioxide associated with the use of gas cooking stoves and a variety of combustion home heaters. Various studies, including several conducted for the CPSC, have shown that the levels of nitrogen dioxide exposure associated with the use of these appliances significantly exceed the ambient air standard as well as the short term standard previously recommended by the EPA staff.

Various gas stove and combustion heater industry representatives have indicated a willingness to modify their product in order to reduce consumer exposure to nitrogen dioxide but there remains some disagreement as to what the target level should be. In an effort to expedite this process, I believe that the CASAC, since it has recently reviewed the data on NO_2, could give the Commission guidance on questions such as:

- -the levels of nitrogen dioxide for which there are data indicating adverse health effects;
- -the identity of subsets of the population more sensitive to nitrogen dioxide than others; and
- -whether exposure to nitrogen dioxide leads to irreversible lung damage.

Please advise me as to the feasibility of obtaining such assistance, and, if it is feasible, the process for obtaining CASAC review. Thank you for your assistance in this matter.

Sincerely yours,

Terrence Scanlon

Terrence Scanlon
Chairman

APPENDIX D

Letter from EPA Administrator Lee Thomas
May 1, 1985

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

MAY 1 1985

THE ADMINISTRATOR

Honorable Terrence Scanlon
Chairman
U. S. Consumer Product Safety
Commission
Washington, D. C. 20207

Dear Mr. Scanlon:

Thank you for your March 29 letter in which you request the assistance of EPA's Clean Air Scientific Advisory Committee (CASAC) in evaluating consumer exposures to nitrogen dioxide associated with the use of gas cooking stoves and a variety of combustion home heaters. The Committee, which has reviewed the scientific basis of EPA's National Ambient Air Quality Standard for Nitrogen Dioxide, is well qualified to address the issues identified in your letter, and I support your request to solicit its scientific advice. I know that the CASAC Chairman, Dr. Morton Lippmann of New York University, would also be willing to assemble his panel to undertake this review.

I suggest that your staff coordinate the preparation for the CASAC meeting with Dr. Terry F. Yosie, Director of EPA's Science Advisory Board, (382-4126) and Mr. Bruce Jordan of EPA's Office of Air Quality Planning and Standards (919) 541-5655. Two specific requests that I have of the Commission staff is to work closely with Dr. Yosie and Mr. Jordan in preparing the scientific materials to submit to CASAC, and to provide budgetary support to defray the cost of the meeting. I estimate that the costs of the review will approximate $15,000 - $20,000.

Thank you for your interest in working with the Agency on this important public health issue.

Sincerely,

Lee M. Thomas
Administrator

APPENDIX E

Literature Cited

LITERATURE CITED

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CPSA 6 (b)(1) Cleared
No Mfrs/Prvtlbls or Products Identified
Excepted by Firms Notified,
Comments Processed.

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OFFICE OF THE SECRETARY
FEB 29 10 26 AM '82
CONSUMER PRODUCT
SAFETY COMMISSION

UNITED STATES GOVERNMENT

Memorandum

U.S. CONSUMER PRODUCT SAFETY COMMISSION

FEB 25 1982

TO: Commission
Through: Sayde Dunn, Office of the Secretary
Through: Margaret A. Freeston, Acting General Counsel
Through: Kirke Harper, Acting Executive Director
Through: Peter W. Preuss, AED, Health Sciences

FROM: Sandra Eberle, Senior Project Manager, Chemical Hazards Program
Health Sciences

SUBJECT: Summary of National Academy of Sciences Report on Indoor Pollutants

Attached for your information and review are summaries, prepared by the Health Sciences staff, of several recent reports on the general subject of indoor air quality. The purpose of this report is to provide the Commission with 1) a summary of the current knowledge of indoor pollutants, and 2) an update on the research in progress on indoor air quality in this and other agencies.

The most significant of the recent reports, Indoor Pollutants, prepared by the Committee on Indoor Pollutants of the National Academy of Sciences (NAS), is a definitive treatment of the information assessed to date on the sources, health effects, measurement methodology, and control strategies for pollutants in the indoor environment. The NAS committee found that hazardous pollutants are emitted from sources in individual residences, public buildings and public areas. Many of the pollutants with known human health effects are released from consumer product sources under the Commission's jurisdiction.

The staff also is involved actively with the Interagency Research Group on Indoor Air Quality and the Ad Hoc Task Force on Indoor Air Pollution. Brief staff summaries of the reports of these multi-agency groups are also included in the attached paper.

Indoor air quality was designated a Commission priority in FY 1981 and FY 1982. The staff is working on the various aspects of IAQ through several projects, including Particleboard/Plywood, UFFI, Home Combustion Products, and Home Materials. In these projects the staff is attempting to determine the extent of the potential hazard by characterizing the chemicals present in the indoor environment, the degree of consumer exposure, and the relative contributions of various consumer product sources, to the collective indoor air environment.

The staff summary includes the recommendations made by the NAS, and although the staff agrees with many of the recommendations, this package does not request any Commission decisions on the NAS recommendations. Any staff recommendation for additional resources to further address selected issues will be presented in the context of operating plan review.

Copies of the reports summarized in this paper are available for public inspection in the Office of the Secretary. This report was reviewed and approved by the Directorates of Compliance and Administrative Litigation, Economics, Engineering Sciences, Epidemiology; Health Sciences, and the Offices of Budget, Program Planning and Evaluation; Program Management and Outreach Coordination.

Attachments

Summary of

National Academy of Sciences

Report on
Indoor Pollutants

FOR FURTHER INFORMATION:

Sandra Eberle
Senior Project Manager
Chemical Hazards Program
(301) 492-6984

I. PREFACE

Indoor air pollution, a problem which has recently attracted significant governmental and scientific attention, originates primarily from products used in our homes. Recent air sampling has established that, in some older homes, but especially in new energy-efficient homes, pollutants can exceed established national health limits. Many of these pollutants are the same as those in the ambient air and the workplace, locations which have been studied extensively. But to date, there have been few studies of health effects resulting from exposure to toxic substances in homes and public buildings.

Efforts to improve air quality have focused largely on controlling sources of outdoor pollution and reducing occupational exposure. The growing recognition that significant levels of hazardous pollutants are generated indoors is but one factor that has caused scientists and health officials to reconsider the traditional emphasis on outdoor air alone. Other significant factors are:

- Americans spend about 90 percent of their time indoors;
- indoor exposures are unwitting and often unrecognized;
- among the exposed are our most vulnerable individuals - young children, the aged, and the ill.

The National Academy of Sciences, in their 1981 report Indoor Pollutants addresses the question of the quality of indoor environments. The study sought to identify the indoor sources of these pollutants and to evaluate 1) their known effects on human health and welfare, 2) the techniques for measuring and estimating human exposure, and 3) the methods and strategies for controlling the pollutants. The NAS Committee on Indoor Pollutants set out to "review, compile and appraise the available knowledge" on potential adverse health effects of indoor pollutants. The Academy concluded that the quality of the indoor environment is a matter of immediate and great concern. In order to focus directly on the problem, the NAS Committee limited its study to several pollutants found in homes and public buildings for which, "human exposures are large and common enough to account for substantial morbidity and premature mortality."

- Radon gas and its radioactive decay particles emitted from soil, ground water, and building materials containing uranium and radium bearing rock; known to cause lung cancer in uranium workers.

- Formaldehyde - released from urea-formaldehyde foam insulation, particle board, plywood, and fabrics containing formaldehyde based resins; known to cause irritation and sensitization in humans, and nasal cancer in laboratory animals.

- Asbestos - fibers released from insulation and other building materials during installation or when the surface is disturbed by deterioration, renovation, or other human activities; known to cause lung cancer and mesothelioma (cancer of the lining of the thoracic and abdominal cavities).

- Tobacco Smoke - a combination of carbon monoxide, nicotine, formaldehyde, benzo(a)pyrene and altogether about 2,000 compounds; causes lung cancer in smokers, also suspected of causing respiratory problems in nonsmokers who live and work with smokers.

- Indoor combustion products - carbon monoxide, nitric oxide, nitrogen dioxide, formaldehyde, and carbon particles from unvented gas appliances, as well as from wood and coal-burning space heaters and fireplaces. Carbon monoxide and nitric oxide combine with hemoglobin and reduce the blood's ability to carry oxygen to the cells; studies cited by the committee reported a higher incidence of respiratory illness and dysfunction among children living in homes with gas cooking stoves than among those with electric cooking stoves.

- Microorganisms and allergens - bacteria, viruses, and fungi increase, as does the liklihood of their causing infection, when ventilation is reduced.

The Committee also expressed concern over the many organic pollutants possibly found in indoor air, but pointed out that little is known about these substances.

The Academy presents extensive conclusions in the general areas of health effects, economic considerations, and control strategies. Those of most significance to the Commission are paraphrased, or when appropriate, quoted directly in the following sections:

HEALTH EFFECTS

- "Comparison with reported indoor concentrations makes it evident that -- by some established ambient, occupational, or indoor standards -- current exposures to some contaminants indoors could constitute a health risk to occupants."

- Because many Americans spend 90 to 95 percent of the day indoors, it is the indoor concentration of many pollutants which is of greatest significance in evaluating the pollutants potential to cause health effects, loss of productivity, discomfort, or material damage.

- "For specific indoor contaminants, two basic inadequacies in the available data must be resolved rapidly: poor definition of population exposures and lack of understanding of the health and welfare consequences of exposure to contaminants in the indoor environment."

- "The committee believes that the research efforts to characterize indoor air pollution and human exposures indoors must continue and intensify, if we are to determine total human exposure to pollutants and understand environmental contamination and its effect on health and the quality of life."

- "The contents of consumer products should be investigated, and the chemical constituents should be tested for their toxic, mutagenic, carcinogenic, and teratogenic properties. . . .Their synergistic and interactive effects with other indoor pollutants should be tested."

- Environmental air contaminants that are liberated indoors are an important stress on human health and welfare.

ECONOMIC CONSIDERATIONS

- The need to conserve energy has placed increased attention on reducing the exchange of indoor "conditioned" air for "unconditioned" outdoor air. Heating and cooling of air in buildings accounts for an estimated 20 to 50 percent of the energy consumed in the U.S. Reduction of air exchange rates in homes reduces energy consumption, but also increases retention and concentration of indoor air pollutants.

- "The most tangible effects of air quality on productivity and quality of life are the adverse effects on health and longevity. However, of greatest economic significance may be the indoor pollutants effect on productivity."

- "Premature death or chronic disability removes the individual producer . . . any increase in illness or malaise can reduce productivity through absenteeism, . . . even momentary distraction, discomfort, annoyance, or physical irritation will reduce the quantity or quality of production."

- The health risk may be greater for persons in low-income groups because this subpopulation suffers from a high proportion of chronic health conditions which keep them indoors, and because low-income housing, e.g. older buildings, mobile homes, crowded apartments, is likely to have several conditions such as improper ventilation, poor maintenance, improperly operating stoves and space heaters, and lead based paints which contribute to higher indoor concentrations of indoor pollutants.

- "Home exposure to asbestos due to aging, cracking, or physical disruptions of insulated pipes or asbestos-containing ceiling tiles and spackling compounds may be greater than public exposure in schools," which EPA estimates will cause over 1,000 deaths. Estimated societal costs of these deaths range from $170 million to $3.2 billion.

SOURCES AND POTENTIAL CONTROL STRATEGIES

- "The potential for consumer products to contaminate the indoor environment needs to be evaluated. . . Testing in homes is needed to assess the extent of contamination, allergic reactions, and other health effects of . . . consumer products."

- "Public education offers an effective way of reducing exposure of the population to many contaminants encountered indoors. People informed about the potential for exposure to pollutants from consumer products, tobacco smoke, combustion products, etc., will exercise some control to reduce the pollutant concentrations in their environments."

- The implementation of energy conservation measures and the introduction of new building materials have intensified the problem of indoor air contamination.

- Pollutant sources may be well recognized, such as cigarette smoking, or pollutants sources may be new products, or old products in expanded uses. Building materials; insulation; cooking; use of household chemicals; floor and wall coverings; space heating devices, including the reemergence of coal and woodburning stoves and the expanded use of home hobby and craft products; all are pollutant sources of concern only in the indoor environment.

- "Efforts to improve indoor air quality should concentrate on reducing the number and the strength of the contaminant sources by substitution of other materials." The Academy expected specific source controls to vary for each contaminant, mentioning for example, local exhaust control for combustion products from gas ranges and prohibition for asbestos-containing products. The NAS also recognized that practical realities and cost considerations might require that source control be supplemented by engineering controls, such as dilution, ventilation, and air cleaning.

- Of the five general control strategies identified (ventilation, source removal, source modification, pollutant removal and education) source removal is judged to be the most effective means of controlling indoor pollutants.

To respond to the public need for a safe and healthful indoor environment, the Academy proposed a multi-agency sponsored national assessment of the "exposure of the general population to indoor pollutants and the effects of such exposures on health and welfare."

CURRENT FEDERAL EFFORTS

Through the Interagency Research Group (IRG) on Indoor Air Quality, established in 1980, the Federal agencies are seeking to coordinate the ongoing research on the pollutants described in the NAS report. The IRG concludes, as does the NAS, that studies are needed to define the role of the identified pollutants in human respiratory problems, cancer, and hemoglobin and cardiovascular function. Also stressed is the need for research which extends beyond the individual pollutants to 1) determine the cumulative effects of mixtures of contaminants, 2) understand the influence of air exchange rates, building design, and human activities on concentrations of pollutants, and 3) design special monitoring devices for indoor air analysis.

At the present time, the Environmental Protection Agency and the Department of Energy are sponsoring limited monitoring studies, to determine total exposure to selected pollutants, and work on the development of monitoring equipment. A primary indoor pollutant of concern for these agenices has been radon progeny. However, the staff is concerned that EPA research on indoor air quality, as such, has been largely suspended because of budgetary constraints and lack of statutory mandate.

The Commission's efforts are largely concentrated in identifying products associated with in-home sources of asbestos, formaldehyde, carbon monoxide, nitrogen oxides, and organic pollutants. As discussed in-depth in the following sections, the primary sources of these significant pollutants are consumer products under the Commission's jurisdiction. Expanded knowledge of indoor pollutants will provide a more accurate estimate of an individuals total exposure to hazardous pollutants. This knowledge will, in turn, provide a more rational basis for public and private decisions on how and where controls to limit the exposure to pollutants, if determined to be necessary, can most effectively and efficiently be applied.

II. Indoor Combustion

A. Introduction

As energy conservation becomes more important, the use of alternate heating sources, such as portable space heaters and wood and coal-burning stoves, is expanding. Home ventilation rates are also being decreased due to tighter construction and other energy saving measures. The combination of these changes would be expected to cause increased concentrations of gases and particulates from indoor combustion processes such as heating, cooking, and smoking.

The major pollutants associated with combustion processes include: carbon monoxide (CO), nitrogen oxides (NO_x, e.g., nitrogen oxide NO, nitrogen dioxide NO_2), aldehydes (e.g., formaldehyde), carbon dioxide (CO_2), sulfur dioxide (SO_2), other organic compounds, and fine particles.

Data indicate that levels of CO, NO_x, and SO_2 in many homes exceed corresponding outdoor levels as well as National Ambient Air Quality Standards (NAAQS) set by EPA. Average hourly concentrations indoors may also exceed highest hourly concentrations outdoors.

Both NO_x and SO_2 are irritants at relatively low levels. Some studies have shown an association between gas cooking and a higher incidence of respiratory disease in children, which has been attributed to NO_x. CO produces both cardiac and behavioral changes following acute exposure. The effects of long term human exposure to low levels of CO and other combustion products are not, however, well understood at the present time.

Because both NO_x and CO are odorless at typical concentrations, the presence of increased or possibly hazardous concentrations may go undetected. Both SO_2 and formaldehyde (HCHO) have been shown to cause respiratory irritation at concentrations measured in homes. Formaldehyde has also been shown to be carcinogenic in animals and mutagenic in many test systems. The health effects and indoor levels of HCHO are discussed in the Home Materials section of this report which also deals with the major sources of HCHO in the indoor environment, including urea-formaldehyde foam insulation, particleboard, and plywood. The available evidence suggests that important population exposure to HCHO, NO_x, and CO can occur indoors and may constitute a sufficient threat to the general public health to justify remedial action by appropriate governmental agencies.

B. Sources

The primary sources of combustion products in homes are unvented or improperly vented space heaters and furnaces, gas ranges and ovens, and gas water heaters. Exhaust from automobiles in attached garages as well as wood fires, oil and kerosene lamps, can contribute to elevated levels

of combustion products indoors.

Externally vented central furnaces or space heaters can also contribute to indoor air pollution. When these systems are properly designed, maintained, and functioning this does not occur. If however, a negative pressure develops in the interior space or if there is some fault in the exhaust system, direct and serious degradation of indoor air quality can result. Several studies cited by NAS illustrate this point. In one study, a faulty heater caused the indoor concentrations of SO_2 to be 3.8 times greater than the outdoor concentration. In another study, a home with an antiquated central coal heating system had elevated SO_2 levels approaching 1 ppm while CO levels of over 50 ppm were reached. These levels exceed established NAAQS of 0.052 ppm for SO_2 and 9 ppm for CO. A gas-fired heating system in another home, although vented to the outside, produced higher NO and NO_2 concentrations indoors than ambient levels outdoors.

Unvented gas and kerosene heaters contribute to a much greater extent to the degradation of indoor air quality than vented systems since all the combustion products are released directly into the living area. Depending upon occupant density, living space volume, extent of ventilation and infiltration, and use of unvented combustion sources, the concentration of CO, CO_2 and other combustion products in the indoor atmosphere can rise substantially above the normal range.

Unvented gas cooking is probably responsible for a large portion of the NO_x exposure in our population. A limited study by EPA showed that peak NO_2 concentrations up to 1 ppm and 1 hour averages of 0.25-0.50 ppm were reached in a closed kitchen with no external ventilation. These levels can affect sensory perception, especially dark adaptation, and produce eye irritation. In another study, gas stove emissions of NO, NO_2, and CO levels were measured in four homes and the following conclusions were drawn:

1. Gas stoves contribute NO_2, NO, and CO to indoor air, and the concentrations in kitchens respond rapidly to stove use, with a rough correlation between average NO_2 concentrations and average stove use.

2. NO_2 and NO were produced in roughly equal amounts and the indoor concentrations were invariably higher than those outside.

3. NO_2 concentrations in kitchens averaged over 0.05 ppm for the two week sampling period.

Gas water heaters and gas clothes dryers, although designed to vent to the outside, often have leakage around the flue collar which results

in increased NO_2 concentrations indoors.

Table I lists sources, possible concentrations, and indoor to outdoor concentration ratios of some combustion-generated pollutants in various environments. Available evidence suggests that higher population exposures to various pollutants occur indoors than outdoors and many constitute a sufficient threat to the general public health to justify further investigation and possible remedial action by the appropriate governmental agencies.

C. Health Effects of Combustion Products

Although the Academy expressed concern about the possible health effects of a number of pollutants, such as CO, NO_x, SO_2, HCHO, particulates and other organics, only CO and NO_x were considered in-depth in this section and are summarized below:

Carbon Monoxide

The documented adverse health effects of CO are due primarily to its ability to combine with hemoglobin (Hb). The Hb molecule in the blood serves as the vehicle to transport oxygen throughout the body. Oxygen and CO compete for the same binding site on the Hb molecule, and the affinity of Hb for CO is 210 times greater than for oxygen. Exposure to CO increases the carboxyhemoglobin (COHb) level and decreases the oxygen carrying ability of the blood with resultant cellular asphyxiation. In healthy people, a slightly elevated COHb level is probably functionally insignificant, with small physiological changes demonstrable only under extreme exertion. In individuals with a limited cardiovascular or pulmonary reserve, such as persons with heart or lung impairment, any reduction in the oxygen carrying capacity of the blood can be of great importance.

The tissues most sensitive to the adverse effects of CO appear to be the heart, brain, and exercising skeletal muscle. Although the no effect level for CO is still unknown, clinical data indicate that brief exposures resulting in COHb levels of 3-5% can produce the following cardiac effects:

1. restricted coronary blood flow;
2. oxygen deficiency in the heart muscle;
3. earlier onset of pain on exercise in persons with angina pectoris.

The threshold level (concentration below which effects would not occur) of COHb for cardiac effects is not known, but it would be less than 3%. COHb levels of 3-5% have also been associated with behavioral changes such as decreased vigilance. These COHb levels can be obtained

TABLE I

Sources, Possible Concentrations, and Indoor-to-Outdoor* Concentration Ratios of Some Indoor Pollutants

Pollutant	Sources of Indoor Pollution	Possible Indoor Concentration[a]	I/O* Concentration Ratio	Location
Carbon monoxide	Combustion equipment, engines, faulty heating system	100 ppm	>>1	Skating rinks, offices, homes, cars, shops
Respirable particles	Stoves, fireplaces, cigarettes, condensation of volatiles, aerosol sprays, resuspension, cooking	100-500 $\mu g/m^3$	>>1	Homes, offices, cars, public facilities, bars, restaurants
Organic vapors	Combustion, solvents, resin products, pesticides, aerosol sprays	NA	>1	Homes, restaurants, public facilities, offices, hospitals
Nitrogen dioxide	Combustion, gas stoves, water heaters, dryers, cigarettes, engines	200-1,000 $\mu g/m^3$	>>1	Homes, skating rinks
Sulfur dioxide	Heating system	20 $\mu g/m^3$	<1	Removal inside
Total suspended particles without smoking	Combustion, resuspension, heating system	100 $\mu g/m^3$	1	Homes, offices, transportation, restaurants
Sulfate	Matches, gas stoves	5 $\mu g/m^3$	<1	Removal inside
Carbon dioxide	Combustion, humans, pets	3,000 ppm	>>1	Homes, schools, offices

by continuous exposure to 15-30 ppm CO. Short-term exposure to high levels of CO can cause headache, dizziness, nausea, and possibly lead to collapse and death.

The current NAAQS for CO is 9 ppm for an 8-hour average exposure (which would limit estimated COHb levels to 1.4%), and 35 ppm for a 1-hour average exposure (which would limit the estimated maximum COHb level to 2.9%). These exposure levels should prevent adverse effects of CO exposure in patients with cardiac and peripheral vascular disease. The Occupational Safety and Health Administration (OSHA) presently has a workplace standard of 50 ppm for CO over an 8-hour period, while the National Institute of Occupational Safety and Health (NIOSH) recommended a workplace standard of 35 ppm for CO over an 8-hour exposure period.

Nitrogen Oxides

Inhalation of nitrogen oxides (NO_2 and NO) can induce blood effects similar to CO. Nitrogen oxides react with Hb, decreasing the Hb molecule's capacity to carry oxygen and thus, increasing cardiovascular stress. It appears that the effects of exposure to concentrations of NO_x of 3 ppm are physiologically comparable to exposure to concentrations of 10-15 ppm of CO. NO_x exposure may also affect the activity of several enzymes and produce substantial decreases in Hb and hematocrit (volume percentage of red blood cells) values which decrease the oxygen carrying capacity of the blood. Finally, NO_2 can produce transient and long-term damage to both small bronchial airways and alveolar tissue (lung sac tissue).

In homes with gas ranges, indoor concentrations of NO_2 are frequently twice as high as outdoor concentrations. Studies cited by NAS indicate that concentrations of NO_2 in the home can exceed the NAAQS of 0.05 ppm. Epidemiologic data on increased respiratory illness from chronic exposure to NO_2, although not conclusive, indicate that children living in homes with gas ranges have more respiratory illness and poorer respiratory function than children living in comparable homes with electric ranges. The presence of multiple pollutants (SO_2, sulfuric acid, sulfates, and nitrates) during these exposures and variable experimental conditions make it difficult to quantify the relationship between NO_2 alone and increases in respiratory disease. Table II summarizes the health effects of short-term human exposure to NO_2. Table III summarizes the health effects in animals and humans of exposure to NO_x over longer periods of time.

D. Recommendations

NAS identified the following research needs in the area of combustion processes:

1. Determine various sources and distribution of selected

TABLE II

Summary of Human Responses to Short-Term Nitrogen Dioxide Exposures Alone[a]

Effect	Nitrogen Dioxide Concentration		Time to Effect
	mg/m^3	ppm	
Odor threshold	0.23	0.12	Immediate
Threshold for dark adaptation	0.14	0.075	Not reported
	0.50	0.26	Not reported
Increased airway resistance	1.3-3.8	0.7-2.0	20 min[b]
	3.0-3.8	1.6-2.0	15 min
	2.8	1.5	45 min[c]
	3.8	2.0	45 min[d]
	5.6	3.0	45 min[e]
	7.5-9.4	4.0-5.0	40 min[f]
	9.4	5.0	15 min
	11.3-75.2	6.0-40.0	5 min
	13.2-31.8	7.0-17.0	10 min[g]
Decreased pulmonary diffusing capacity	7.5-9.4	4.0-5.0	15 min
Increased alveolar-arterial pO_2 difference	9.4	5.0	25 min[h]
No change in sputum histamine concentration	0.9-6.6	0.5-3.0	45 min

[a]Reprinted from National Research Council.[33]

[b]Exposure lasted 10 min. Effect on flow resistance was observed 10 min after termination of exposure.

[c]Effect was produced at this concentration when normal subjects and those with chronic respiratory disease exercised during exposure.

[d]Effect occurred at rest in subjects with chronic respiratory disease.

[e]Effect occurred at rest in normal subjects.

[f]Exposure lasted 10 min. Maximal effect on flow resistance was observed 30 min later.

[g]Also failed to find increased flow resistance over the range of nitrogen dioxide exposures from 5.1 to 30.1 mg/m^3 (2.7-16.0 ppm).

[h]Effect occurred 10 min after termination of 15-min exposure.

TABLE III

Controlled Exposure to Nitrogen Oxides

Species	Exposure	Health Effects Observed
Mouse (6-8 wk old)[19]	NO_2, 10 ppm, 2 h/d, 5 d/wk, up to 30 wk	Lung damage, suppressed immune function with chronic exposure, enhanced immune reactivity with shorter exposures
	NO, 10 ppm	Paraseptal emphysema, suppressed immune function with chronic exposure, enhanced immune reactivity with shorter exposures
Mouse[27]	NO_2, 0.5-28 ppm, 6 mo to 1 yr	Mortality after Streptococcus pyogenes, mortality increased with increasing dose and exposure time
Guinea pig[25]	NO_x, 1 ppm, 6 mo	Disturbed glycolysis, enhanced catabolic processes in brain, inhibited respiration, decreased brain aminotransferase activity, morphologic alterations in blood vessels
Human (asthma, n = 13; bronchitis, n = 7)[23]	NO_2, 0.5 ppm, 2 h	Lightness in chest, burning of eyes, headache, or dyspnea; pulmonary-function changes; nasal discharge
Human (asthma, n = 20)[35]	NO_2, 0.1-0.2 ppm	Increased bronchoconstriction[a]
Cat[26]	NO_2, 80 ppm, 3 h	Diffuse alveolar damage
Guinea pig[41]	NO_2, 0.506 ppm; NO, 0.05 ppm; 122 d	In lungs: decreased phosphatidylethanolamine, sphingomyelin, phosphatidylserine, phosphatidic acid, phosphatidylglycerol 3-phosphate; increased lysophosphatidylethanolamine
Mouse[12]	NO_2, 1.5-5.0 ppm, 3 h	Mortality in mice challenged with Streptococcus aerosol significantly increased at 2.0 ppm and above
Mouse[38]	NO_2, 0.5 ppm; 10, 12, 14 d	Average protein content of lungs significantly higher
Hamster[24]	NO_2, 30 ppm, 3 wk	Loss of body weight, increased dry lung weight, decrease in lung elastin and collagen

[a]Carbachol provocation.

[b]Elastin and collagen later returned to normal.

pollutants, measure ranges of concentrations, identify populations at risk, and characterize indoor pollutant emission sources under actual conditions.

2. Identify pollutants and their rates of emission from various types of fuel-fired appliances under various controlled chamber experimental conditions. These experiments should focus principally on gas and electric cooking appliances (for comparison) and supplemental heating systems such as natural gas, propane, and kerosene heaters and coal and wood-burning stoves.

3. Conduct comprehensive and systematic surveys to identify the range of combustion product concentrations encountered and the number of people exposed to them.

4. Conduct studies to determine the population exposure to NO_2 for short and long periods.

5. Determine the applicability of ambient air fixed location monitors for recording NO_x and CO concentrations indoors to assess individual exposures.

6. Investigate concentrations, mechanisms of formation and control methods for nitrosamines which can be formed during cooking and smoking.

7. Investigate the extent of emissions of polynuclear aromatic hydrocarbons which can be emitted from self-cleaning ovens and fireplaces.

8. Clarify several other areas dealing with decreases in pulmonary function and increases in respiratory tract infection rates among children living in homes with gas ranges vs. homes with electric ranges:

   a. Are effects due entirely to increased NO_2 concentrations from gas stoves or are they affected by the presence of other combustion effluents from stoves, such as CO, HCHO, and particulates?

   b. Are the effects related more closely to peak concentrations or long term average exposures?

   c. Are exposures to side stream cigarette smoke and to NO_2 additive or synergistic?

   d. Can exposure-response relationships be developed, and if so can an effective threshold concentration for peak or average exposure be set?

E. Health Sciences Comments

The importance of indoor combustion in relation to indoor air quality has been highlighted in the NAS report. Actual levels of most pollutants produced by specific sources are not well known, nor are the health effects of long term, low level exposure. In addition to pollutants such as CO, NO_x and HCHO which occur in the combustion process, several carcinogens, including benzo(a)pyrene, nitrosamines and polynuclear aromatic hydrocarbons, are also produced. These latter pollutants have not been investigated thoroughly and require greater attention due to their potential toxicological effects. Even the health effects of well known pollutants, such as CO and NO_x which were discussed in the NAS report, are not fully documented, and quantification of relationships between specific toxicant levels and health effects in the exposed population is not possible from currently available data.

Determination of various sources of combustion products, distribution of pollutants, their ranges of concentrations, identification of populations at risk, and characterization of indoor pollutant sources under actual use conditions are all projects recommended as necessary by NAS and endorsed by Health Sciences. Of prime importance to Health Sciences is the identification of specific consumer products as sources of pollutant emissions and levels of those emissions under home conditions. Both epidemiological and long term animal exposure studies are necessary to determine the health effects of the levels of pollutants which are identified. Based on ongoing Health Sciences studies on pollutants in indoor air, Health Sciences may make recommendations for additional toxicity testing.

F. Health Sciences Activities

During consideration of a mandatory standard for the unvented gas space heater (UVGSH), the potential problem of consumer exposure for prolonged periods to low levels of CO was raised. A comprehensive literature survey was conducted to determine the chronic effects of CO in man and animals. Subsequently, a HS report entitled, "Health Effects of Carbon Monoxide", was submitted to the Commission. This study concluded that COHb levels of 3-5% have been shown to produce both cardiac and behavioral effects, but more work needs to be done to elucidate the chronic health effects of CO exposure.

A study is currently under way at Lawrence Berkeley Laboratories to characterize the pollutants emitted by UVGSH and to determine if these appliances produce excessive levels of CO. Using the information of pollutant emission levels and the current information available on these pollutants' health effects, a preliminary health hazard assessment of UVGSH is planned for FY 83. Additional emission studies on gas ranges and ovens are planned for FY '83.

III. Home Materials

A. Introduction

The most widely recognized indoor pollutants released by home materials are formaldehyde and asbestos. Due to their toxic effects and potential for widespread consumer exposure, they comprise one of the most important groups of indoor pollutants. Formaldehyde is released from a wide variety of indoor sources, but primarily from urea-formaldehyde foam insulation, particleboard, and plywood. Formaldehyde released from these sources has resulted in skin, eye, and throat irritation as well as respiratory disorders and allergies. More importantly, recent studies have indicated that exposure of rats and mice to formaldehyde produces nasal cancer. The Committee concluded that a substantial number of persons are at risk of developing adverse health effects from indoor formaldehyde exposure.

Asbestos is found indoors as a result of its past use in insulation and decorative materials, and can be released through deliberate or inadvertant disruption of these materials. Given the formerly common use of asbestos in homes, schools, and other buildings, there is a need to identify structures where there is asbestos exposure so that what constitutes a risk to humans can be identified. While the extent of exposure of the general public is not as yet known, the adverse health effects of asbestos exposure are reasonably well known. Inhalation of asbestos fibers can lead to fibrosis of the lung (asbestosis), or a variety of malignant tumors, including mesothelioma, lung cancer, and gastrointestinal tract cancers. All of these diseases have been seen in humans who had chronic occupational exposures to asbestos fibers.

Formaldehyde and asbestos received in-depth toxicological evaluation in the NAS report. Other pollutants, such as the large class of organic contaminants from products containing plastics and resins, as well as those associated with particulates and dusts, may have very significant impacts on human health; however, because evidence of adverse health effects and/or exposure is meager, NAS concluded that a comprehensive review of these latter chemicals was not feasible.

B. Sources

Indoor concentrations of formaldehyde are almost always higher than outdoor concentrations. Variations in indoor concentrations are not well understood, as emission rates from the various sources are not well quantified, and comprehensive indoor monitoring data for U.S. homes are few. Home materials involved as major indoor sources of formaldehyde

include urea-formaldehyde foam insulation, plywood, and particleboard, with floor coverings, textiles, and carpet backing acting as less important sources. Indoor concentrations often exceed 0.1 ppm, and may exceed 3 ppm in houses with urea-formaldehyde foam insulation. In homes with formaldehyde-containing products, a 24 hour average of 0.05-0.2 ppm is common. The Committee stated further that in buildings in which particleboard and plywood products are used extensively for walls, floors, and furniture, low air exchange rates and the high surface to volume ratio of these products can result in formaldehyde concentrations of 1 ppm or higher. Emission rates from these sources also depend on a number of other factors, such as the manufacturing process, ambient temperature, age of the sources, and humidity.

Asbestos (and fibrous glass) is also present in the environment of homes as well as schools and other public buildings. Indoor sources of these fibers are thermal and acoustic insulation, decorative materials, fireproofing, and textiles. Abrasion, mechanical vibration, and deliberate disruption can lead to increased fiber concentrations, and a small number of studies have documented fiber counts in association with normal building use. Airborne fiber concentrations range from 0 during background and routine activity, to over 100 fibers per ml. during the stripping of ceiling material. However, routine activities can result in contamination in some situations. For example, a school population's routine activity, along with custodial work, in a building with accessible asbestos surfaces, may cause contamination. Vandalism and maintenance procedures can also involve direct contact with asbestos surfaces; custodial work can cause disturbance and reentrainment of accumulations of fibers. Home exposures could result from aging, cracking insulated pipes, ceiling tiles, and spackling compounds, especially in homes located in northern climates and built before 1950. Uncontrolled removal of asbestos surfaces during renovation can cause high fiber concentrations, exposing the renovation workers as well as the routine building user.

C. Health Effects

Formaldehyde

The total number of people exposed to formaldehyde who manifest adverse health effects is difficult to determine. There is evidence that such responses may occur in a substantial proportion of the exposed population. The Committee estimated that perhaps 10-20% of the general population may react to formaldehyde even at concentrations of 0.25 ppm or less, although such reactions would be more severe at higher concentrations. On the basis of these estimates and the number of persons exposed to formaldehyde indoors, the Committee concluded that "a substantial number of persons are at risk of developing adverse health effects associated with formaldehyde".

The carcinogenic effects of formaldehyde exposure in humans have not been fully assessed. Studies in rats and mice done by the Chemical

Industry Institute of Toxicology have shown that formaldehyde produces nasal carcinoma in rats after several months of exposure at 15 and 6 ppm, and in mice at 15 ppm. Dose-related cellular changes in the nasal mucosa of rats were also observed as were excess nasal tumors at all exposure levels. Formaldehyde has also shown mutagenic activity in a variety of microorganisms and some insects.

Adverse health effects due to formaldehyde may occur after exposure by inhalation or dermal contact. Symptoms reported by residents of homes containing formaldehyde-yielding products include skin, eye, and throat irritation, nausea, headache, rash, tiredness, and thirst. Respiratory disorders and allergies have also been associated with high formaldehyde concentrations. The Committee felt that ascribing specific health effects to specific concentrations is difficult, however, because people vary in their subjective responses and complaints, and the threshold for response is not constant among all segments of the population (e.g., hypersensitive persons or those with hyperreactive airways).

The principal health effect noted at low concentrations of formaldehyde is irritation of the eyes and mucous membranes. A wide range of concentrations of airborne formaldehyde has been reported to cause specific human health effects. Human eye irritation is produced by concentrations in the 0.05-0.5 ppm range. Nasal airway irritation and sore and dry throat usually appear in the same concentration range. Formaldehyde concentrations as low as 0.1 ppm can produce upper airway irritation. Eye and upper airway tolerance may also develop with continued exposure, but such tolerance is lost when there is a break in exposure.

Lower airway irritation, characterized by cough, chest tightness, and wheezing, is reported especially at exposures of 5-30 ppm, but may occur at lower concentrations. Accumulation of fluid in the lungs, pneumonitis, and death can result from very high concentrations of formaldehyde, probably in the 50-100 ppm range. Formaldehyde has also been shown to aggravate or, in some instances, cause bronchial asthma in humans. Again, concentrations at which attacks occur are highly variable, and the exact mechanism of the asthma syndrome is not known. Additionally, skin contact with formaldehyde has been reported to cause a variety of cutaneous problems in humans, including irritation, allergic contact dermatitis, and hives.

Asbestos and Other Fibers

Current knowledge of the adverse health effects of fibrous home materials is reasonably complete only for asbestos. Other materials, such as fibrous glass and rock wool, are becoming more widely used, but little is known about their health effects in humans. Inhalation is the major route of entry of these fibrous particulates, although inadvertant

ingestion and subsequent dispersion through the gastrointestinal tract wall into the peritoneal cavity and blood stream has been shown in animals.

In humans, exposure to asbestos results in both non-malignant and malignant health effects. The most important of the non-malignant health effects is the fibrotic process occuring in the lung known as asbestosis, the leading cause of death among some occupationally exposed groups. Asbestosis usually develops only after considerable time, often more than 20 years. However, exposure need not be continuous during this time; a relatively brief exposure may be sufficient to cause disease many years later.

A variety of malignant tumors are associated with exposure to asbestos: mesothelioma, lung cancer, and gastrointestinal tract cancers, particularly of the colon, rectum, stomach, and esophagus. These diseases also demonstrate the principle of latency; in general, 15-20 years must pass before the signs of marked asbestos-related disease are detected. It is also now evident that the severity of asbestosis and the risk of developing lung cancer or mesothelioma depend on the total exposure to fibers. An exposure might be brief, but if intense enough, disease may result following the latency period.

No epidemiologic investigation has demonstrated substantial health hazards related to other fibrous materials used as asbestos substitutes in construction. However, these materials, such as glass fiber, rock wool and slag wool, have been shown in animal injection and implantation studies to be capable of producing lung fibrosis and mesothelioma. Animal experimentation with asbestos has also produced all the major health effects seen in humans.

D. Recommendations

The NAS Committee on Indoor Pollutants proposes a comprehensive list of research recommendations for home materials and their contribution to indoor air pollution as follows:

Formaldehyde

Monitoring formaldehyde is difficult due to the influences of temperature, humidity, and analytic problems affecting its detection. Both continuous and passive monitors are needed with sensitivities in the range of 10-30 ppb. A systematic study of formaldehyde concentrations in a variety of indoor locations is needed to estimate potential exposure of humans, identify indoor sources of formaldehyde, and assess emission rates. Long term effects in humans continuously exposed to low concentrations of formaldehyde need to be studied, especially to assess carcinogenic potential. Epidemiologic investigations are needed to assess human health effects, magnitude and duration of exposure

and influence of cigarette smoking habits. Animal studies to validate findings of carcinogenicity should be repeated, including mutagenic, embryotoxic, and teratogenic studies. Exposure-effect relationships and the mechanisms involved in the biologic effects require animal toxicologic research. In humans, the mechanisms of airway and target cell responses need to be evaluated as to sensitization and adverse effects in susceptible populations.

Asbestos

There is a need for a systematic survey of the distribution, integrity, and concentrations of asbestos in buildings known to contain asbestos. This survey will require new instruments to record fiber counts continuously with size determination and fiber identification. Synergistic and interactive toxic effects of asbestos fibers in combination with other air pollutants should be examined in animal toxicologic and mutagenicity studies. The incidence of mesothelioma in humans should be monitored as an indicator of general population exposure. Guidelines should be developed for the control of asbestos exposures during building maintenance, renovation, and reconstruction.

E. Health Sciences Comments

The emphasis placed on the health hazards posed by formaldehyde and asbestos and the recommendations made by the NAS are appropriate, except that with regard to validation of the carcinogenicity of formaldehyde, no further studies are needed. Of particular interest were the NAS estimates of the percentage of the population that may be more susceptible to the adverse acute effects at low concentration of formaldehyde to be 10-20% of the U.S. population. This estimate is in agreement with both of the previous NAS reports on formaldehyde. The report also singled out asthmatics as a population liable to be particularly susceptible to low levels of formaldehyde. This statement also agreed with that made by the previous NAS report on formaldehyde and other aldehydes. It should be noted that these estimates do not include other groups previously identified by NAS as likely to be more susceptible to formaldehyde, such as all of those with hyperreactive airways and persons sensitized to formaldehyde. Thus, the total of population groups with increased susceptibility to low level exposure of formaldehyde may actually be higher.

The Committee on Indoor Pollutants also expressed concern about the possible carcinogenicity of formaldehyde. Although the Committee did not actually evaluate the data demonstrating the carcinogenicity of formaldehyde, various other groups have evaluated these data. These include the Federal Panel on Formaldehyde, the Panel at Mt. Sinai, the New York University group, and most recently, IARC. All of these groups concluded that formaldehyde was carcinogenic to animals and, with the exception of the Mt. Sinai Panel, concluded that it should be presumed to be a carcinogenic risk to humans. The Mt. Sinai Panel stated that, based on the available carcinogenicity data, human exposure to formaldehyde

should be "reduced or eliminated". With regard to asbestos, Health Sciences agrees with the NAS conclusion that although knowledge of adverse health effects from exposure to asbestos in the house was not extensive, there was sufficient knowledge of the adverse health effects of this chemical overall to justify "a cautious approach to any exposure".

F. Health Sciences Activities

Health Sciences has been continually updating the toxicological data on acute effects, sensitization, and carcinogenicity of formaldehyde, as well as working with scientists from the National Academy of Sciences, the National Toxicology Program, Department of Energy, Environmental Protection Agency, National Institute for Environmental Health Sciences, National Cancer Institute, Occupational Safety and Health Administration, National Institute for Occupational Safety and Health, and academia.

Studies are in progress to determine the formaldehyde levels emitted from particleboard, plywood, paneling, carpets, drapes and other textiles at Lovelace Biomedical and Environmental Research Institute, and textiles at USDA Cotton Research Laboratory. Potential human exposure to formaldehyde from clothing is also being examined at the USDA Laboratory.

Health Sciences is evaluating the formaldehyde exposure to students in biology laboratories from specimens preserved in formaldehyde. CPSC staff is currently evaluating available international product standards for some of the products that emit formaldehyde, such as particleboard, plywood, and textiles. The suitability of substitutes that do not emit formaldehyde may also be explored. Finally, CPSC has proposed a ban on urea-formaldehyde foam insulation.

CPSC has banned the use of asbestos-based spackling and patching compounds and asbestos fibers used decoratively with gas fireplace logs. General use garments containing asbestos are also banned under the Federal Hazardous Substances Act. A corrective action plan for hair dryers with asbestos linings was developed by CPSC and participating companies to include voluntary recall, replacement, or repair. At present, the release of asbestos fibers from a number of consumer products is being tested. In addition, the Commission staff is working with EPA to prepare and publish a booklet entitled "Asbestos in the Home".

IV. Household Chemicals

A. Introduction

Several of the major organic pollutants (hydrocarbons, chlorinated hydrocarbons, aromatic hydrocarbons, alcohols, aldehydes, ketones, ethers, and esters) found in the indoor atmosphere result for the most part, from the occupants' use of household consumer products as well as from the presence of furnishings and building materials. Although there is little human epidemiologic evidence implicating organic indoor pollutants (other than formaldehyde) as human health hazards, animal studies and human experience reveal a potential for adverse health effects. These effects are diverse and may include neurologic dysfunction, liver, kidney and heart damage, reproductive toxicity (including teratogenesis) and tumor production. Since data on household chemicals is rather limited, the pollutants were not discussed in depth by NAS.

B. Sources

NAS has estimated that the average U.S. residence contains 45 aerosol sprays at any given time. Aerosols are widely used for packaging consumer items such as cleaners, waxes, polishes, paints and adhesives. Chemicals, both volatile and non-volatile, are released into the indoor air whenever these products are used, thus making them available for direct inhalation. Other non-aerosol consumer products, such as paints, detergents, paint strippers, cleaners, dry-cleaning agents and deodorizers introduce primarily volatile chemical pollutants into the indoor air. Additionally, products such as building materials, plastics, textiles, wood furnishings, and carpets can also release volatile chemicals into the living space over long periods of time. Information on the presence of many of these chemicals in indoor air, as well as their contribution to indoor air pollution, is currently unavailable. The NAS Report concluded that many household products may emit gaseous and particulate contaminants into the indoor environment during their use or even during storage.

C. Health Effects

Assessment of the potential health effects attributable to organic chemical contaminants indoors, however, is complicated by a lack of knowledge of the likely levels and durations of consumer exposure to these chemicals. In addition, little is known about possible chemical reactions that may occur between these contaminants, or about additive or synergistic effects that they may exert on human health. Willful abuse of aerosols or careless use of solvents in enclosed spaces has resulted in acute and chronic disorders and even death. As pointed out by NAS, halogenated hydrocarbons can induce cardiac arrhythmias and may eventually lead to coronary heart disease with prolonged exposure. Liver and kidney damage are also caused by the chlorinated hydrocarbons as well as by other solvents.

The wide variety of the chemicals in consumer products makes it difficult to discuss all the possible adverse health effects of household organic chemicals. Also, since little exposure data were available for these chemicals, the NAS did not discuss their health effects to any significant extent.

The health effects of a number of these chemicals have, however, been reviewed by Health Sciences and are briefly discussed below. These include low molecular weight hydrocarbons (propane, butane and isobutane), higher molecular weight hydrocarbons (n-hexane, heptane and octane), chlorinated hydrocarbons (methylene chloride, perchloroethylene), aromatic hydrocarbons (xylene, toluene) and other organic solvents found in consumer products.

Aliphatic hydrocarbons such as propane, butane and isobutane, with less than five carbon atoms, are gases at room temperature Their most common use is for cooking and heating. These gases may cause asphyxia by displacing oxygen from a closed environment, but usually are of more concern as fire hazards. Higher molecular weight hydrocarbons including n-hexane are used as solvents in aerosols and in a wide variety of consumer products, such as spray adhesives, rubber cements, and thinners, which are used in art work. Acute exposure to n-hexane for example can temporarily impair neurological function. Cutaneous exposure to high levels may lead to degeneration of peripheral nerves. An estimated concentration of 200 ppm to 600 ppm during a period of 3 to 6 months represents the minimal inhalation exposure to n-hexane which has been reported to cause neurotoxicity in humans.

Chlorinated hydrocarbons, including primarily methylene chloride and perchloroethylene, are used as solvents in consumer products. These solvents have been associated with neurological disorders, liver and kidney damage, and cardiac rhythm alterations following acute and chronic exposure. Some chlorinated hydrocarbons are carcinogenic.

Methylene chloride is used in paint removers and as a propellant in aerosols. Acute exposure affects the central nervous system (CNS) and may produce cardiac injury. It is metabolized to carbon monoxide which combines with hemoglobin, thus hindering oxygen utilization and stressing the cardiovascular system. Chronic exposure may result in liver damage. Long term animal studies are being conducted by NCI to determine the carcinogenic potential of methylene chloride in rats and mice. Preliminary results from a study by the Dow Chemical Company indicate that methylene chloride induces cancer in rats.

Perchloroethylene (PERC) is widely used as a dry-cleaning agent. In addition to CNS effects, acute exposure causes eye irritation in animals and skin sensitization in humans. Repeated exposure can produce liver and kidney damage and cardiac effects. It has also been shown to be a liver carcinogen in mice.

Aromatic hydrocarbons (e.g., xylene and toluene) are frequently found in consumer products such as paints, varnishes, glues, enamels, and lacquers. Acute exposures have been associated with CNS effects and eye, skin, and respiratory tract irritation. Chronic exposure may cause liver and kidney damage. Another member of this class, benzene, which is not currently used in consumer products, is a carcinogen in humans and animals.

Other organic chemical pollutants, such as alcohols, ketones, ethers, and esters, are also widely used in consumer products. Alcohols, for example, are used in such products as aerosols, window cleaners, paints, paint thinners, and adhesives. Alcohols irritate mucous membranes and produce depression of the central nervous system. Methyl alcohol affects the retina at higher concentrations causing blindness. Ketones such as acetone, methyl ethyl ketone, and methyl isobutyl ketone are used in lacquers, varnishes, polish removers, and adhesives. These cause CNS depression and pulmonary vascular dilation. Additionally, methyl n-butyl ketone is converted to 2,5-dione which causes peripheral nerve degeneration. Ethers, including methyl-, ethyl-, and butyl ethers of ethylene glycol, dimethylether, tetrahydrofuran, and dioxane, are used widely in solvents. Exposure to ethers causes headaches, weakness, drowsiness, disorientation, and lethargy. Dioxane produces dermal and eye irritation, liver and kidney damage, and cancer in animals. Esters, such as ethyl acetate, butyl acetate and ethyl butyrate, are used in plastics, resins, plasticizers and lacquer solvents. Esters can irritate the eyes, nose and throat; some cause CNS depression. The alkyl phthalates, some of which cause cancer in animals, are also important esters.

D. Recommendations

NAS recommends that the contents of consumer products should be investigated, and the chemical constituents tested for their toxic, mutagenic, carcinogenic, and teratogenic properties. Synergistic and interactive effects with other indoor pollutants should be tested. In addition, information is needed on the identity and on the levels of these pollutants in indoor ambient air. NAS also recommends that consumer products whose use involves the release of gaseous and particulate materials to the indoor atmosphere should be labeled with their components. Warning labels for consumer products that can seriously pollute the indoors should state that they are to be used only in areas with adequate ventilation.

E. Health Sciences Comments

Health Sciences agrees with the NAS recommendations. There is a great need to determine the organic chemicals to which consumers may be exposed indoors. We need, in addition, to identify the health effects

associated with consumer exposure to these chemicals and possible interactions that may occur. For chemicals identified as being toxic there is a need to identify sources of these chemicals in the home and the relative contributions of each to the total exposure. With many of these chemicals very little is known about the health effects of long term low level exposure. Health Sciences investigation should not limit itself to organics released by the use of consumer products, but should consider those released by household structural elements as well as furnishings. The determination of the synergistic and additive effects exerted by these chemicals on health is especially important.

F. Health Sciences Activities

Health Sciences conducted a comprehensive review of available toxicological data and simulated consumer exposure which will form part of a supplemental briefing package on control options for n-hexane, scheduled for Commission consideration in 1982. Health Sciences has also prepared a report on the health effects of perchloroethylene, a chemical to which consumers may be exposed in coin-operated dry-cleaning establishments. The health hazard and risk associated with perchloroethylene exposure will be assessed when the results of the NCI bioassay become available in 1982. Health Sciences has also determined the health effects likely to be associated with consumer exposure to benzene.

The health effects of methylene chloride, xylene, and other solvents in aerosol products will also be evaluated in 1982, as will the health effects of two or three selected organic chemicals present in indoor air. This latter project will focus on volatile organic chemicals identified in indoor air as a result of the work currently being conducted for Health Sciences by the Oak Ridge National Laboratory. Based on the above information, Health Sciences will prioritize the list of chemicals for further investigation. Health Sciences is also defining the health effects of the phthalates and is determining potential exposure to these chemicals in the home. It should be recognized that the Health Sciences efforts in this area are very limited and are concerned only with chemicals found in specific consumer products.

V. Miscellaneous

A. Introduction

In addition to the indoor air pollutants considered under the topics previously discussed, several other contributors to the deterioration of indoor air quality were reviewed by the NAS Committee on Indoor Pollutants. Among these are microorganisms, biological allergens, moisture, temperature, and odor. Microorganisms are associated with human activity and the presence of domestic animals indoors. Microorganisms tend to multiply in the presence of increased humidity; thus reduced ventilation could increase the concentrations of microorganisms. Many of these microorganisms can produce infection, disease, or allergic reactions, especially in the respiratory tract. Respiratory infections are an important cause of morbidity and result in discomfort, absenteeism, and lost earnings. Excess relative humidity indoors can also contribute to increased deterioration of building materials. Other disruptions in indoor air quality control, such as extremes of temperature and the buildup of odor and particulates, may result in physical manifestations such as decreased appetite, nausea, headaches, sleep disturbances, and eye, skin, and respiratory irritation.

B. Sources

Among the pollutants of indoor air identified by the Committee are biological particles produced when people cough, sneeze, spit, talk, or blow their noses. The sparseness of satisfactory measurement methods and the resulting lack of an adequate quantitative data base pose major problems in assessing indoor biogenic pollutants. Biogenic pollutants bear complex and varied organic structures which make automatic chemical assay impossible. Airborne contagion is the mechanism of transmission of most acute respiratory infections, and these infections are the greatest of all causes of morbidity. Several airborne allergens are found in enclosed spaces; house dust, pollen, molds, animal dander and excreta, and insects are important factors in provoking allergic symptoms. The individual contributions of these factors to the overall problem of allergic diseases is entirely speculative at present.

Substances that make their presence known primarily by their ability to evoke odor sensations generally arise from humans and their activities as well as from building materials and furnishings. Completely odorless conditions occur indoors only rarely. Odors due to humans arise from routine indoor activities such as occupancy, cooking, bathroom use, etc.

Water vapor in confined spaces is a product of metabolic and respiratory processes, indoor combustion, and evaporation. Decreases in ventilation tend to increase the indoor relative humidity. Excess

water vapor adsorbs or condenses on dry or cold surfaces, giving rise to increased deterioration and corrosion of building materials and furnishings as well as possibly increasing the growth of microorganisms.

C. Health Effects

There is evidence that a number of contagious disease organisms (e.g., those associated with influenza, Legionnaires' disease, the common cold etc.) are capable of airborne transmission indoors. Although indoor transmission is not fully understood, the generally accepted basis for transmission models is the droplet nucleus mechanism whereby respiratory droplets which are released upon sneezing or coughing remain airborne and are dispersed rapidly throughout the air of a room by air currents. Thus, infectious contact by airborne transmission requires proximity in time and space between host and victim, unless the building uses a shared, recirculating ventilating system.

A small number of airborne allergens are also found in indoor environments, but the health effects of exposure to them alone are difficult to estimate. Allergic reactions can occur on the skin and in the nose, airways, and alveoli. The direct action of allergenic irritants on the skin produces acute contact inflammation. Allergic responses to inhaled materials cause a local inflammatory reaction that can affect the nose (allergic rhinitis) or the airways (allergic asthma). Particles found in the indoor environment cannot be assumed to have indoor origins, as a large proportion of indoor particles have natural sources (e.g., pollen, fungi, insects, and algae). To the extent that infectious and allergenic microorganisms remain viable and airborne, reductions in ventilation rates will tend to increase concentrations and the probability of infection.

A final group of air pollutants emanating from indoor sources are those which affect human comfort and welfare rather than health. The human response of discomfort is evoked as a result of undesirable sensory stimuli, such as malodors and extremes of temperature and humidity, and it may be relieved by attenuation of the stimulus. Discomfort is a sensitive indicator of the need for adjustments in environmental quality control, and the smell of the indoor environment, for example, is a measure of such environmental quality. There is little information regarding whether long term exposure to sources of discomfort will eventually cause adverse health effects.

D. Recommendations

Little is known about the sources, concentrations, and survival rates of many aeropathogens indoors. Relationships among the incidence of respiratory infections, concentrations of aeropathogens, and air exchange rates in buildings should be examined.

A sample of commercial, institutional, and residential buildings should be evaluated for types and concentrations of aeropathogens under a variety of conditions of occupancy, activity, ventilation, humidity, temperature, and contaminant control, paying special attention to air cleaning systems, air-heat exchangers, humidifying systems, and air conditioning systems.

Further work is needed to characterize the size distribution of allergen particles and their sources. Epidemiologic and immunologic studies are needed to clarify their exposure-effect relationships. Additionally, the joint action of biologic and non-biologic agents should be explored in animal toxicology studies.

E. Health Sciences Comments/Activities

Considerable discussion was devoted in the report to the topic of microorganisms and allergens. The report concludes that while the health effects of this particular category are not well defined, it is reasonable to assume that decreased ventilation would increase the concentration of microorganisms and the likelihood of respiratory infection, allergy, and undesirable health effects, such as headache, nausea, eye and skin irritation, and insomnia. In addition, effects on human welfare, such as discomfort, decreased productivity, soiling, and corrosion, may result from poor indoor air quality. Although Health Sciences has little involvement in this area since these effects are, for the most part, not related to specific products, we still have an interest in the data that may result from these recommendations in order to better understand indoor air quality. This area does, however, have relevance for some specific products such as urea formaldehyde foam insulation, which is purported to support the growth of microorganisms and dust mites. Also, for example, a better understanding of the effects of humidity could assist the Commission in advising consumers on the best use of home humidifiers.

VI. Indoor Air Quality Research Needs Identified by the Interagency Research Group on Indoor Air Quality

In addition to NAS, two other groups have been involved in assessing the problems of indoor air quality. These are the Interagency Research Group on Indoor Air Quality and the Ad Hoc Task Force on Indoor Air Pollution. Section VI presents the recommendations of the Interagency Research Group; the recommendations of the Ad Hoc Task Force follow in Section VII. It is of interest to note that the recommendations of both of these groups are similar to those of NAS.

The report of the Interagency Research Group on Indoor Air quality, published in April, 1981, summarizes the Workshop on Indoor Air Quality Research Needs (December 3-5, 1980), and presents the findings and recommendations that were developed. This report is divided into four sections corresponding to the four subgroups of the workshop and was intended to help prepare an integrated Federal research program on indoor air quality.

A. Monitoring

The monitoring subgroup identified three unifying themes that need to be kept in mind in developing monitoring efforts: a) the total exposure concept, i.e., 24 hour exposure, should be an integral part of all monitoring efforts, as a person's health can be affected by indoor, outdoor, and in-vehicle exposure; b) careful planning of field monitoring is needed to ensure that all pertinent information necessary to achieve the stated objectives is collected; and c) the "sick building" concept, i.e., buildings in which considerable proportions of their inhabitants are affected adversely, is important because such buildings present an opportunity to study a condition that affords a high probability of source identification. The group also stressed that coordination and interagency cooperation is required as a result of the complexity of the indoor air quality problem.

More specific monitoring recommendations are presented according to specific pollutant classes as follows:

Organics

The method for broad-spectrum sampling needs to be improved and standardized to allow comparison of results. Once the method is improved, there is a need to study problem buildings, ventilation rates, and sources. A monitoring study of organic emissions from building materials is recommended, including examination of ventilation rates, temperature, and humidity effects. Studies of wood burning stoves, fireplaces, kerosene space heaters, unvented heaters, and correlations of breath and blood levels of organic pollutants with indoor exposures are recommended. Organic particulate matter must also be characterized; adequate technology is not now available. Monitoring for pentachlorophenol, especially in log homes, should be conducted. Finally, the organics subgroup recommended initiation of a study of mutagens derived from cooking.

Nitrosamines and Odors

Monitoring studies should examine nitrosamine concentrations with respect to the following variables: gas vs. electric cooking, vented vs. unvented hoods, the effect of tobacco smoking on indoor concentrations, and the effectiveness of charcoal filters in reducing nitrosamine concentrations. A monitoring program is needed as a first step in learning the extent of the problem associated with hydrazine, a possible product of reactions involving bleaches and washing materials. The group also urged that a comprehensive investigation of the increasing number of complaints about odors be initiated.

Aerosols and Asbestos

Instrumentation for aerosols and asbestos is inadequate. A personal monitor is needed for respirable particles, and a monitor is needed that gives an instantaneous readout of mass. The highest priority should be given to a total exposure monitoring study to measure indoor, outdoor, and intransit exposures. Controlled or chamber experiments for sources such as tobacco smoke, cooking, vacuum cleaning, and wood burning should also be carried out. A third priority is for site-specific studies on office buildings, residences, and automobiles. For asbestos, instrumentation is not adequate for airborne analysis; continuous instrumentation and fiber counts with size determination and fiber identification are needed.

Criteria Pollutants

These include carbon monoxide (CO), nitrogen oxides (NO_x), sulfur oxides (SO_x) and ozone. CO is the only pollutant for which instrumentation appears adequate and for which a large scale study could now be undertaken. Recommended studies include gas heating, cooking, transportation sources, and 24-hour exposure studies. Also recommended is a chamber study that would involve a single specially designed house to study combustion and appliance emissions of NO_2.

Formaldehyde

The formaldehyde subgroup determined that sampling methods are inadequate with regard to population exposure and source assessment, where a desirable level of detection would be 20 ppb rather than the 200 ppb level currently available. The group also recommended examining correlations between formaldehyde levels, sources, and ventilation effects.

B. Health Effects

Research needs were identified on a substance-by-substance basis following some general comments. The group emphasized, however, that indoor air quality is the result of a very complex interaction of chemical and physical factors, and that eventually the problem of

multiple risks and synergism will have to be resolved. With regard to target organs, the respiratory system was felt to be the most vulnerable; however, the nervous system and behavioral effects should also be a matter of concern and study. The group also stated that in order to study the effects of toxic substances found in the indoor environment, two classes of instruments are needed: those that measure peak short-term exposures, and long-term integrating instruments. Recommendations for specific pollutants are as follows:

Formaldehyde

There is a need to characterize rates of formaldehyde emissions and factors affecting these rates from structural and consumer products in buildings. Also needed is toxicologic research concerning the carcinogenic effects of low levels of formaldehyde, the mechanism of sensitization, and identification of sensitive populations. Additionally, epidemiologic studies are required to better define the relationship between health effects induced by formaldehyde and levels of indoor exposures.

Combustion Products

The combustion products of most concern are CO, NO_x, formaldehyde, aromatic hydrocarbons, and particulates. Monitoring, epidemiological, and chronic exposure studies are most critical.

Biological Pollutants

The Health Effects group considered biological pollutants a high priority research need from both the allergenic aspect and the infectious agent aspect. Hypersensitivity syndrome from continued exposure to allergens, for example humidifier fever, requires further study. Incidents of infectious disease should be correlated with levels of air exchanges in buildings.

Organic Pollutants

With the exception of formaldehyde, these chemicals were assigned lesser priority since knowledge of exposure and hazard is limited. The group recommended additional studies for this class of chemicals to better define the potential problems posed by exposure. There is a specific need for consumer product emission data and appropriate labeling to prevent misuse. A central repository of toxicologic data should be provided for consumer products.

Particulates

Basic research is needed to characterize and measure indoor particulates wihin defined environments, including pathways of particulates into the indoor environment.

C. Control Technology

Recommendations of the subgroup for control technology research strategies were divided into four categories as follows:

Ventilation

There is a need to determine sources and generation rates, and to reexamine the current prescriptive ventilation rates to ascertain if they provide adequate levels of contaminant control Techniques for use of air-to-air heat exchanges must also be evaluated. An inexpensive means of measuring the actual ventilation rate experienced by a building over a period of time is also needed. Finally, the concentrations of pollutants as a function of the ventilation rate should be determined.

Source Removal and Exclusion

Source removal and exclusion are the most effective ways in which to control a pollutant. A prerequisite of this technology is identification of sources and rates of emission. Also needed is a comprehensive listing of coating and encapsulation compounds as a basis for identifying and evaluating additional applications. Further research is needed on the development of construction technologies that will exclude pollutants.

Contaminant Removal

There is a need for the development of energy efficient kitchen range hoods which are effective for actual contaminant removal via the use of charcoal or other types of filters. There is also a potential for implementing technology transfer in the area of air cleaning mechanisms and equipment used by industry.

Product Substitution

This group did not identify specific research needs in the product substitution area as it is a product specific method of control and few data are available.

D. Health Sciences Comments/Activities

Health Sciences agrees in essence with the recommendations proposed by the Interagency Research Group which basically call for increased monitoring, ventilation, and health data to better define indoor air and the possible health problems posed by indoor exposure. Health Sciences feels, however that organic pollutants as a whole should be given higher priority than given by the interagency group, especially in light of health problems identified for one member of the class, formaldehyde. As with the NAS recommendations, the Commission would place less emphasis on biological pollutants. Relevant CPSC research has been discussed previously (Sections II-V).

VII. Report of Ad Hoc Task Force on Indoor Air Pollution, Dudney, C.S., and P.J. Walsh, eds. April, 1981.

The Ad Hoc Task Force on Indoor Air Pollution began life as the Ad Hoc Task Force on the Epidemiology of Formaldehyde. The Task Force was originally commissioned in 1979 to address the question of whether DOE should promote the use of UFFI in view of possible human health effects due to formaldehyde. As the question was actually only one of many complicated issues related to indoor air pollution in general, the Task Force enlarged its scope to cover these issues in a series of meetings between May, 1979, and March, 1980. The document produced by the Task Force provides a brief background and overview of indoor air pollution and recommendations concerning these issues. It was intended to be used to focus attention on the issues of indoor air pollution and on appropriate paths to solutions. As in the previous section, (VI), only the recommendations of the Task Force will be discussed as follows:

The Task Force recommended two major inputs to assess the overall health impact: an exposure assessment and a health effects assessment. The exposure assessment would require data on the types and concentrations of pollutants, and could, according to the Task Force report, be achieved through the use of a large number of personal air monitors. This approach would account for human activity patterns and would more accurately reflect exposure histories. Currently however, a device to measure the full spectrum of pollutants in the indoor environment is not available. The assessment of population exposure must, in addition, include quantification of sources and strengths, ventilation rates, definition of a set of representative residence types, definition of a set of climatic regions, an enumeration by census of the American public with respect to residence type and climatic region, and quantification of hazardous agents in typical residences. Based on the above information, appropriate exposure models need to be developed to accurately predict exposure under a variety of conditions.

With regard to the health effects assessment, there is no direct, experimental method by which to establish quantitative human health risk estimators for in-home exposure. Therefore one must rely on risk estimators derived by extrapolation from other experimental studies, such as: chemical structure-activity relationships, microbiological studies, mammalian-cell culture studies, animal studies, and epidemiological studies. The Task Force also pointed out the possibility of synergistic interactions among the complex mixture of compounds found in the indoor environment and the need to consider these in any epidemiological study of indoor air pollution. In addition, the health effects assessment should identify the most critical information needed with respect to the relative impacts of the buildup of toxic materials due to reduced air turnover rates.

In order to better define the potential health problems associated with indoor air, the Task Force report advocated the need for epidemiological studies to assess the health impact of indoor air pollution. They state that in order to successfully perform such studies, the following questions need to be addressed:

1. Do problems occur generally in buildings with low air turnover rates?

2. Do problems occur in homes that use natural gas as opposed to electricity for cooking?

3. What is the frequency of problems?

4. What would be a representative sample of the population at risk?

5. What building types (including furnishings) represent the range of variation?

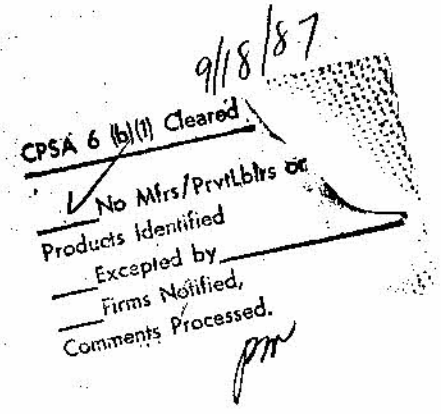
Finally, the Task Force recommended that a national policy on indoor air quality be developed and promulgated, the Federal authority for establishment of indoor air quality guidelines and recommendations be clarified, and a lead agency be identified to develop a coordinated plan for studies of indoor air pollution with sufficient resources for its implementation.

Health Sciences Comments/Activities

Health Sciences agrees that there should be a unified approach to the indoor air pollution problem and that the activities of the various agencies need to be coordinated. Further, however, Health Sciences feels the federal efforts must be coordinated with those of the private sector. Health Sciences also agrees with the need for the kinds of data identified as being necessary for exposure and health assessments, as well as with the need for these assessments. These data needs have also been identified by the NAS (II-V) and by the Interagency Group (VI). The Task Force approach however, is a complete assessment approach; it would be better at this time to perform more limited research on what is actually present in homes and then to proceed to investigate a more limited number of high priority pollutants. The Commission's efforts would, as usual, be more on a product by product basis. More specific comments are to be found in Sections II-V, as will a list of pertinent activities.

NRC

Indoor Pollutants



Committee on Indoor Pollutants
Board on Toxicology and Environmental Health Hazards
Assembly of Life Sciences

INDOOR POLLUTANTS

COMMITTEE ON INDOOR POLLUTANTS

BOARD ON TOXICOLOGY AND ENVIRONMENTAL HEALTH HAZARDS

ASSEMBLY OF LIFE SCIENCES

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This report has been reviewed by a group other than the authors according to procedures approved by a Report Review Committee consisting of members of the National Academy of Sciences, the National Academy of Engineering, and the Institute of Medicine.

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COMMITTEE ON INDOOR POLLUTANTS

JOHN D. SPENGLER, Harvard School of Public Health, Boston, Massachusetts, Chairman

MICHAEL D. LEBOWITZ, University of Arizona Medical Center, Tucson, Arizona, Cochairman

RONALD W. HART, National Center for Toxicological Research, Jefferson, Arkansas

CRAIG D. HOLLOWELL, University of California, Berkeley, California

MORTON LIPPMANN, New York University Medical Center, New York, New York

DEMETRIOS J. MOSCHANDREAS, GEOMET Technologies, Inc., Gaithersburg, Maryland

JAN A. J. STOLWIJK, Yale University School of Medicine, New Haven, Connecticut

DAVID L. SWIFT, The Johns Hopkins University, Baltimore, Maryland

JAMES E. WOODS, JR., Iowa State University, Ames, Iowa

JAMES A. FRAZIER, National Research Council, Washington, D.C., Staff Officer

NORMAN GROSSBLATT, National Research Council, Washington, D.C., Editor

LESLYE B. GIESE, National Research Council, Washington, D.C., Research Assistant

JEAN E. PERRIN, National Research Council, Washington, D.C., Secretary

CONTRIBUTORS TO THE REPORT ON INDOOR POLLUTANTS

JAMES BERK, University of California, Berkeley, California

WILLIAM F. BRANDOM, University of Denver, Denver, Colorado

DAVID M. BURNS, University Hospital, San Diego, California

BENJAMIN BURROWS, Arizona Health Sciences Center, Tucson, Arizona

WILLIAM CAIN, Yale University School of Medicine, New Haven, Connecticut

ROY R. CRAWFORD, Iowa State University, Ames, Iowa

CHARLES W. DENNIGER, Stanley Consultants, Inc., Muscatine, Iowa

DOUGLAS DOCKERY, Harvard School of Public Health, Boston, Massachusetts

NURTAN ESMEN, University of Pittsburgh, Pittsburgh, Pennsylvania

HUGH EVANS, New York University Medical Center, New York, New York

ARTHUR FRANK, Mount Sinai School of Medicine, New York, New York

RALPH F. GOLDMAN, Institute of Environmental Research, U.S. Army, Natick, Massachusetts

JACK D. HACKNEY, Rancho Los Amigos Hospital, Downey, California

CHARLES M. HUNT, National Bureau of Standards, Washington, D.C.

GEORGE JAKAB, The Johns Hopkins University, Baltimore, Maryland

JOHN E. JANSSEN, Honeywell, Inc., St. Paul, Minnesota

EDUARDO A. B. MALDONADO, Iowa State University, Ames, Iowa

PRESTON E. McNALL, National Bureau of Standards, Washington, D.C.

BRIAN MOKLER, Lovelace Biomedical and Environmental Research Institute, Albuquerque, New Mexico

GERALDINE M. MONTAG, Iowa State University, Ames, Iowa

ANTHONY NERO, University of California, Berkeley, California

ANTHONY NEWMAN-TAYLOR, Brompton Hospital, London, England

WAYNE OTT, Stanford University, Stanford, California

GARY L. REYNOLDS, Iowa State University, Ames, Iowa

RICHARD RILEY, Petersham, Massachusetts

GEORGE ROYAL, American Institute of Architects, Washington, D.C.

ROBERT N. SAWYER, Yale University Health Services, New Haven, Connecticut

FREDRICK H. SHAIR, California Institute of Technology, Pasadena, California

DONALD SIBBERT, GEOMET Technologies, Inc., Pomona, California

SAMUEL SILBERSTEIN, National Bureau of Standards, Washington, D.C.

WILLIAM R. SOLOMON, University of Michigan Hospital, Ann Arbor, Michigan

JAMES STEBBINGS, JR., Los Alamos Science Laboratory, Los Alamos, New Mexico

THEODOR D. STERLING, Simon Fraser University, Burnaby, British Columbia, Canada

JAMES E. TROSKO, Michigan State University, East Lansing, Michigan

McDONALD E. WRENN, University of Utah, Salt Lake City, Utah

JOHN YOCUM, TRC Corporation of New England, Wethersfield, Connecticut

BOARD ON TOXICOLOGY AND ENVIRONMENTAL HEALTH HAZARDS

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A. MYRICK FREEMAN, Bowdoin College, Brunswick, Maine

RICHARD HALL, McCormick & Company, Hunt Valley, Maryland

RONALD W. HART, National Center for Toxicological Research, Jefferson, Arkansas

MICHAEL LIEBERMAN, Washington University School of Medicine, St. Louis, Missouri

BRIAN MacMAHON, Harvard School of Public Health, Boston, Massachusetts

RICHARD MERRILL, University of Virginia Law School, Charlottesville, Virginia

ROBERT A. NEAL, Chemical Industry Institute of Toxicology, Research Triangle Park, North Carolina

IAN NISBET, Chemical Associates, Washington, D.C.

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GERALD WOGAN, Massachusetts Institute of Technology, Cambridge, Massachusetts

Ex Officio Members

EDWARD BRESNICK, University of Vermont, Burlington, Vermont

DAVID CLAYSON, Eppley Institute for Cancer Research, Omaha, Nebraska

JAMES F. CROW, University of Wisconsin, Madison, Wisconsin

JOHN DOULL, University of Kansas Medical Center, Kansas City, Kansas

ROGER O. McCLELLAN, Lovelace Biomedical and Environmental Research Institute, Albuquerque, New Mexico

ROBERT MENZER, University of Maryland, College Park, Maryland

ROBERT MILLER, National Cancer Institute, Bethesda, Maryland

SHELDON MURPHY, University of Texas, Houston, Texas

NORTON NELSON, New York University Medical Center, New York, New York

JOHN D. SPENGLER, Harvard School of Public Health, Boston, Massachusetts

JAMES L. WHITTENBERGER, Harvard School of Public Health, Boston, Massachusetts

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EXECUTIVE SUMMARY

Many people spend large amounts of each day indoors--in many cases, 80-90%--in a house, an automobile, a waiting room, an office or other workplace, or a confined space accessible to the general public, such as a store or a restaurant. It has been shown that indoor exposure to environmental pollutants can be substantial. Although there is little epidemiologic evidence on the health effects of indoor pollutants, indoor concentrations of some pollutants that already have primary ambient-air quality standards exceed those standards. Indoor exposure has been largely overlooked in research on the health effects of environmental pollutants, but it can constitute an important fraction of the total exposure to many pollutants.

Indoor pollution in residences, public building, and offices is created for the most part by the occupants' activities and their use of appliances, power equipment, and chemicals, by wear and tear and outgassing of some structural or decorative materials, by thermal factors, and by the intrusion of outdoor pollutants.

In some cases, the outdoor pollutants that penetrate to the indoors may represent the most important pollutant stress on human health and welfare, and such effects have been addressed at length in reports of previous National Research Council committees. This report is focused primarily on the indoor air contaminants that are liberated indoors. When they attain high concentrations, they may cause nuisances, irritation of sensitive tissues, illness, and death from acute as well as chronic exposures. Some pollutant sources--such as cigarette-smoking--have been recognized for a long time, but their importance has only recently been evaluated. Others arise from new products or from old products in new uses, such as building materials, pesticides, and insulation. A number of sources are of concern only in the indoor environment, e.g., cooking, use of consumer products, space-heating devices, and floor and wall coverings. The expanded use of wood and coal for residential space-heating, of home hobby and craft products, and of products that liberate organic substances is a potential contributor to the contamination of indoor environments. *Infectious microbes and allergenic agents can grow indoors or* be transmitted into indoor environments.

Ventilation alone (whether natural or forced) may not be sufficient to dilute indoor pollution to an acceptable point. Natural ventilation may be inappropriate because it is variable and generally not controllable in a precise way for many indoor settings. Exclusive dependence on forced ventilation is inappropriate because it is not universally available; moreover, introduction of untreated outdoor air may not always be desirable. Also, natural ventilation and forced ventilation may have substantial energy penalties, owing to heating and cooling losses. The adoption of energy-saving proposals to reduce ventilation rates could aggravate problems in indoor air quality, create new problems, and perhaps be generally detrimental to health and property, unless appropriate pollution-control measures are also taken.

Public-health laws are broad enough to permit evaluation and, when required, control of indoor environments. The public expects a safe and healthful outdoor environment, and the same expectation applies to public indoor environments. The regulatory authority to control contaminant sources, to set or recommend building codes, and to support or conduct research on or monitoring of indoor contaminants rests with diverse federal, state, and local government units, but no specific government unit has been directly charged with the responsibility for protecting the quality of indoor environments.

For all the reasons stated above, the present quality of the indoor environment and how this quality may change are matters of immediate and great concern.

SCOPE OF THE REPORT

This report was prepared, at the request of the Environmental Protection Agency (EPA), by the Committee on Indoor Pollutants, which was appointed by the National Research Council in the Board on Toxicology and Environmental Health Hazards, Assembly of Life Sciences. It is intended to characterize the quality of the indoor environment--primarily with respect to airborne pollutants, although others are discussed--and to determine the potential adverse health effects of indoor pollutants. The charge was to review, compile, and appraise the available knowledge. The Committee has also identified the research needed for abatement of indoor pollution. "Indoor" refers to the environments in homes, schools, public buildings, and similar spaces to which the public has access; industrial working environments, however, are excluded from consideration here.

It is beyond the scope of this report to list all the pollutants found indoors that are hazardous to human health. The examples given make it plain that humans are exposed to a variety of potentially hazardous indoor pollutants from diverse sources. It is hoped that this report will encourage researchers to broaden the list of hazardous indoor pollutants and to characterize the hazards, so that the general public and those responsible for pollution control and abatement can be informed.

Throughout this report, pollutants are mentioned without discussion of their health effects. This does not constitute an oversight on the part of the Committee, but rather reflects a decision that the discussion here be adequate to show that there are indoor pollutants that cause adverse health effects in humans. The reader's attention is directed to Chapter III, which offers some recommendations for further health research with respect to these pollutants, for further exposure studies, and for public education about effective ways of reducing exposure to many contaminants encountered indoors. The Committee on Indoor Pollutants and its contributors prepared concise reviews of such physical aspects as sources and concentrations and of such biologic aspects as the physiologic and toxicologic effects of a variety of contaminants encountered indoors. In addition, the effects of those contaminants that bear on human well-being in a more general way, such as soiling and corrosion, and the available means of controlling the presence of the contaminants are discussed in some detail. The report attempts to focus personal, corporate, and government attention on present and potential problems related to indoor contaminants.

The Committee notes that documentation of excessive indoor air pollution should not in itself be considered sufficient reason to relax standards for ambient air. The barriers between indoor air and outdoor air are not absolute, and ambient air contributes to indoor air. Furthermore, outdoor and indoor air pollutants may interact chemically and physiologically. The Committee recognizes the complexity of human exposures that have multiple sources. The development of effective and efficient strategies for mitigating hazardous contamination requires improved understanding of responses to exposure and of pollutant interactions.

The Committee has not attempted to set priorities for research on or regulation or control of indoor pollutants. Nor has it attempted to develop risk analyses for these pollutants. The order in which contaminants are presented in this report does not constitute a ranking of importance by the members of the Committee.

To set priorities for differentiating among indoor contaminants and to establish objectives for research and control programs, there must be a system for comparison. The dimensions of this system include the numbers of people exposed, the severity of exposures, and the consequences of the exposures. To be comprehensive, the system must also deal with ecologic and material damage, loss of productivity, degradation of artifacts, and other kinds of impact not related to health. Priorities could be derived from a ranking of these variables for pollutants of interest, but proper risk analysis would require measurement of exposures by population subgroups and weighting of exposure-response relationships by importance of outcome.

The Committee unanimously agreed that establishing firm priorities for research on indoor pollutants that ranks one contaminant as more important than another is premature. In most instances, we do not appreciate the extent of population exposures. Available reports on indoor air pollutants contain almost no data on the incidence of disease or even annoyance related to changes in pollutant

concentrations. Nevertheless, the Committee has made judgments based on its interpretation of reported concentrations, of prevalence of sources, and of evidence of effects on health and welfare. The Committee offers guidance for research, development, and educational programs by specifying current gaps in information and scientific and engineering deficiencies. Priorities within these programs will have to depend on the extent of federal and private funding; the Committee believes that the determination of such priorities is urgent.

ORGANIZATION OF THE REPORT

The introduction, summary and conclusions, and recommendations of the Committee are in Chapters I, II, and III, respectively. This material is based on both the body of the report and Committee deliberations. Some conclusions and recommendations were derived from consideration of the documented information in specific sections of the report and the substantiating discussions therein. Others, based on Committee consensus, were formulated from a more comprehensive perspective on the subject of indoor pollutants.

After the introduction, summary and conclusions, and recommendations in Chapters I, II, and III, the treatment of indoor pollutants is presented in three primary chapters and three secondary chapters. The three primary chapters are Chapter IV, on the sources and characterization of indoor pollution; Chapter VII, on the health effects; and Chapter IX, on the control of indoor pollutants. They are the most voluminous chapters and respond most directly to the Committee's charge to review and appraise the available knowledge on indoor air pollution. The other chapters--Chapter V, on factors that influence indoor pollution; Chapter VI, on the measurement of indoor pollution and exposures; and Chapter VIII, on other non-health-related effects--offer important additional information. They elaborate on material presented in the primary chapters, and they introduce factual and conceptual material that the Committee feels essential for evaluating indoor pollution comprehensively.

Chapter IV, on the sources and characterization of indoor pollution, covers radon and its decay products, formaldehyde and other organic substances, asbestos and fibrous glass, combustion products, tobacco smoke, consumer products, odors, temperature and humidity, and other pollutants not specifically treated. The objective of the chapter, as of Chapter VII (on health effects), is not a global treatment of every possible hazard encountered indoors, but rather a selective treatment. The chief criterion for selection is direct or circumstantial evidence that a contaminant causes or is reasonably likely to cause human stress, illness, or material damage indoors.

Chapter V, on factors that influence indoor pollution, expands on the physical characterization of indoor pollutions in Chapter IV. Aspects of geography, building design, and human activity that lead to variations in ventilation rates and in the distribution of pollutant sources are presented.

Chapter VI, on the measurement and monitoring of indoor pollution and exposure, reviews the design and components of indoor and personal monitors, mathematical models for estimating indoor pollutant concentrations, and methods of estimating total personal exposure.

Chapter VII, on the health effects of indoor pollution, relates the current understanding of the toxicologic and physiologic effects of specific contaminants that are found at high enough concentrations and in a broad enough range of indoor environments to constitute an actual or reasonably likely challenge to the occupants of those environments. In some cases, as in Chapter IV, the discussion is structured by source, such as involuntary smoking or indoor combustion products; the health effects may be attributable to specific components of a mixture of gases and particles, or it may be attributable to the general, mixed exposure. The chapter also considers indoor airborne contagion and allergens.

Chapter VIII, on the effects of indoor pollution on human welfare, covers a number of items related to comfort, productivity, and material protection in indoor environments.

Chapter IX, on the control of indoor pollution, emphasizes the engineering aspects of air-conditioning and indoor air-cleaning. Ventilation codes and standards are reviewed, and mechanical systems for conditioning and cleaning air are described. The chapter discusses strategies for controlling contaminants to maintain acceptable indoor air quality in general. Some pollutants, because of their sources or their physical and chemical properties, cannot be treated with conventional control systems, and strategies for controlling these pollutants are described specifically.

Appendix A lists national primary ambient-air quality standards and occupational-health standards (for the industrial environment) established for the United States. In addition, it lists indoor-air pollution standards and guidelines of several foreign countries. Ventilation standards for dwellings are also listed. This appendix is not exhaustive with respect to relevant pollution or ventilation standards, but it does offer a point of reference for some of the more commonly used standards.

Appendix B presents an example of the interactions among energy conservation, comfort, and indoor air pollution in a residence. This simulation exercise illustrates the tradeoffs among energy-cost savings, retrofit costs, and thermal comfort under the constraints of maintaining various hypothetical conditions of indoor air quality in a particular kind of single-family residence.

PRINCIPAL FINDINGS ON SPECIFIC POLLUTANTS AND CLASSES OF POLLUTANTS

RADON

Radon and its alpha-emitting decay products contribute a major portion of the biologically significant dose associated with natural background radiation. Many natural substances contain radium, a

precursor of radon gas. Soil, construction materials, and groundwater are the major sources of indoor radon. Indoor radon concentrations are often an order of magnitude greater than outdoor concentrations. The dose-response relationships for alpha-emitting radionuclides are not sufficiently well accepted to allow quantitation of the health risk associated with measured indoor concentrations, and surveys of radiation in homes are very limited. Expansion of the data base on the variation of indoor radon exposure with time and location is a necessary prerequisite for assignment of a health risk to indoor radon. In theory, techniques for controlling radon exposure are available; but they need development and evaluation before they can be applied economically on a large scale. The effectiveness of several contaminant control strategies, other than ventilation, has not been demonstrated in practice. Codes for new construction may be necessary to prevent the occurrence of high radon concentrations in modern or refurbished dwellings. In any event, prudent judgment concerning reduced ventilation in residences must be based on a better understanding of radiation exposures in present houses.

FORMALDEHYDE

The major indoor sources of formaldehyde have been identified. Aldehydes and other organic substances emanate from outgassing of urea-formaldehyde foam insulation, particleboard, plywood, fabrics, and, to a lesser extent, cigarettes and indoor combustion sources. The high surface-to-volume ratio of particleboard and plywood used as building materials in mobile homes, as well as lower air-exchange rates, causes the high measured formaldehyde concentrations. Formaldehyde can cause skin, eye, and throat irritation in occupants. Moreover, potential health problems associated with formaldehyde exposures are readily identified in acute cases when concentrations are high and tolerance is low. In addition to irritation, respiratory disorders and allergies have been associated with high formaldehyde concentrations. There is evidence of a decreased threshold of sensitivity with prolonged exposure. Recent studies have indicated that exposure of rats and mice to formaldehyde produces nasal cancer. Owing to the ubiquitous and increasing use of resins and solvents in building materials and furnishings, indoor formaldehyde concentrations have increased. Abatement technology is available, although at times expensive; new and less expensive techniques are being developed. The Committee is especially concerned with long-term and essentially continuous indoor exposures to low concentrations of formaldehyde.

ASBESTOS AND OTHER FIBERS

The health hazard posed by indoor exposure to asbetos has been perceived as a problem by virtue of the presence of asbestos fibers in insulating and decorative materials. Abrasion, mechanical vibration,

or deliberate disruption of asbestos-containing surfaces can result in increased fiber concentrations in the indoor environment. There have been a small number of studies in which fiber counts have been documented in association with normal building use. Extrapolation from what we currently understand about the exposure-response relationships for asbestos fibers to the very low concentrations reported in indoor spaces, such as schools, suggests a small health risk under conditions of normal use. However, deliberate modification of surfaces to remove asbestos from buildings may create a risk of exposure of occupants and workers. Buildings in which asbestos exposure is likely to occur can be identified. The risk of exposure from dislodged fibers can be reduced by containment. The occurrence of mesothelioma (a specific form of cancer believed to result only from the inhalation of asbestos fibers) may provide a very sensitive indicator of the exposure of the general population. Home exposure to asbestos due to aging, cracking, or physical disruption of insulated pipes or asbestos-containing ceiling tiles and spackling compounds may be greater than public exposures in schools, which have received the most attention. Homes built before 1950 in northern climates are more likely to have pipes insulated with asbestos plaster. Given the very common use of asbestos in homes, schools,and other buildings, there is a need for further assessment to identify structures where actual asbestos exposure constitutes substantial risk to humans.

The extent of exposure of the general public to asbestos fibers has not been assessed; however, the occurrence of mesothelioma should be carefully monitored in the general population. Man-made fibers have produced skin irritation, but have not otherwise been demonstrated convincingly as hazardous to health. Epidemiologic and toxicologic investigation of synthetic fibers should continue. On the basis of present knowledge, synthetic fibers in the indoor environment should not cause undue concern.

TOBACCO SMOKE

Virtually every member of our society is exposed to tobacco smoke: 33% of the population smokes, and the rest are exposed to the smoke released by others. The constituents of tobacco smoke are well documented as hazardous, the prevalence of population exposures is very high, and there is an increased incidence of respiratory tract symptoms and functional decrements in children residing in homes with smokers, compared with those in homes without smokers. These considerations and recent evidence of increased lung-cancer rates among nonsmoking women living with smoking husbands have led us to conclude that indoor exposure to tobacco smoke has adverse effects. Coughing, headache, nausea, and irritation of eyes, nose, and throat are among the reported symptoms. Although many studies have measured various components of tobacco smoke indoors, total exposure has not been determined. Passive exposure to tobacco smoke may constitute an important exposure to respirable particles, such gaseous compounds as

acrolein and formaldehyde, benzo[a]pyrene, and various trace metals. Reduced ventilation increases concentrations of tobacco smoke. As an energy-conserving compromise, smoking could be restricted to zones that are well ventilated. Public policy should clearly articulate that involuntary exposure to tobacco smoke has adverse health effects and ought to be minimized or avoided where possible. Under this framework, the prohibition or restriction of smoking in public buildings, offices, etc., is a control option to be considered with ventilation and air-cleaning.

INDOOR COMBUSTION

When fuel combustion occurs indoors--e.g., for heating, cooking, and power machinery, including automobiles--it gives rise to increased concentrations of gases and particles. Unvented gas cooking is probably responsible for a large portion of nitrogen dioxide exposures in our population. In many homes, chronic exposures to nitrogen dioxide indoors may exceed established national ambient-air quality standards. Shorter-term 1-h average concentrations indoors often exceed the highest hourly concentrations measured outdoors. The concentrations of nitrogen dioxide and carbon monoxide in residences have not been fully documented. However, some studies have shown an association between gas cooking and the impairment of lung function in children. Gas cooking appliances are also sources of carbon monoxide, carbon dioxide, formaldehyde, hydrogen cyanide, sulfate particles, organic particulate matter, and organic vapors. The problem of chronic or even peak exposures to combustion products indoors will be accentuated with decreased ventilation and the increased use of portable space-heaters, wood- and coal-burning stoves, and indoor venting of gas dryers. Carbon monoxide, nitrogen oxides, and particles from automobile exhaust can produce increased concentrations in office buildings and public areas. Concentrations exceeding 1-h carbon monoxide national ambient-air quality standards (NAAQS) by a factor of 2-4 have been reported in several ice-skating rinks that use gasoline-powered ice resurfacing machinery. Office buildings and apartment buildings with attached or underground garages can also have sustained high concentrations of carbon monoxide indoors. Because both carbon monoxide and nitrogen dioxide are odorless at typical concentrations, the presence of increased and possibly hazardous concentrations may go undetected.

Although confirmation is necessary, the available evidence suggests that important population exposures to nitrogen dioxide and carbon monoxide can occur indoors and may constitute a sufficient threat to the general public health to justify remedial action. Reducing exposure to those gases is relatively straightforward. Source removal or direct venting of combustion sources should be considered.

Efforts to conserve energy present other potential problems indoors. Effective energy-conservation measures can result in an overcapacity of existing heating equipment. Operation of such

equipment at low load factors may decrease its overall combustion efficiency and increase emission of the products of combustion.

MICROORGANISMS AND ALLERGENS

Microorganisms are present in the indoor environment and are associated with human activity and the presence of domestic animals. The microorganisms include bacteria, viruses, and fungi. Many microorganisms--such as spores, molds, and fungi--multiply in the presence of increased humidity. It is possible that reduced ventilation and the increased use of untreated recirculating air could increase the concentrations of microorganisms. Many of these microorganisms can produce infection, disease, or allergic reactions. Respiratory viruses and bacteria can be transmitted from person to person in buildings and confined spaces. Certainly, respiratory infections are an important cause of morbidity that results in lost earnings and discomfort. It is reasonable to assume that some of the incidence of respiratory disease results from airborne transmission, but it is not at all clear what effect ventilation, air-conditioning, or air-cleaning will have on incidence. If the main transmission is between persons in contact with or close to each other, the mechanism and efficiency of disease transfer will be relatively insensitive to ventilation rates and other operating conditions of the air handling systems. However, to the extent that infectious and allergenic microorganisms remain viable and airborne, substantial reduction in ventilation rates will tend to increase concentrations and most likely the probability of infection and allergy.

MOISTURE

Water vapor in confined spaces is a product of metabolic and respiratory processes, as well as of indoor combustion and evaporation from clothes and dish-washing and bathroom functions. Condensation of water indoors has been shown to increase corrosive effects of absorbed gases. Decreases in ventilation tend to increase the indoor relative humidity during the heating seasons. Excess water vapor adsorbs or condenses on drier or colder surfaces, and that gives rise to increased deterioration or corrosion of building materials, furnishings, decorations, artwork, and other artifacts. Increased relative humidity may also promote the growth of molds, algae, and fungi. Thus, humidity control may become an important component of reduced-ventilation strategies. Some energy penalty may result that should be considered in relation to the energy savings that may be obtained through reduced ventilation.

RESPONSIBILITIES

The quality of the indoor environment is not the responsibility exclusively of any individual or government body. Even a single home

built in isolation (away from outdoor sources of contaminants) has the potential to be contaminated by its occupants, soil, outgassing from piped-water use, building materials, cooking, space-heating, consumer products, pesticides, molds, and fungi. Responsibility for the indoor environment is on four levels: individuals; product manufacturers; building designers, contractors, and owners; and government.

Most sources of contamination are associated with human activity. Individuals also exercise some control over contamination sources and ventilation. Hence, the individual can directly affect his or her own exposure, as well as the exposure of those with whom the indoor space is shared. This is also true, to a lesser extent, in the nonindustrial workplace and in public buildings.

Building owners and operators are responsible for maintaining the indoor environment and ensuring that at least minimal ventilation standards are being met. It should be noted that there is little or no enforcement of ventilation standards, once building plans are approved. Architects, engineers, and contractors should treat indoor environmental quality as a design objective. There are many opportunities in building design to separate people from the sources of contamination or to remove the sources entirely.

Manufacturers have the responsibility to warn the consumer adequately of the potential hazards of products. As evidence on specific contaminants--such as formaldehyde and nitrogen dioxide from gas stoves--becomes available, less harmful substitutes can be considered, and results of research on the control of the sources can be made available.

Government shares the responsibility to ensure that the indoor environments to which the public has access are healthful. Clearly, in the assessment of indoor concentrations, in instrument development, and in the control of health-directed research on indoor pollutants, government can serve the public interest. Government can establish ordinances (regulations) to protect the general public from nuisances, contamination, or direct health damage; such ordinances may include performance standards related to building materials and ventilation codes for public and private buildings. In the same vein, government can require product certification in the case of known or potential hazards. Furthermore, government can establish concentration standards or source-control specifications. Radon, a naturally occurring substance, is a clear example of contaminants that would require government attention, as opposed to that of industry or the individual.

CONCLUSIONS

Definitive conclusions on the character of indoor air are prevented by the lack of systematic studies. The available data base has been generated by a series of pilot studies and does not fully characterize the variety of pollutants, indoor environments, and occupancy conditions. Furthermore, the implementation of energy-conservation measures and the introduction of new building materials have intensified the problem of indoor air contamination.

Studies explicitly addressing both long-term and episodic events have not been undertaken. Episodic release of contaminants in the indoor environment may be rare, but can lead to short-term high contaminant concentrations, which must be considered (in addition to long-term low concentrations) in assessing the overall health risk of indoor contaminants.

Measurement of indoor contaminants necessitates a sampling protocol that considers the spatial and temporal profile of several pollutants, as well as air diffusion and ventilation characteristics. In addition, measurement techniques for assessing indoor concentrations have to meet more rigorous requirements, particularly with regard to sensitivity and interferences. Unfortunately, many of the instruments required to characterize long-term and short-term indoor pollutant concentrations do not exist.

From a practical viewpoint, it would be desirable to determine the emission rate of an indoor pollutant by simple physical measurements and to infer the dose received by a human inhabiting the indoor space. But several intervening steps must be evaluated that involve degrees of uncertainty ranging from good estimates to total ignorance. The first process to be considered is the transport by diffusion and convection; transport is influenced initially by the fluid motion of the air near the source and throughout the indoor space. These modes of transport depend on a number of factors and are usually spatially and temporally variable. They lead to a concentration profile of the contaminant as a function of position and time. Measuring such profiles is virtually impossible, so the usual approach is to use mathematical models of dispersion. The human receptor is not stationary. Therefore, to obtain an exposure history, the spatial history of the receptor should be specified or estimated. Inexact knowledge of this function introduces a further degree of uncertainty. Dose to the receptor is related to exposure through deposition functions that express the fraction of the exposure that is available to reach specific receptor sites and produce effects. These deposition functions are themselves functions of several variables that are usually poorly specified or unknown. Thus, several layers of uncertainty are embedded between emission rate and receptor dose.

Indoor air pollutants generated or released indoors typically occur in concentrations and mixtures that are often episodic and generally vary over a wide range of time and from one space to another. As a result, human exposures are difficult to assess for individuals or groups. If, in addition, the adverse health effects are subtle, and especially if they are delayed, associations between indoor air pollutants and disease or premature mortality are unlikely to be discovered or demonstrated without a specific and substantial effort. Thus, efforts to improve indoor air quality most likely will have to be guided by information on the adverse health effects of pollutants demonstrated and studied in other settings, such as the occupational environment.

For a limited number of air contaminants that can be found in residential and public buildings, there is direct and circumstantial evidence that human exposures are large enough and common enough to

account for substantial morbidity and premature mortality. These include radon progeny, sidestream tobacco smoke, formaldehyde, carbon monoxide, nitrogen dioxide, aeropathogens, and aeroallergens. However, our knowledge of the extent of the exposures and of the exposure-response relationships is inadequate to permit measurement or even estimation of the resulting mortality and morbidity. Furthermore, the kinds of health effects and the latent periods between exposures and effects are quite varied. For example, they extend from acute intoxication from carbon monoxide and formaldehyde to cancer that appears long after exposure to radon decay products and asbestos.

In evaluating health risks, it is reasonable to compare indoor exposure with ambient air-quality standards for pollutants for which there are such standards (carbon monoxide, sulfur dioxide, nitrogen dioxide, ozone, lead, and total suspended particles). Depending on how the standards were developed, however, they may or may not consider exposure of the most sensitive population groups (the ill or infirm or the very young). In addition, the composition of particulate matter indoors, the potential synergistic interaction of gaseous and particulate matter, and the time characteristics of exposures can differ widely between the indoors and the ambient environment. Hence, for some pollutants, the ambient air-quality standards may actually underestimate the health risks.

Current knowledge would permit the establishment of defensible indoor-air quality standards for only a few, if any, contaminants. In any case, the establishment of such standards would not necessarily lead to rational or enforceable controls beyond ventilation codes for dilution. Economical and reliable techniques for sampling and analyzing the airborne contaminants of interest at very low concentrations have not been developed; nor have methods been developed to relate spot samples from specific locations to integrated doses.

We conclude that the best approach to the reduction of health damage from exposure to indoor contaminants is to reduce the population exposure to those contaminants. Control strategies for some pollutants would target the high-exposure groups. For other pollutants, lowering the population-weighted mean exposure would, by best estimates, reduce the health hazards.

Because of the diversity of indoor pollutants and their sources and because of the unpredictability of the distribution of such pollutants over time and in different buildings, efforts to improve indoor air quality should concentrate on reducing the number and strength of the sources by substitution of other materials.

Control strategies based on the specifications of source control are preferred, whenever feasible, because they are generally the most dependable, with respect to their extent and reliability of exposure reduction. That suggests that reducing exposure through source control or removal or through material substitution must be tempered by the practical realities of existing sources, which might not be easy to eliminate, and by the unknown long-term toxicity of substitute materials. When those strategies cannot achieve the desired degree of

control at reasonable cost, they can be supplemented or replaced by other engineering controls, such as dilution ventilation or air-cleaning. However, it should be recognized that the concentration reduction achieved through dilution may be less than that achievable with source control or air-cleaning, whereas the effectiveness of air-cleaning devices usually depends on frequent and effective application of maintenance procedures.

The specific source controls appropriate to each kind of contaminant can be expected to vary. For example, local exhaust control is most appropriate for nitrogen dioxide and carbon monoxide from gas ranges, the sealing of walls and floors for radon control, prohibition for asbestos-containing products, and specifications for care of furniture, drapery, and carpeting materials prepared with formaldehyde-containing substances.

Air-conditioning systems are generally designed to provide for all or most of the thermal environmental requirements (i.e., heating, cooling, and ventilating) of the occupied space. In the design of these systems, it is necessary to select components that will meet the particular requirements, such as heating coils or furnaces to meet winter design temperatures, evaporator coils and condensing units to meet summer design temperatures and humidities, and air-cleaners or ventilation air-flow rates to meet the air-quality requirements of the occupied space. The functional requirements of the space (i.e., residential, office, theater, etc.) also impose constraints on the type of system that might be selected. Because of the wide variety of functional requirements of indoor environments and the other constraints on design, a vast variety of control systems are used. For instance, lighting and acoustic requirements can influence the size and location of the air-conditioning system, the location of air supply and return devices, and air velocities in the air distribution system. Available information suggests that this trend will continue.

Although the requirements may be described discretely and some performance specifications are available for components of the system, the effectiveness of the system as a whole, including its impact on indoor air quality, must be evaluated. Unfortunately, very few data are available to indicate whether these systems, under actual loads, perform in accordance with their designs.

RECOMMENDATIONS

1. A staged assessment of the exposures of the general population to indoor pollutants and of the effects of such exposures on health and welfare should be conducted by the federal government in both residential and office buildings. Federal agencies with substantial interests in definition of the indoor exposures--i.e., the Environmental Protection Agency, the Department of Energy, the Consumer Product Safety Commission, the National Institute for Occupational Safety and Health, the Centers for Disease Control, the National Institute of Environmental Health Sciences, the Food and Drug Administration, the National Center for Toxicological Research, the

Department of Housing and Urban Development, etc.--should participate in the formulation of the study objectives and protocols and should provide technical assistance and financial support, as appropriate and feasible. Congress should enunciate clearly its interest in healthful and safe indoor environments.

2. Monitoring protocols and properly designed monitoring instruments must be developed to satisfy the special requirements involved in investigating the indoor environment.

3. The indoor pollutants that should receive the initial major focus in investigation of sources, concentrations, dispersion, and removal are radon and its progeny, tobacco smoke, formaldehyde, nitrogen dioxide, carbon monoxide, pesticides, water vapor, carbon dioxide, and airborne contagion, including allergens.

4. The health effects that should receive the initial major focus in investigation are respiratory-infection rates and respiratory mechanical function in relation to nitrogen dioxide, tobacco-smoke, and airborne-contagion exposures; respiratory-tract irritation and potential carcinogenic effects in relation to formaldehyde, tobacco smoke, asbestos, and alpha-emitting radon decay products; and acute intoxication, blood carboxyhemoglobin, and cardiovascular-disease aggravation in relation to carbon monoxide and nitrogen dioxide exposures.

5. The low-level acute and chronic complaints of malaise, headache, stuffiness, and eye and throat irritation that are reported with increasing frequency in large buildings deserve careful study.

6. The welfare and behavioral effects that should receive the initial major focus in investigation are material damage from mold formation in relation to relative humidity; corrosion and surface deterioration in relation to nitrogen dioxide, sulfur dioxide, and water vapor; fabric fading and deterioration in relation to solar radiation in combination with nitrogen dioxide, sulfur dioxide, house dust, and water vapor; soiling due to tobacco smoke; and the lowering of work productivity due to indoor air pollution and associated discomfort.

7. Responsibility for conducting a well-coordinated investigation of the influence of building design and operational factors on the concentrations of pollutants in both residential and commercial facilities should rest with the federal government, assisted by the appropriate professional and scientific organizations.

8. The building factors that should receive the initial major focus in investigation are as follows:

a. The effects of reducing infiltration rates in existing buildings on combustion efficiency of space-heating equipment and on increases in relative humidity and concentrations of indoor-generated air pollutants and airborne contagion.

b. The effects of materials of construction and furnishings on indoor-pollutant content--specifically, there should be systematic evaluations of outgassing and surface attrition of particleboard and plywood (for formaldehyde and other organic substances); of wall and floor coverings and fabrics (for organic substances); of masonry products (for radon and dust); of wallboard, plaster, and spackling compounds (for dust and fibers); and of the materials used for heat storage in dry solar systems (for radon, dust, surface molds, etc.).

c. The differences in air distribution, diffusion, mixing, etc., associated with the use of different climate-control systems, such as forced-air, baseboard, and radiant floor or ceiling systems.

d. The effectiveness of air-cleaning systems in capturing pollutants in recirculating air--specifically, this will require in-place testing of systems, rather than test-stand evaluation of components, and the effectiveness of a variety of commonly used systems should be evaluated for radon and radon progeny, formaldehyde and solvent vapors, and cigarette smoke.

9. The potential for consumer products to contaminate the indoor environment needs to be evaluated. Hazardous components of these products must be identified and tested. Adequate labeling, warning users of hazards associated with product use and misuse in enclosed spaces, should be required. Testing in homes is needed to assess the extent of contamination, allergic reactions, and other health effects of pesticides, residues, and consumer products.

I

INTRODUCTION

This report was prepared, at the request of the Environmental Protection Agency (EPA), by the Committee on Indoor Pollutants, which was appointed by the National Research Council in the Board on Toxicology and Environmental Health Hazards, Assembly of Life Sciences. It is intended to characterize the quality of the indoor environment, primarily with respect to airborne pollutants, and to determine the potential adverse health effects of indoor pollutants. The charge was to review, compile, and appraise the available knowledge. The Committee has also identified the research needed for abatement of indoor pollution. "Indoor" refers to the environments inside homes, schools, public buildings, and similar spaces to which the public has access; industrial working environments, however, are excluded from consideration here.

It is beyond the scope of this report to list all the pollutants found indoors that are hazardous to human health. The examples given make it plain that humans are exposed to a variety of potentially hazardous indoor pollutants from diverse sources. It is hoped that this report will encourage researchers to broaden the list of hazardous indoor pollutants and to characterize the hazards, so that the general public and those responsible for pollution control and abatement can be informed.

Throughout this report, pollutants are mentioned without discussion of their health effects. This does not constitute an oversight on the part of the Committee, but rather reflects a decision that the discussion here be adequate to show that there are indoor pollutants that cause adverse health effects in humans. The reader's attention is directed to Chapter III, which offers some recommendations for further health research with respect to these pollutants, for further exposure studies, and for public education about effective ways of reducing exposure to many contaminants encountered indoors.

Attention has recently been drawn to the problems of specific pollutants that originate indoors, e.g., formaldehyde released from urea-formaldehyde foam insulation and from urea-formaldehyde resins used to bind laminated-wood products, asbestos in building materials,

and radon and its progeny. Efforts to reduce infiltration of outdoor air for energy conservation have heightened the interest in indoor pollution. It is hoped that this report will be useful to the Administrator of EPA and other agencies and individuals in considering indoor environments as a source of exposure of members of the general public to hazardous pollutants. Some of the same pollutants, of course, are now regulated as pollutants in the outdoor atmosphere and in the workplace (see the lists of air-quality standards in Appendix A).

The Committee's report outlines the scope of the problems regarding indoor pollutants and discusses their sources, their effects on human health and welfare (human comfort, productivity, soiling, and corrosion), the technologies available for their control or abatement, and concerns about the effects of energy-conservation strategies on the indoor concentrations of pollutants. It approaches the subject of indoor pollution from three viewpoints:

- Physical factors, such as indoor-pollutant sources and concentrations and population exposures to those pollutants.
- Biomedical evidence on the effects of several pollutants found in the indoor environment.
- Engineering, air-handling and -cleaning systems, and other control options for reducing indoor exposures to pollutants.

The report reviews current understanding of these subjects, assesses the quantity and quality of available information, and offers recommendations for additional studies where appropriate.

Because of the multidisciplinary and complex nature of the indoor pollution question, this document could not possibly treat all pertinent subjects. One important exclusion from the assessment is the indoor industrial environment. It is recognized that many of the pollutants found in areas to which the public has access are also common to industrial settings, often in higher concentrations. The Committee chose to consider only indoor environments to which the general population has access; these include residences, public facilities, recreational facilities, vehicles and transportation-related buildings, educational facilities, and many work settings. Examples of workplaces to which the public has access and in which the public may be compromised by indoor pollution include service stations, automobile showrooms with attached maintenance areas, banks, offices, and buildings with multiple uses.

This document reviews the information on the health and welfare effects of selected indoor pollutants, with emphasis on air pollutants. It includes a critical appraisal of reported measurement and exposure studies, but it does not attempt a quantitative assessment of exposure to the hazardous pollutants in the indoor environment, because in most cases current methods are inadequate for that.

There is no discussion here of the legal, social, or economic implications of regulating the indoor environment in public buildings or homes. Sociopolitical controls of pollution are quite complex and beyond the scope of this document.

Throughout the Committee's deliberations, and reflected in its conclusions and recommendations, were the following questions:

- Do indoor pollution exposures adversely affect the health, welfare, productivity, or sense of well-being of the population or any portion of the population?
- Does the indoor environment constitute an important component of exposure to pollutants?
- Are some groups or individuals at risk by virtue of high indoor concentrations of air pollutants or by virtue of susceptibility?
- What is known about the relative magnitudes of indoor and outdoor pollutant concentrations? Are the sources, ventilation rates, and reaction and removal factors that influence the indoor-outdoor relationships sufficiently well known to predict indoor concentrations and prescribe controls?
- What control strategies are effective for reducing population exposures to specific indoor pollutants?
- Will future changes in housing materials, products, ventilation codes, and activity patterns adversely affect health and welfare through changes in indoor exposures to air pollution?

It is very important that health and welfare problems related to indoor pollution be clearly differentiated from perceived problems or pseudoproblems. This requires measurements that are both accurate and sufficiently representative to identify or estimate the population at risk. And it requires that health research provide reasonable assurance that current or projected exposures can cause unacceptable effects in a portion of the population. Only when these two components are present can prudent judgments on recommended concentrations and control strategies be made.

Efforts to improve the public health and protect the public from hazardous airborne pollutants have been directed primarily toward improving the ambient and industrial environments. Improvements in outdoor (ambient) air have been achieved fundamentally through source control or removal; dilution by tall stacks and source relocation are not considered control strategies. In the indoor industrial environment, however, ventilation or dilution with outdoor air has usually proved to be the most cost-effective way of reducing worker exposure.

The indoor concentrations of airborne contaminants depend on five factors: the generation rate (for indoor-generated pollutants) or the ambient concentration (for outdoor-generated pollutants), the volume of the indoor environment, the air-exchange rate, the mixing efficiency of the indoor space, and the decay (removal) rates of the pollutants.

Until recently, the air in most buildings has been controlled for comfort and odor considerations, not for contaminants. Depending on heating, cooling, and humidity requirements for the indoor environment, the natural or forced infiltration of outdoor air to displace "conditioned" indoor air may entail a considerable energy penalty. Diluting the indoor air with outdoor air reduces

concentrations of airborne pollutants generated by indoor sources, such as building materials, appliances, and tobacco-smoking. But outdoor air can also introduce pollutants of outdoor origin, and these pollutants may react with surfaces or other indoor contaminants. Conversely, reducing air exchange without compensating with air-cleaning will result in increased concentrations of contaminants that are generated indoors.

The current growing interest in the quality of the indoor environment is in part a result of efforts to reduce ventilation for energy conservation. In the United States, an estimated 20-50% of energy consumed is for space-heating and -cooling. In many buildings, the energy used to move and condition ventilating air can be as much as 90% of the total energy demand. Buildings lose energy by conduction and radiation through windows, walls, and ceilings and by exchange of indoor conditioned air with unconditioned outdoor air. Reducing ventilation in residential and commercial buildings can be a cost-effective way to achieve energy conservation. However, it is reasonable to expect concentrations of contaminants generated by building occupants, equipment, appliances, and materials to increase when ventilation is reduced. Predicting the results of ventilation changes is limited in some cases by lack of knowledge of the nature and behavior of contaminant sources, of the existing concentrations of pollutants, and of their chemical and physical reactions and removal rates. Even for current situations, the significance of the potential indoor-pollution problem is undefined for many pollutants, because the populations at possible risk have not been identified and the physiologic, behavioral, or welfare effects of various degrees of exposure have not been determined.

Although there is considerable mass-media coverage of the quality of indoor environments, the concern for indoor pollution in the nonindustrial setting is not new. Some countries have tried to regulate pollutants in nonindustrial environments (see Appendix A). Asbestos-fiber contamination in homes and schools has been monitored and in some cases contained or removed. Ozone generation by office copying machines has been regulated. Minimal acceptable ventilation rates for smoke and odor control are incorporated into municipal and state building codes. Several countries have set standards for residential or public facilities to limit exposures to formaldehyde, carbon dioxide, and radon. There have been surveys, but not systematic evaluation, of indoor pollutant concentrations in a variety of locations. The indoor environment has become an issue for the public, government, scientific groups, and corporations because of three phenomena: energy-conservation efforts, which may exacerbate an indoor-pollution problem; the realization that little is known on the hazards of many compounds that are commonly found indoors and outdoors, including consumer products and fuels in common use (note, for example, the resurgence in residential wood- and coal-burning); and the evaluation of pollutant hazards by federal agencies, which have begun to recognize the need to understand the total exposure.

For a large proportion of the population, normal activity occurs disproportionately indoors. In a consideration of integrated

pollutant exposures, indoor concentrations are relevant. Between 80% and 90% of an average person's day or year is spent in enclosed areas. On the average, people spend approximately 16 hours/day in their homes. And a rather consistent 1-2 hour/day is spent in transit. Thus, for at least some pollutants, the indoor concentrations are the most important, with respect to potential health effects or material damage.

The time-integrated exposure is perhaps important in determining chronic effects, such as corrosion. But the short-term peak or transient pollutant exposures may be more important, causing or contributing to both acute and chronic effects. Using the average amount of time a person is outdoors or indoors or the time-averaged concentrations may be misleading, if the concern is for peak-exposure effects. Peak exposures may occur indoors or outdoors. They may be encountered only during specific activities or in locations occupied only infrequently. In fact, short-term peak concentrations may contribute only a small proportion of a person's total time-integrated exposure. Both time-integrated concentrations and short-term, transient high concentrations must be considered, whether they occur indoors or outdoors.

Although the indoor and outdoor environments have not been sufficiently assessed to characterize all pollutant constituents comprehensively, it is useful to categorize indoor pollutants into three groups. Table I-1 groups pollutants by source. Those in the first group are principally of outdoor origin; thus, their concentrations are generally higher outdoors. This group includes sulfur dioxide; ozone; many elemental, inorganic, and organic species of particles; pollen; and some organic vapors. They are encountered indoors primarily because they are carried in with infiltrating air. Some may be carried indoors on surfaces. Once inside, particles can be resuspended, or organic substances may volatilize because temperatures and partial pressures are different. The higher indoor surface-to-volume ratios increase the removal rates of many of these pollutants.

Pollutants in the second group have both indoor and outdoor sources. Generally considered as belonging to this class are pollutants produced during combustion, such as carbon dioxide, carbon monoxide, nitrogen oxides, and some components of suspended particulate matter (primarily fine particles--diameter less than 3.0 μm). Because of the limited indoor mixing volume and longer residence times, concentrations of these and other combustion products often exceed outdoor concentrations. This group also includes organic vapors from solvents that can be used outdoors, as well as indoors. Biologic materials, such as fungal spores, have both indoor and outdoor sources. Fibers, including asbestos fibers, have indoor and outdoor sources. Serpentine rock, brake linings, and industrial facilities contribute asbestos fibers to the outdoor air. Insulation, fireproofing, and decorative materials used indoors may contain asbestos. Similarly, water vapor, sound, and nonionizing radiation can be considered to belong to this group.

TABLE I-1

Typical Sources of Some Pollutants Grouped by Origin

Pollutants	Sources
Group I--Sources predominantly outdoor:	
Sulfur oxides (gases, particles)	Fuel combustion, smelters
Ozone	Photochemical reactions
Pollens	Trees, grass, weeds, plants
Lead, manganese	Automobiles
Calcium, chlorine, silicon, cadmium	Suspension of soils or industrial emission
Organic substances	Petrochemical solvents, natural sources, vaporization of unburned fuels
Group II--Sources both indoor and outdoor:	
Nitric oxide, nitrogen dioxide	Fuel-burning
Carbon monoxide	Fuel-burning
Carbon dioxide	Metabolic activity, combustion
Particles	Resuspension, condensation of vapors and combustion products
Water vapor	Biologic activity, combustion, evaporation
Organic substances	Volatilization, combustion, paint, metabolic action, pesticides, insecticides, fungicides
Spores	Fungi, molds
Group III--Sources predominantly indoor:	
Radon	Building construction materials (concrete, stone), water
Formaldehyde	Particleboard, insulation, furnishings, tobacco smoke
Asbestos, mineral, and synthetic fibers	Fire-retardant, acoustic, thermal, or electric insulation
Organic substances	Adhesives, solvents, cooking, cosmetics, solvents
Ammonia	Metabolic activity, cleaning products
Polycyclic hydrocarbons, arsenic, nicotine, acrolein, etc.	Tobacco smoke
Mercury	Fungicides, in paints, spills in dental-care facilities or laboratories, thermometer breakage
Aerosols	Consumer products
Viable organisms	Infections
Allergens	House dust, animal dander

The third group of pollutants contains those whose sources are predominantly indoor. To this third group we may add pollutants whose concentrations are high enough only indoors to warrant concern for their effects. These pollutants are either generated by the occupants or associated with building materials, appliances, machines, consumer products, or art and craft materials. They include radon, formaldehyde, other organic substances from a variety of materials, asbestos and other fibers, odors, molds, and the numerous compounds identified in tobacco smoke.

Greater attention recently has been drawn to the third group of indoor pollutants. There have been reports of complaints about formaldehyde indoors after application of urea-formaldehyde foam insulation and particleboard and the installation of furnishings. Higher formaldehyde concentrations in European homes were reported in the early seventies. Radon and its progeny have been found in high concentrations in homes built on land reclaimed from phosphate mining and in other areas. Building materials, concrete, granite, and groundwater enriched in uranium are the apparent sources of radon. A plaster-resin material containing 10-30% asbestos has been used for fireproofing, acoustics, and, in some cases, decorative purposes. Asbestos concentrations above U.S. occupational concentrations occasionally have been found indoors.

The three general groups of contaminants found indoors are listed in Table I-1. Those in groups II and III are the prime focus of this report. Chapter IV discusses their sources and concentrations, and Chapter V, factors that affect indoor concentrations and personal exposures. The current understanding is reported with an illustrative but not exhaustive review of pertinent related work. Those two chapters discuss the relationships among sources, personal activity patterns, building factors, and ventilation that influence indoor concentrations and individual pollutant exposure. Temperature, light, and especially relative humidity also help to determine concentrations, chemical activity, and effects. Measurement of these effects to the point of predicting the ramifications of altering ventilation or introducing new products is not possible for all pollutants of interest, and in many cases the measurements have not been made. For other pollutants, the data will not be available until instruments are developed. For still others, the sources may be known, but their prevalence and distribution in buildings are not known.

Table I-2 summarizes some typical pollutant concentrations found in the indoor environment and compares them with outdoor concentrations. An indoor-to-outdoor ratio greater than 1 does not imply that hazardous concentrations occur indoors. This table shows that high concentrations of some pollutants have been reported in a variety of buildings that are commonly used during normal daily activities.

Water vapor is not reported in Table I-2 as a contaminant, but it is very important in the indoor environment. At low relative humidities, odors, particles, and such vapors as acrolein may be more irritating. Higher relative humidities favor mold and mite growth,

TABLE I-2

Sources, Possible Concentrations, and Indoor-to-Outdoor Concentration Ratios of Some Indoor Pollutants

Pollutant	Sources of Indoor Pollution	Possible Indoor Concentration[a]	I/O Concentration Ratio	Location
Carbon monoxide	Combustion equipment, engines, faulty heating system	100 ppm	>>1	Skating rinks, offices, homes, cars, shops
Respirable particles	Stoves, fireplaces, cigarettes, condensation of volatiles, aerosol sprays, resuspension, cooking	100–500 $\mu g/m^3$	>>1	Homes, offices, cars, public facilities, bars, restaurants
Organic vapors	Combustion, solvents, resin products, pesticides, aerosol sprays	NA	>1	Homes, restaurants, public facilities, offices, hospitals
Nitrogen dioxide	Combustion, gas stoves, water heaters, dryers, cigarettes, engines	200–1,000 $\mu g/m^3$	>>1	Homes, skating rinks
Sulfur dioxide	Heating system	20 $\mu g/m^3$	<1	Removal inside
Total suspended particles without smoking	Combustion, resuspension, heating system	100 $\mu g/m^3$	1	Homes, offices, transportation, restaurants
Sulfate	Matches, gas stoves	5 $\mu g/m^3$	<1	Removal inside
Formaldehyde	Insulation, product binders, particleboard	0.05–1.0 ppm	>1	Homes, offices
Radon and progeny	Building materials, groundwater, soil	0.1–30 nCi/m^3	>>1	Homes, buildings

Table I-2 (contd)

Pollutant	Sources of Indoor Pollution	Possible Indoor Concentration[a]	I/O Concentration Ratio	Location
Asbestos	Fireproofing	<1 fiber/cc	1	Homes, schools, offices
Mineral and synthetic fibers	Products, cloth, rugs, wallboard	NA	--	Homes, schools, offices
Carbon dioxide	Combustion, humans, pets	3,000 ppm	>>1	Homes, schools, offices
Viable organisms	Humans, pets, rodents, insects, plants, fungi, humidifiers, air conditioners	NA	>1	Homes, hospitals, schools, offices, public facilities
Ozone	Electric arcing, UV light sources	20 ppb 200 ppb	<1 >1	Airplanes Offices

[a]Concentrations listed are only illustrative of those reported indoors. Both higher and lower concentrations have been measured. No averaging times are given. NA, not appropriate to list a concentration.

greater formaldehyde release from particleboard, and deterioration of many materials.

Exposure of some members of the population to many of these pollutants may be determined by the frequency and duration of activities that place them in particular buildings. Hence, knowing use and activity patterns of the population and how these patterns change with age, sex, socioeconomic status, race, and geographic region is important, if we are to assess the population exposure to pollutants.

Seven classes of environmental factors with indoor sources have been identified as having substantial known or reasonably likely effects on human health: sidestream cigarette smoke, radon and radon progeny, mineral and vitreous fibers, formaldehyde, products of indoor combustion, agents of contagion and allergy, and extremes of temperature and humidity.

Chapter VII presents the evidence on health effects of the seven classes of indoor environmental factors. The seven classes were identified as particularly relevant to indoor exposures of the general population. For other contaminants that may represent special concerns for particular indoor locations--such as exposures to organic compounds found in pesticides, cleaning products, varnishes, or craft and hobby products--the reader is referred to the literature developed by the National Institute of Occupational Safety and Health, the Environmental Protection Agency (Office of Toxic Substances), the Consumer Product Safety Commission, the Food and Drug Administration, and the National Center for Toxicological Research. In the case of other compounds that may be in products found in homes or institutional buildings, not enough is known about their concentrations or their effects to evaluate their health effects.

Although this report does not recommend specific standards for the indoor environment, it discusses standards that have already been established for the outdoor, indoor working, and indoor public environments. It is clear that there is a divergence of opinion in the national and international health and regulatory communities as to what constitutes a safe exposure to contaminants and which contaminants are hazardous. Comparison with reported indoor concentrations makes it evident that--by some established ambient, occupational, or indoor standards--current exposures to some contaminants indoors could constitute a health risk to occupants.

A full risk assessment of these pollutants that would identify the population exposed and assign a health-damage function aimed at determining current and projected health consequences of indoor pollution has not been attempted. In many instances, the review of health-effects literature on specific pollutants produces conclusions that are similar. At higher concentrations, these pollutants have known carcinogenic, allergenic, respiratory, or other physiologic effects. However, except for some contaminants that cause irritation, the evidence of direct or important health damage at reported concentrations is not well established. The evidence in some cases--as in passive smoking and the use of gas appliances--is a statistical association between a health response and the source. For

other indoor contaminants, such as radon and asbestos, the direct health effects have been demonstrated in experimental animals and in occupational studies. And for still others, such as formaldehyde, information is from experimental conditions and anecdotal reports of complaints. That these noncriteria pollutants do or will cause harm through current or projected exposures of the general public has not been demonstrated by epidemiologic studies. Indeed, direct evidence from epidemiologic studies may not be forthcoming; epidemiologic studies would be compromised from the outset by uncertainties in indoor pollution concentrations and personal exposure. Nevertheless, if there is consistency of toxicologic and occupational evidence of the harmful nature of specific pollutants at the reported indoor concentrations, then there is reason for serious concern.

In the absence of a confirmed dose-response relationship, careful judgment is required. We should cautiously consider secondary consequences of conservation strategies to the indoor environment. Some pollutants may exert effects only at concentrations above a threshold; others may have no threshold. There may be synergism between pollutants or between pollutants and temperature, humidity, or disease organisms. Some pollutants may manifest effects subtly in behavioral changes. Others may have long latent periods between exposure and effects. In view of the uncertainty in the myriad potential outcomes, one fundamental relationship is clear: if, either deliberately or inadvertently, we systematically modify indoor environments by reducing ventilation or by increasing sources of indoor contaminants without ameliorating efforts, we will be increasing the population exposure to pollutants of indoor origin.

A review of indoor pollutant concentrations and possible health significance would not be complete without a discussion of the implications of these exposures for epidemiologic studies of ambient-air pollution. Several substances generated indoors are present in both indoor and outdoor air, including carbon monoxide, nitric oxide, nitrogen dioxide, and particulate matter. Recent investigations have confirmed that personal exposures to nitrogen dioxide and respirable particles are not well represented by ambient measurements if there are substantial indoor sources. For pollutants of outdoor origin, the evidence indicates that personal and indoor exposures are less severe than outdoor exposures.

These observations have implications for epidemiologic studies attempting to establish a relationship between ambient concentrations and health responses. Air-pollution epidemiology attempts to establish a statistical relationship between the dependent health variable and the independent variable of pollution exposure, correcting for other influential variables, such as age, sex, smoking, occupation, and socioeconomic factors. The air-pollution exposure most often chosen is derived from ambient monitoring appropriate to the study population. Leaving aside the question of spatial representation, consider the potential misclassification of exposure that may result from indoor pollution. Depending on study design and pollutant investigated, the results could overestimate, underestimate, or simply incorrectly estimate the relationship between air-pollution

exposure and health. Table I-3 illustrates by examples the potential bias imposed by indoor air pollution. The effects on the study can reflect a systematic bias or a random bias in exposure. Regardless of whether the pollutant is primarily outdoor or both indoor and outdoor in origin, the effects of a random misclassification of population exposure are the same. It tends to reduce the statistical power of the association.

The imprecision in air-pollution health-effects data may be due in part to indoor air-pollution concentrations. Indoor air-pollution exposures may sufficiently complicate epidemiologic investigations of the effects of outdoor pollutants so that assessments of indoor exposures, and thus larger study populations, will be needed to discern effects.

In Chapter VIII, the objective is to discuss the welfare effects of contaminants in existing enclosures of all general types and the impact of energy-conservation measures on indoor environmental quality. The effects on human comfort and productivity are presented in separate sections, and the effects of contaminants causing soiling in another section. Chapter IX discusses some of the relevant ventilation codes and standards. (Appendix A lists national primary ambient-air quality standards and occupational-safety and -health standards.) The effects of air-cleaning equipment and air diffusion control are also in separate sections, followed by a general discussion on the strategies used for control of indoor pollutants. In an effort to exemplify this complex interaction of choices, Appendix B presents some hypothetical assumptions for a residence.

TABLE I-3

Possible Consequences of Indoor Air Pollution in Epidemiology

Type of Pollution	Source of Bias	Result of Bias
Outdoor pollutant--indoor concentration lower than outdoor; air-conditioning causes further reduction (ozone, sulfur dioxide)	Systematic bias--air-conditioned homes not uniformly distributed	Overstated relationship: if studied (polluted) population has no air-conditioning, this overstates relationship between outdoor pollutant and health effect
		Understated relationship: if studied (polluted) population has more air-conditioning than other population, relationship between pollutant and health effects will be understated
	Random bias--distribution of air-conditioned homes not known	Underestimated relationship: effect of pollutant on health will be understated because unknown number of people have reduced exposure
		Increased error of estimate
Indoor pollutant--indoor concentration higher than outdoor (nitrogen dioxide, respirable particles)	Systematic bias--gas-cooking homes not randomly distributed	Overstated relationship: if gas-cooking homes are found mostly in studied (polluted) area, this will overstate effect of outdoor pollutant
		Incorrect relationship: if different pollutants are studied and outcome health variable is influenced by indoor pollutant concentrations, effect may be understated or not detected and attributed to wrong pollutant
		Increased power: if distribution of indoor air pollution sources or ventilation factors are known, analysis is stratified by exposure and statistical power to measure effects may increase
		Understated relationship: if gas-cooking homes are found mostly in clean area, effect of outdoor concentration will be understated

TABLE I-3 (continued)

Type of Pollutant	Source of Bias	Result of Bias
	Random bias: gas-cooking homes and those with cigarette-smokers randomly distributed among populations studied	Understated relationship: effect of outdoor pollutant may be understated if indoor concentration dominates exposure and exerts an effect
		Increased statistical power: if distribution of smokers or gas stoves is known and study is large enough, statistical power to discern effects may increase

II

SUMMARY AND CONCLUSIONS

CHARACTERIZATION OF INDOOR AIR POLLUTION

The air quality of the indoor environment has been characterized in a limited number of pilot studies. Because of the large variety of distinct indoor environments--single and multifamily residences, offices, hospitals, restaurants, schools, recreational facilities, transportation facilities, etc.--there is a major difficulty in characterizing "the indoor air environment." Moreover, even within one indoor environment differences in structure, in the operation and strength of emission sources, and in human activities add to the complexity of characterizing air quality. The available data, mostly from the residential environment, amply demonstrate the diversity of characteristics of indoor air and help in identifying subjects that warrant further research.

RADIOACTIVITY (pp. IV-2--IV-26)

The data base on sources and source strengths of indoor radon is just beginning to be established. Initial attention focused on building materials and groundwater. Recent evidence from regional studies in the United States points to ground soils (under buildings) as perhaps the major source of radon. Only a small number of buildings in the United States have been measured for radon and radon progeny. Indoor concentrations are affected by various factors, including ventilation rate, deposition of radon progeny on indoor surfaces, and interactions of radon progeny with fine particles from various sources (e.g., tobacco smoke and house dust).

Data from several studies indicate that indoor radon-222 concentrations vary by at least two orders of magnitude, with average values of about 1 nCi/m^3. Such a large range is not surprising, inasmuch as the studies included various types of buildings, building materials, underlying materials, and ventilation rates and used many different measurement techniques. Radon progeny concentrations are often given as potential alpha-energy concentrations (PAEC), expressed

in working levels (WL). Limited measurements indicate that typical average radon progeny concentrations in residential buildings range from 0.004 to 0.02 WL in some houses. Concentrations are much higher indoors than outdoors.

ALDEHYDES (pp. IV-27--IV-44)

Aldehyde concentrations are almost always higher indoors than outdoors. Formaldehyde is the most important aldehyde. Sources of aldehydes include building materials (particleboard and plywood), urea-formaldehyde (UF) insulation, and, to a lesser extent, combustion appliances, tobacco smoke, and other consumer products. Variations in indoor aldehyde concentrations are not well understood, and emanation rates from the various sources are not well quantified. Owing to the time spent inside residences (including mobile homes), offices, and other indoor environments, human exposures to indoor formaldehyde are markedly higher than exposures to outdoor formaldehyde.

Typical indoor formaldehyde concentrations in buildings with products containing urea-formaldehyde resins range between 0.05 and 0.3 ppm, although in some unusual instances concentrations of a few parts per million have been measured in houses with UF foam insulation. In residences with sources of high rates of emission of formaldehyde-containing products, the concentrations typically range from 0.01 to 1 ppm.

CONSUMER PRODUCTS (pp. IV-44--IV-55)

Many consumer products may emit gaseous and particulate contaminants into the indoor environment during their use or even during storage. Most of the chemicals in these gases and particles may be known or can be identified, but the chemical products resulting from mixtures and interactions of them are not known. Likely exposures and durations are poorly understood, even for cases in which the products are used as directed. Willful abuse of aerosols or careless use of solvents in enclosed spaces have resulted in acute and delayed disorders and in death. The carcinogenicity of some compounds, such as benzene and vinyl chloride, has led to voluntary removal from consumer products, but many chemicals with potentially toxic effects are still in wide use. The use of insecticides, pesticides, and herbicides is widespread. Even when applied outdoors, some compounds have been measured indoors and have persisted over a considerable time.

ASBESTOS AND OTHER FIBERS (pp. IV-55--IV-72)

Asbestos is a widespread component of the structural environment in schools, homes, and private and other public buildings. Its release in the indoor environment depends on the cohesiveness of the asbestos-containing material and the intensity of the disturbing

force. Most contamination is episodic, activity-related, and local. Fiber counts and mass concentrations of fibers have been measured and shown to exceed those outdoors, and on occasion they may approximate the occupational limit of 2 fibers per cubic centimeter. Fortunately, during normal use, buildings containing asbestos have not shown indoor fiber counts higher than outdoor counts. Current data are very limited and apply mostly to schools and a few office buildings, but it appears that the general public exposure to asbestos fibers is exceedingly low in public buildings. A systematic and comprehensive survey of indoor asbestos fiber contamination is needed and will require reliable, portable, and continuous monitors. Asbestos control technologies have been applied in various indoor environments. Asbestos removal requires a complex protocol to be carefully applied, because the very activity of removal may cause severe asbestos contamination.

INDOOR COMBUSTION (pp. IV-78--IV-112)

Unvented combustion appliances, especially gas stoves, are major sources of indoor air pollution. Although emission rates from a small number of gas stoves have been determined for several pollutants, the data base is very limited. Indoor concentrations of carbon monoxide and nitrogen dioxide associated with incomplete combustion have been observed to exceed current ambient-air quality standards. Carbon dioxide emission from unvented combustion appliances may build up to concentrations in the range of occupational air quality standards. Local exhaust ventilation appears to be the most effective control strategy for reducing pollutants from combustion. Improved combustion efficiency and source elimination (i.e., adsorbers or a change to the use of electric ranges) are two additional control approaches. Residential wood and coal stoves are also potential sources of indoor contamination. Attached and underground garages can contribute to indoor carbon monoxide, nitrogen dioxide, and particle concentrations.

Carbon Monoxide

Indoor carbon monoxide concentrations are often higher than corresponding outdoor concentrations. High indoor concentrations may be attributed to emission from such sources as gas cooking facilities, attached garages, faulty furnaces, and cigarette-smoking. Typical average indoor carbon monoxide concentrations in residences vary between 0.5 and 5 ppm; observed peak values reach 25 ppm. In public buildings, the indoor concentrations are usually lower than observed residential concentrations, except under conditions of exceptionally heavy smoking, as in bars, or in office buildings with underground garages and improperly designed or malfunctioning HVAC systems.

Carbon Dioxide

The indoor-to-outdoor ratio is greater than 1 for at least 90% of the total monitored hours. Hourly indoor carbon dioxide concentrations often exceed 2,000 ppm. Observed typical outdoor carbon dioxide concentrations are approximately 400 ppm. The principal sources of indoor carbon dioxide are the metabolic activity of occupants and unvented combustion appliances.

Nitrogen Oxides

Emission from cooking appliances and emission from unvented heaters are the principal contributors of oxides of nitrogen in the indoor environment. The range of observed hourly indoor (residential) nitric oxide concentrations is 30-300 ppb, with a maximum of about 500 ppb. Indoor hourly concentrations of nitrogen dioxide vary between 50 and 500 ppb; indoor peaks of 700 ppb have been measured. Typical weekly indoor concentrations of nitrogen dioxide range from 20 to 100 ppb. The upper values in all the ranges just noted are associated with unvented gas appliances.

SMOKING (pp. IV-93--IV-112)

Passive exposure of many nonsmokers to the contaminants in tobacco smoke occurs repeatedly. The indoor concentrations of tobacco-smoke compounds that have other sources exceed the concentrations found outdoors. For many people, the main or sole exposure to numerous gaseous and particulate compounds results from passive exposure to tobacco smoke. Children of smoking parents are among the largest identifiable groups in this category. For the most part, however, the specific contribution of tobacco combustion products to personal exposures has not been documented. Most nonchamber measurements have been of the survey type; many have measured a single component of smoke without reference to outdoor concentrations, ventilation, or air dispersion.

Smoking is the major source of indoor particles, but other human activities (e.g., cooking and vacuum cleaning) also contribute indoor particles. Particulate matter has variable composition, and the data base indicates that there are no constant ratios of indoor to outdoor concentrations. The ratio of observed daily indoor concentrations of total suspended particles (TSP) to corresponding outdoor TSP concentrations varies from 0.3 to 4. Residences occupied by families with pre-school-age children and smokers often have higher indoor than outdoor concentrations. The TSP 24-h ambient-air quality standard, which must not be exceeded more than once a year, is 260 $\mu g/m^3$. The typical range of observed indoor residential 24-h TSP concentrations is 30-100 $\mu g/m^3$, with an observed maximum of 600 $\mu g/m^3$.

Concentrations of fine particles (diameter, less than 2.5 μm) range from 10 to more than 260 $\mu g/m^3$ for a 24-h sample. The higher concentrations are almost always associated with smoking. Concentrations in bars, offices, and cars with smoking can be higher than 500 $\mu g/m^3$.

ODORS (pp. IV-112--IV-147)

Odors arising from occupants and their activities figure in indoor-air quality issues predominantly on the basis of comfort, rather than health. Such routine indoor activities as cooking, smoking, bathroom use, and maintenance give rise to odors that are often disagreeable and in some cases offensive. To a varying degree, almost all building materials and furnishings are sources of odor. The determination of odor attributes--such as intensity, character (pleasantness/unpleasantness), duration, and perceptual threshold--is complex, but can be effectively accomplished with a combination of instrumentation and the use of panels of human observers. Odor controls increase in complexity from good housekeeping to ventilation to masking and, finally, to air-cleaning.

OTHER CHEMICAL POLLUTANTS (pp. IV-26--IV-55, IV-78--IV-93)

Nonmethane Hydrocarbon

The ratio of indoor to outdoor total nonmethane hydrocarbon (NMHC) concentrations is greater than 1 for about 90% of the total monitored hours; that is, the NMHC concentrations observed in the residential environment are almost always higher than the outdoor concentrations. Fluctuations in the indoor concentrations may be associated with cooking, cleaning, and other activities. Typical concentrations in residential buildings vary between 0 and 8.0 ppm, whereas typical outdoor concentrations are between 0 and 3.5 ppm. Measured NMHC concentrations in new office buildings often exceed 10 ppm and reach as high as 50 ppm; this may be attributed to the extensive use of synthetic organic building materials and furnishings in new office buildings, as well as cleaning solvents and maintenance materials.

Ozone

Indoor ozone concentrations are generally lower than outdoor. Unless there is an indoor generation source of ozone from electric arcing or ultraviolet radiation (such as an electrostatic precipitator or a document copier), the ratio of corresponding hourly indoor to outdoor concentrations is almost always less than 1. Ozone is primarily a product of outdoor photochemical reactions. Precursor pollutants leading to the formation of ozone are primarily of automotive origin, but other sources include the combustion of fuels

for heat and electricity, the burning of refuse, the evaporation of petroleum products, and the handling and use of organic solvents. Ozone is highly reactive and decays rapidly by absorption on indoor surfaces. Indoor ozone has been measured at up to 120 ppb; typical concentrations are between 0 and 20 ppb.

Sulfur Dioxide

Indoor sulfur dioxide concentrations are usually lower than corresponding outdoor concentrations. Sulfur dioxide emission indoors is usually small, and, because it is a relatively reactive contaminant, it is absorbed by indoor surfaces. Indoor hourly sulfur dioxide concentrations are typically below 20 ppb.

Particulate Chemical Composition

There is very limited information on the chemical composition of indoor particles. Measured lead concentrations in residences are commonly low--often below 0.5 $\mu g/m^3$. Lead concentrations as high as 2 $\mu g/m^3$ have been measured in residences with wall paints that contain lead compounds or in residences that are near major roads.

Indoor residential concentrations of nitrates are quite low and are driven mainly by the outdoor concentrations. Observed daily indoor concentrations of nitrates do not vary widely--between 1.0 and 5 $\mu g/m^3$, with typical values at the lower end of the range.

The data base on sulfates shows that the indoor 24-h sulfate concentration is usually lower than the corresponding outdoor concentration. The type of fuel used for cooking and heating is important in determining the indoor-outdoor relationship; houses with gas appliances have a slightly higher indoor/outdoor ratio than houses without gas appliances. Sulfur-containing compounds are added to residential gas for detecting leaks of the otherwise odorless fuel. Indoor daily sulfate concentrations range between 2.0 and 15.0 $\mu g/m^3$, with typical values at the lower end of the range.

AIRBORNE MICROORGANISMS AND ALLERGENS (pp. VII-93--VII-116)

For indoor biogenic pollutants, the sparseness of satisfactory measurement methods and the resulting lack of an adequate quantitative data base constitute serious problems. In contrast with other indoor pollutants, biogenic pollutants bear complex and varied organic structures that defy automatic chemical assay. Biogenic agents exhibit limited direct toxicity, more often provoking infection or allergic responses. Bacterial and viral agents can produce infections in humans; however, the indoor transmission of these agents is not fully understood. A broad array of fungi, algae, actinomycetes, arthropod fragments, and dusts have been confirmed as airborne antigen sources that evoke human allergic responses. Indoor biologic pollutants--most

notably bacteria and fungi--also play important roles in the deterioration of surfaces and spoilage of stored materials.

MONITORING AND MODELING OF INDOOR POLLUTION

Indoor air quality monitoring, in addition to pollutant sampling, must involve ventilation-rate measurements and daily activity logs of occupants. In addition, meteorologic data and outdoor pollution measurements may also be needed for the monitoring and assessment of indoor pollution.

Most indoor monitoring studies have relied on instrumentation developed for monitoring workplace or ambient air. The use of conventional monitoring instrumentation is frequently awkward, expensive, and suitable only for a limited number of comprehensive indoor air quality studies. Owing to the special requirements, instruments and sampling strategies are being developed specifically for indoor residential and office environments. The advent of personal monitors has permitted, in a few cases, the startup of monitoring and exposure studies for specific pollutants--nitrogen dioxide and radon. Personal and portable monitors are being developed for carbon monoxide, formaldehyde, and particulate matter. Monitoring the indoor environment, either with fixed-location sampling devices or with personal monitors, requires special protocols addressing pollutant sampling, instrument calibration, source operations, and occupant activity. When indoor monitoring takes place under normal occupancy conditions, the protocol must ensure that the act of monitoring itself avoids influencing those occupancy conditions.

Indoor-air pollution simulation models provide a theoretical framework for relating outdoor pollutant concentrations, meteorologic factors, building factors, ventilation rates, and indoor source and sink dimensions with indoor pollutant concentrations. Most importantly, a validated simulation model must accurately predict a desired concentration for conditions other than those tested experimentally. Depending on ventilation conditions and the geometry of the structure, a single room, a floor, or a whole building may be adequately approximated as a single air-quality compartment (entity). However, if sources and sinks are not uniformly distributed and if the indoor environment is large, pollutant stratification occurs within a building and a multicompartment numerical model is required to simulate the indoor-air pollution concentrations. Almost all numerical models are mass-balance equations that simulate the dynamic relationships among indoor pollutant concentrations, outdoor concentrations, indoor sources, and sinks (including ventilation).

FACTORS THAT AFFECT EXPOSURE TO INDOOR POLLUTION

Exposure is a dynamic concept that is defined as the joint occurrence of two perhaps independent events: the presence of a person in a specific environment and the presence of a pollutant at a specific

concentration in the same environment. Because both human activities and air pollutant concentrations vary spatially and temporally, pollutant concentrations obtained from outdoor monitoring networks are inadequate for determining human exposure. Human activities are among the factors that must be addressed in assessing exposure to air pollutants. They have been studied by many researchers, mostly sociologists, to determine population mobility patterns and time budgets. The results indicate that, on the average, employed Americans spend 90% of the day indoors, whereas homemakers and retired people spend up to 95% of their time indoors. General sociologic studies may be used in air-pollution research, but do not address specific topics of interest for the assessment of human exposure to air pollutants. The exact indoor location (or environmental mode) is of paramount importance in exposure studies. Of all environmental types, the in-transit mode has been studied more extensively than any other microenvironment.

Indoor air quality, and therefore exposure to pollutants, varies geographically as a function of outdoor regional air quality and as a function of the rural, urban, or suburban character of the location of the indoor environment in question. In many residences, the indoor air quality does not vary substantially. In larger buildings with many ventilation zones, indoor air quality may vary in accordance with the function (utility) of each zone. Building factors that influence exposure include the site conditions, such as microclimate and proximity to major outdoor pollution sources, building design (age, size, ventilation systems), occupancy, and building operations. The exact nature of the cause-and-effect relationships between these factors and indoor air quality has not been established.

HEALTH EFFECTS OF INDOOR POLLUTION

Several classes of pollutants with major indoor sources were identified as having important known or reasonably likely effects on human health: sidestream cigarette smoke, radon and radon progeny, mineral and vitreous fibers, formaldehyde, indoor combustion products, agents of indoor contagion and allergens, and, to a lesser extent, temperature and humidity extremes, noise, and odors.

Other classes of indoor pollutants may have impacts on human health, such as consumer-product aerosols and pollutants from hobby, interior-decorating, and maintenance activities (e.g., solvents and pigments). Because the evidence of their effects on health is meager, the Committee could not determine whether specific effects were attributable to them and concluded that effective review at an appropriate depth was not feasible. Many airborne solvents, pigments, mineral dusts, and other products used in hobbies and interior decoration are present in the indoor air. The best data base on the effects of exposure to those substances is that drawn from studies of the industrial workplace, and the reader is therefore referred to the occupational-health literature.

INVOLUNTARY SMOKING (pp. VII-63--VII-81)

Tobacco smoke is a major source of both gaseous and particulate pollution in the indoor environment, and the nonsmoker absorbs measurable amounts of carbon monoxide and nicotine, as well as small amounts of other smoke constituents, owing to involuntary smoking. The carbon monoxide absorbed varies from negligible in well-ventilated office buildings to amounts that raise the carboxyhemoglobin (COHb) concentration by 2-3% in an exposure of 1-2 h.

The COHb produced by the most severe involuntary-smoking exposure likely to occur in everyday living is capable of reducing the maximal exercise capacity of normal healthy adults, but does not measurably affect submaximal exercise capacity. Carbon monoxide has been shown in one study to reduce the amount of exercise that patients with hypoxic chronic obstructive lung disease can perform before the onset of dyspnea.

Patients with angina pectoris have a reduced exercise tolerance after involuntary smoking that may be a combination of psychologic stress and a carbon monoxide-induced decrease in oxygen delivery to the myocardium. Carbon monoxide clearly reduces the amount of exercise possible before the onset of angina in patients with angina pectoris.

Small changes in visual and auditory vigilance have been demonstrated at COHb concentrations that can be produced by involuntary smoking, but no change in tests of complex function has been demonstrated. Involuntary smoking has not been shown to produce acute changes in lung volumes or in a number of small-airway resistance measurements in normal healthy adults. Long-term exposure to cigarette smoke has been related to small-airway dysfunction in healthy nonsmoking adults.

Children whose parents smoke have been shown in some studies to be more likely to have respiratory symptoms, bronchitis, and pneumonia as infants. This relationship has been found in some studies to be independent of parental symptoms, socioeconomic class, and the smoking habits of other children in the household. It shows, in those studies, a dose-response relationship with the number of cigarettes smoked per day by the parents. To the extent that these associations may be due to cigarette smoke, it is reasonable to assume that the particle mass or a specific compound contained therein, rather than nitrogen dioxide or carbon monoxide, is responsible.

A twofold risk of cancer mortality in nonsmoking women has been associated (in a Japanese study) with having husbands who smoke. Apparently, the risk is proportional to the amount of passive smoking.

RADON AND RADON PROGENY (pp. VII-6--VII-21)

The radon gas that diffuses out of radium-bearing building materials, subsurface soil beneath buildings, and well water into the indoor air undergoes radioactive decay. As a result, the indoor air contains both radon gas and alpha-emitting decay nuclides in particulate form, herein referred to as "radon progeny."

The health effects of radon and radon progeny are well established from studies of workers. Exposure to radon and its progeny at high concentrations has resulted in several hundred excess cases of lung cancer among uranium miners in the western United States. The health effects of much smaller amounts of radon progeny from indoor exposures can be estimated on the basis of a linear, no-threshold dose model, which yields upper-limit estimates of excess cancer in populations exposed to various indoor concentrations of radon and radon progeny. Lifetime cumulative exposures to radon progeny that result from current indoor exposures are lower by approximately a factor of 100-10,000 than those received by the U.S. uranium miners who have been studied.

The reliability with which the uranium-miner lung-cancer experience can be extrapolated to the effects of indoor exposures to radon progeny among the general population is limited by several important differences between the populations and by uncertainty about the extent of the effect of cigarette-smoking on the incidence and latent period for lung cancer related to radon progeny. The population differences include: (1) an adult, male, healthy working population versus a general population that includes the very old, the very young, and the chronically ill; (2) coexposures to relatively high concentrations of silica dust and diesel exhaust among the miners versus coexposures to relatively low concentrations of household pollutants and consumer products among the general population; and (3) differences in the ethnic and social backgrounds and smoking histories among the different populations.

ASBESTOS AND OTHER FIBERS (pp. VII-38--VII-49)

The inhalation of asbestos fibers can lead, many years later, to pulmonary fibrosis, lung cancer, and mesothelioma of the pleura and peritoneum. All these diseases have been seen in humans who had chronic occupational exposures to airborne asbestos fibers, and they have all been reproduced in animals. Lung cancer and mesothelioma have also been seen in humans who had no occupational exposures, but who lived either in the same households as asbestos-workers or in neighborhoods where the ambient air had increased asbestos-fiber concentrations resulting from proximity to an asbestos-related industry or a geologic anomaly that acted as a source of airborne fiber.

Asbestos and asbestos-containing products--such as ceiling tiles, floor tiles, pipe insulation, and spackling compounds--were widely used in homes and public buildings because of their excellent thermal and acoustic insulation and structural properties. When these materials and products are displaced or disturbed by abrasion of deteriorating surfaces during housekeeping and maintenance operations, renovations, redecorating, or, especially in public buildings, malicious mischief, asbestos fibers can be released into the air. Concern about the inhalation of fibers that can result has led to extensive and expensive programs to remove asbestos, under controlled conditions, from accessible regions of public buildings, such as schools and libraries.

Fibrous materials used as substitutes for asbestos include glass fiber, rock wool, and slag wool. They have been shown, in animal injection and implantation studies, to be capable of producing lung fibrosis and mesothelioma. However, they are much less important in this regard than asbestos, and there is no corresponding human-health evidence associated with the forms in which they are used in industrial and consumer products. Thus, their substitution for asbestos appears to be beneficial, inasmuch as such substitution reduces the risk associated with asbestos exposure.

FORMALDEHYDE (pp. VII-21--VII-37)

Formaldehyde has been the subject of numerous complaints regarding irritation of the eyes and respiratory tract, nausea, headache, rash, tiredness, and thirst. These symptoms have been reported mainly by residents of mobile and conventional homes in which formaldehyde-yielding products have been identified. Documented cases of bronchial asthma due specifically to formaldehyde are few; more commonly, asthma is aggravated by the irritating properties of formaldehyde.

Aqueous solutions of formaldehyde damage the eye and irritate the skin on direct contact. Repeated exposure to dilute solutions may lead to allergic contact dermatitis. Poisoning from ingestion is uncommon, because the irritancy of formaldehyde makes ingestion unlikely.

A preliminary report from the Chemical Industry Institute of Toxicology has shown that formaldehyde induces nasal cancer in laboratory rats and in some of the laboratory mice similarly exposed at the high dose. Nasal cancer has developed in the group of rats exposed at 15 ppm and 6 ppm, and dose-related histologic changes of the nasal mucosa in rats exposed at 2 and 6 ppm. Although the human mutagenic and teratogenic potential of formaldehyde is not known, it has exhibited mutagenic activity in a wide variety of organisms.

Data on the health effects of other environmental factors and their interactions--such as cigarette-smoking history, variability of health status, age, and genetic predisposition (which may modify responses to formaldehyde)--have not been adequately evaluated. That makes it difficult to assess accurately the health risks attributable to exposure to formaldehyde. However, the complaints of residents of homes with formaldehyde-containing products are similar to complaints made by persons studied in the laboratory at similar formaldehyde concentrations; hence, these health complaints may be related to formaldehyde exposure in the home. Accordingly, a substantial proportion of the U.S. population may be likely to develop symptoms as a result of exposure to formaldehyde at low concentrations. It has been estimated, on the basis of laboratory tests and various kinds of population surveys, that perhaps 10-20% of the general population may be susceptible to the irritant properties of formaldehyde at extremely low concentrations. For example, some persons report mild eye, nose, and throat irritation and other symptoms at concentrations less than 0.5 ppm, and some note symptoms at concentrations as low as 0.25 ppm.

These concentrations could also cause bronchoconstriction and asthmatic symptoms in some susceptible persons, and chronic exposure to low concentrations can result in sensitization. There appears to be a wide range of individual susceptibility to formaldehyde exposure. We cannot determine the exact numbers of susceptible people residing in indoor environments where exposure to formaldehyde could produce adverse responses. On the basis of estimates of the number of susceptible persons among the general population and the estimate that about 11 million persons in the United States now reside in mobile homes of varied age, construction, and quality, it may be concluded that a substantial number of persons are at risk of developing adverse health effects associated with formaldehyde.

INDOOR COMBUSTION (pp. VII-49--VII-63)

The combustion of fossil fuels in air results in the generation of effluent streams containing carbon monoxide, nitric oxide, nitrogen dioxide, formaldehyde, carbonaceous particles, and other products of incomplete combustion, as well as the products of complete combustion--carbon dioxide, water, and sulfur dioxide. When the effluents are not vented to the outside, as in the case of most gas ranges and some space-heaters, the effluents are mixed into the indoor air.

The percentage increase in the indoor concentration of the combustion effluents resulting from such indoor sources is generally greatest for nitric oxide and nitrogen dioxide. For homes with gas ranges, indoor nitrogen dioxide concentrations are frequently twice as high as outdoor concentrations. The long-term integrated concentrations can exceed the national annual ambient-air quality standard (NAAQS) of 100 $\mu g/m^3$ (0.05 ppm) in some houses. Although chronic animal inhalation studies and community air-pollution epidemiology studies using central monitoring-station data have not established that exposures at or near the NAAQS for nitrogen dioxide produce measurable health effects, several recent studies of the health status of children living in homes with gas ranges have shown that they had more respiratory illness and poorer respiratory function than children living in comparable homes with electric ranges.

Increases in carbon monoxide sufficient to cause measurable health effects are usually associated with improperly operated flames or especially prolonged use of unvented space-heaters. Both carbon monoxide and nitric oxide bind with hemoglobin and reduce tissue oxygenation. Carbon monoxide from indoor combustion sources and sidestream cigarette smoke can be shown to cause measurable increases in COHb of exposed persons, but the health implications of such increases remain speculative. The importance of increased carbon monoxide and formaldehyde concentrations in indoor air was discussed above.

INDOOR AGENTS OF CONTAGION AND ALLERGY (pp. VII-81--VII-116)

There is considerable evidence that a number of contagious-disease organisms--including those associated with influenza, Legionnaires' disease, tuberculosis, measles, mumps, and chicken pox--are capable of airborne transmission in the indoor environment. Other respiratory diseases, such as the common cold and pulmonary infections, involve airborne transmission. Because of the important role of respiratory diseases in overall acute morbidity, airborne transmission of contagious agents is important in the indoor environment.

The droplet-nucleus theory--whereby liquid particles emitted from the human respiratory tract evaporate to a particle size that can remain airborne for a period sufficient to be carried by natural air currents or convective ventilation flows and later deposited in the human airways--is generally accepted and used as a basis for transmission models. The effect of reduced ventilation in residences and offices on the incidence of infections is unknown.

Only a few airborne allergens are found in enclosed spaces. Their health effects are difficult to estimate, although their impact is sometimes appreciable.

EFFECTS OF INDOOR POLLUTION ON HUMAN WELFARE

Effects on human welfare are taken to include loss of productivity, human discomfort, and effects on materials, primarily soiling and corrosion of exposed surfaces.

SOCIOECONOMIC STATUS (pp. VIII-1--VIII-3)

Members of low income classes are more likely to live in poorly insulated housing with higher air-exchange rates. Several reports have indicated that gas stoves or unvented gas or kerosene heaters are used for supplemental space-heating in northern cities. The percentage of homes with smokers appears to be inversely related to parental educational level. Lead intoxication in children occurs disproportionately in lower-income urban populations; higher ambient airborne-lead concentrations may contribute. However, some potential sources of indoor pollution may occur more frequently in the middle and upper income brackets. Many consumer products, as well as coal and wood stoves, exemplify such sources. Although the distributions of these and other factors may be functions of socioeconomic status that cause some segments of society to be more or less disadvantaged with respect to a hazardous indoor environment, the available data allow little more than speculation.

PRODUCTIVITY (pp. VIII-13--VIII-19)

There is a growing recognition of the difficulties in clearly demonstrating the linkage between environmental quality and productivity. Perhaps as a result of these difficulties, there appears to be a slackening of research in this subject. Anecdotal or observational evidence can be found to support the conclusion that improving air quality should improve productivity, but objective documentation does not appear to exist or to be readily available. The most promising avenues for research appear to be those which demonstrate direct health effects of the various pollutants. Nevertheless, under the modern, broad definition of "productivity," a reduction in productivity is an almost certain consequence of pollution itself.

SOILING AND CORROSION (pp. VIII-19--VIII-27)

Reduced indoor environmental quality can result in degradation and deterioration of indoor materials, furnishings, and artifacts. As efforts required for maintenance and housekeeping increase, the costs of owning or operating a building increase. To protect property or reduce costs of operation, more stringent control of indoor environmental quality may be required than may be indicated for protection of the health of occupants.

DISCOMFORT (pp. IV-147--IV-169, VIII-3--VIII-13)

Control of indoor environments in nonindustrial facilities is designed to provide a degree of comfort acceptable to the occupants. When a stimulus (whether odor, temperature, humidity, noise, or air pollution) is changed beyond an acceptance threshold, there can be adverse effects. This kind of environmental control may be difficult to provide. However, the comfort-discomfort relationship may be one of the more important aspects to consider in evaluating the performance of indoor environmental control systems.

CONTROL OF INDOOR POLLUTION

CONTROL STRATEGIES (pp. IX-39--IX-49)

Three basic strategies have been identified: source removal, dilution, and air-cleaning. In each of these classifications, methods can be selected that will reduce exposure. The appropriate strategies to be used, either separately or in combination, must be selected with respect to other and interacting factors--thermal, acoustic, energy-conservation, and economic.

CODES AND STANDARDS (pp. IX-2--IX-16; Appendix A)

Minimal requirements of acceptability are often stated in building codes and standards in terms of air-exchange rules, temperature limits, and so forth. These documents tend to cause minimal requirements to be established for such direct effects as temperature, humidity, and odors, but may not be sufficient to provide for other effects, such as air pollution or noise. Nor do these codes consider the interactions that can occur among these factors and other system features such as lighting, thermal load, and spatial requirements.

AIR DIFFUSION CONTROL (pp. IX-16--IX-22)

Indoor air quality is most commonly controlled by forced-air systems. However, if diffusion control is designed without considering possible stratification of air within a room or a building, there may be local violations of thermal, humidity, or air-quality criteria for acceptability, and occupants may be exposed to conditions other than expected from the design.

INDOOR ENVIRONMENTAL CONTROL SYSTEMS (pp. IX-16--IX-22)

Control methods for indoor environments require specification of environmental criteria and definition of the control variables. The environmental criteria that are identified in this document are health, comfort, welfare, energy consumption, and costs. The control variables identified are spatial requirements, lighting factors, thermal factors, air quality, and acoustic factors. Although environmental criteria and control variables can be identified and described, the capability of sensing the appropriate variables and controlling the system to meet the specified criteria is severely limited. Moreover, most indoor environmental control systems must attempt to respond activity or passively to all five of the control variables simultaneously.

Residential air-conditioning systems are conventionally designed to respond to spatial, thermal, and air-quality variables and, to a limited extent, acoustic variables. For larger facilities, such as offices and schools, air-conditioning systems must also respond to variations in occupancy and lighting loads, in addition to spatial, thermal, air-quality, and acoustic factors. For other functional spaces (e.g., concert auditoriums, art galleries, museums, and hospitals), some or all of the variables must be controlled with additional precision.

For many years, air-conditioning systems were designed to meet the required environmental criteria (primarily thermal) at minimal first cost. Operating costs were not considered important as first costs, because energy was relatively inexpensive, compared with labor and material. However, as the costs of energy increased rapidly during the last decade, operating costs became a major factor in environmental control. Energy-conservation measures were implemented in many

buildings to reduce operating costs. Some of these measures (e.g., improved system efficiency through better maintenance) had no impact on environmental control, but others (e.g., reduced ventilation, heat, and lighting) had potentially adverse effects. One reason for the occurrence of adverse effects was lack of understanding, by building operators and owners, of the interrelationships among ventilation rates, lighting, and health responses. As an example, changes in lighting can affect thermal loads, which affect ventilation rates. Conversely, results of some energy-conservation measures have indicated that indoor environmental quality need not be degraded and, in fact, may be enhanced by these changes (e.g., reduced stratification within occupied spaces). Thus, two general conclusions can be drawn: control methods may not be capable of adequately responding to environmental changes as energy-conservation measures and cost contraints are applied; and the quality of the indoor environment need not be degraded, but can be enhanced if care is exercised in the selection and implementation of the energy and cost constraints.

AIR-CLEANING EQUIPMENT (pp. IX-22--IX-39)

Air-cleaning equipment for residential and commercial applications is generally limited to particle filtration. Some gas and vapor removal equipment is available, primarily for commercial applications. Methods of rating or evaluating the performance of the gas and vapor removal systems are not yet available. Methods are available for rating and evaluating particle removal equipment, but they are simplistic and outdated. Moreover, in-place methods of system evaluation are available only for special cases, such as hospitals and laboratories.

COST EFFECTIVENESS (Appendix B)

Several economic models are available that can be used to evaluate the costs associated with various control strategies. Cost-effectiveness models that incorporate life-cycle costing are needed for decision-making.

An approach to estimating the impact of residential energy-conservation measures on air quality is discussed in Appendix B. The approach has not been validated or put into practical use, but is presented for illustration and discussion.

III

RECOMMENDATIONS

The Committee on Indoor Pollutants recognizes that decisions affecting the quality of the indoor environment are being made by manufacturers, government agencies, builders, building operators, professional organizations, and private individuals. The decisions encompass a broad range of activities in our society, with important and long-term consequences. Federal agencies are planning energy-conservation programs in buildings, contemplating the banning of some products, and estimating the health risk associated with indoor pollutant exposures. State and local government units are considering revisions of building codes, ordinances to prohibit smoking in public buildings, and requirements that asbestos insulation be removed from schools. It can be presumed that there are similar examples of decision-making at various levels in the private sector that affect indoor environmental quality. In view of the possible impact of these decisions, the Committee is concerned that policy, research, and economic decisions be formulated with proper understanding of their implications for the quality of the indoor environment. For specific indoor contaminants, two basic inadequacies in the available data must be resolved rapidly: poor definition of population exposures and lack of understanding of the health and welfare consequences of exposure to contaminants in the indoor environment. This chapter presents the Committee's general and specific recommendations for remedying these inadequacies.

We have observed that the existing data base is, for the most part, derived from pilot studies or anecdotal reports. The results of the pilot studies reveal the complexity and diversity of the information that must be looked at in evaluating the quality of indoor environments. In some cases, the potential health significance of exposure to indoor contaminants has been alluded to; but the full extent of a potential problem with respect to types of contaminants, concentrations, and numbers of people exposed has not been determined.

We believe that the research problem is large and requires national coordination. A national coordinated research program would have the following advantages:

- It would provide reasonable allocation of research efforts among various federal agencies, national laboratories, and academic institutions.
- It would provide standards for study design and quality assurance that are sufficient for various decision-making purposes.
- It would provide integration of research activities and dissemination of the information derived.
- It would provide maximal effectivenss of available funding.

The objectives of such a research program should be these:

- To determine the various sources and distribution of selected pollutants, to measure their ranges of concentrations, and to identify populations at risk.
- To understand how the contaminants move inside buildings, how they mix and react, and the rate at which they are removed or dissipated under various conditions.
- To characterize indoor pollutant emission source strengths under actual conditions.
- To develop and test the effectiveness of control technologies.
- To determine the effects of energy-conservation measures on indoor air quality.

To meet some of these objectives, there must be research aimed at developing improved instruments and at strategies for using them in the study of indoor pollutants. Instruments used to characterize indoor air quality reflect the early stages of this scientific endeavor. The sampling devices used in indoor studies were originally designed for sampling outdoor or industrial air. Many commercially available ambient-air monitors are bulky, noisy, and expensive and have not been tested for interferences that may be encountered indoors. These devices have mostly been judged to be inadequate for investigating indoor air quality, because their sensitivity, accuracy, and precision are not sufficient for the measurement of pollutants in the small volumes of air in indoor spaces. The Committee recognizes the importance of recent developments in the field of personal monitors; but it also recongizes the need for a simultaneous effort devoted to the development of air sampling devices designed specifically for indoor environments. The new instruments must be designed to record short-term peaks in concentrations.

Instrumentation alone is not sufficient for characterizing the indoor environment. Numerous variables indoors (perhaps more than outdoors) must be considered: some sources of pollutants are peculiar to some indoor environments; there are differences in structure type, ventilation, and other characteristics that affect pollutant concentrations; and activity patterns and time budgets of occupants vary. Human activity patterns and time budgets, which are essential in determining total exposures to air pollutants, are now derived from population surveys. These surveys do not require (and therefore do not obtain) information relevant specifically to indoor air quality. The Committee sees the need for analysis of these variables and their

effects and recommends the development of model strategies (for sampling, etc.) or protocols as guidance for future research. (The formulation of model sampling strategies, for example, is not meant to stifle innovation in sampling designs, but rather to establish the model to be used for comparing data obtained by researchers whose objectives differ.) The Committee recommends the formulation of strategies and protocols for measuring the strength of various indoor pollutant-emitting sources and for assessing the effectiveness of control devices and procedures in abating pollution.

The Committee recommends the formulation of a standard format for reporting data and the development of protocols for standardized statistical approaches that will require only minimal analysis to be used in validating numerical models. These would help to reduce difficulties in comparing existing data and facilitate the development of valid conclusions; conclusions now are often based on exceedingly small samples.

Even comprehensive information about the quality of the indoor environment would not permit determination of total pollutant exposure. It must be recognized that people are exposed to many of the same pollutants outdoors, in transit, in the occupational-industrial environment, and elsewhere. The relative importance of each kind of environment can be established and priorities can be set if and only if pollution exposures in all distinct environments are characterized. Lack of a complete assessment may lead to inefficient allocation of scientific effort and control funds in each kind of environment. The Committee believes that the research efforts to characterize indoor air pollution and human exposures indoors must continue and intensify, if we are to determine total human exposure to pollutants and understand environmental contamination and its effects on health and the quality of life.

The remainder of this chapter presents specific recommendations for research, grouped by class of indoor contaminant discussed in the body of the report, and discusses the need for increased understanding of indoor pollutants in general and the need for consumer protection.

RADON

Nationally coordinated investigations on radon and its progeny should take place on two levels. A well-funded and coordinated national survey of radon concentrations in a representative sample of residential buildings is necessary to estimate the exposure of the total population to radon and radon progeny. Monitors that use the track-etch plastic chip may be adequate for integrated measurements for such national surveys, because they are inexpensive and are specific to radon. However, the performance of these and other passive devices needs to be carefully evaluated. Inexpensive instruments for measuring radon concentrations on a short-term basis need to be developed. These instruments should be available to local health agencies and others for spot surveys. On another level, research on the transport and transformation of radon inside buildings

deserves special attention. Rates of emanation of radon from various sources, building materials, soil, and groundwater should be evaluated in a variety of on-site and controlled conditions. The effectiveness of strategies for abating or eliminating indoor radon (including the use of material sealants and fine-aerosol collection devices) must be evaluated.

There is an urgent need to study the health effects of radon and radon progeny. On the basis of known effects in miners exposed to radon and radon progeny at relatively high concentrations, a plausible case can be made that a substantial fraction of the lung-cancer incidence in nonsmokers is due to the alpha-radiation dose to the respiratory tract epithelium from inhaled and deposited radon progeny particles. It is urgent that this observation be examined quantitatively by studies of appropriate human populations already known to be exposed at below 100 WLM and preferably in the range of 20-50 WLM. It is known that in some geographic areas large populations are being exposed to radon gas and radon progeny particles in their residences at concentrations that, although much lower than those in uranium mines, are substantially higher than those in most residences. Epidemiologic studies of populations exposed to radon and radon progeny are reasonable and can provide the information necessary for the establishment of realistic and needed exposure-response relationships. These research studies should include examinations to determine early pathologic changes other than tumors (e.g., changes in sputum cytology and chromosomal aberrations), with special attention to the possibility of a relationship of those changes to the eventual development of lung cancer. Such studies should be performed as soon as possible.

FORMALDEHYDE

Simple and reliable passive monitors that would easily satisfy the requirements of large surveys of buildings for formaldehyde emission do not exist. Monitoring formaldehyde is extremely difficult, because of the influences of temperature, humidity, and some analytic problems that affect its detection. Both continuous and passive monitors are needed with sensitivities in the range of 10-30 parts per billion. A national survey for indoor formaldehyde exposures is <u>not</u> needed, but a systematic study of formaldehyde concentrations in a variety of indoor locations is needed to estimate potential exposure of humans. This study would also identify sources of indoor formaldehyde by type of building and decorative materials and would evaluate the effects of ventilation rates and other variables on the concentration. Regular measurements over specified periods would help to identify formaldehyde emission rates of insulating building materials and furnishings as functions of temperature, humidity, ventilation rate, and material age.

Formaldehyde emitted from buildings and consumer products has resulted in complaints of adverse health effects by people in some mobile homes and in some conventional residences and other buildings.

Some of this emission occurs over long periods. Thus, long-term effects in humans continuously exposed to formaldehyde at low concentrations need to be studied. There is a particular need to assess the carcinogenic potential in the concentration ranges of human exposures, inasmuch as one study in one strain each of rats and mice has shown that long-term exposure (lasting 24 mo) caused nasal cancer. Humans have been and are now being exposed to formaldehyde in several types of occupations and in a variety of structures. Epidemiologic investigations are needed to assess the human health effects of formaldehyde, the magnitude and duration of exposure, and the influence of cigarette-smoking habits and the presence of other contaminants. The mutagenic, embryotoxic, and teratogenic effects must be included in the epidemiologic and animal studies. In humans exposed to formaldehyde, the mechanisms of airway and target cell responses must be evaluated and characterized as to sensitization and adverse effects in susceptible population groups, such as asthmatics and persons with chronic obstructive lung disease. Exposure-effect relations and the mechanisms involved in the biologic effects require further animal toxicologic research. Formaldehyde should be restricted to the extent that household consumer products and building products in normal use will not release potentially hazardous or irritating amounts of formaldehyde into indoor air.

TOBACCO SMOKE

Tobacco smoke has shown some evidence of being a major contaminant in many indoor environments. Involuntary exposure to tobacco smoke should be assessed to identify locations and populations with high exposure and to determine the factors that contribute to high exposures indoors. Physical and biologic evaluation of tobacco-smoke constituents should be continued. Tobacco-smoke constituents should be tested for their toxic effects, their ability to act as mutagens or promoters of carcinogenesis, and their effects in combination with other indoor pollutants. In addition, such properties of tobacco smoke as mass and age, chemical composition, irritation factors, and odor components should be examined to learn how they are affected by ventilation rate, occupancy, extent of smoking, air-cleaning, and other control strategies.

The extent to which passive exposure to sidestream tobacco smoke produces respiratory tract symptoms and functional decrements in nonsmokers, especially children, needs further documentation and measurement. Prospective studies of children in homes with smokers would be especially desirable to determine rates of lung maturation and illness frequency during childhood and adolescence.

Information on the potential health effects of exposure of nonsmokers to tobacco smoke should be widely disseminated. The "energy-cost penalty" of providing adequate ventilation in indoor environments that permit smoking should be analyzed in a variety of public buildings. Increased cigarette taxation as a mechanism of

reimbursement for the cost of the additional air-conditioning needed to remove tobacco smoke should be explored by governments at all levels.

ASBESTOS AND ASBESTIFORM FIBERS

A systematic survey is needed for the evaluation of the distribution, integrity, and concentrations of asbestos in buildings that contain or are thought to contain asbestos material. However, before this survey can be conducted, there is a need to develop new instruments to record fiber counts continuously, with size determination and possibly asbestiform-fiber identification, because current sampling and analytic techniques are inadequate.

Synergistic and interactive toxic effects of asbestos fibers in combination with other air pollutants, particularly organic vapors, should be examined in animal toxicologic and mutagenicity studies. Although some asbestiform fibers themselves do not appear to constitute an immediate health concern, their role as initiators or promoters in various disease processes should be studied. The incidence of mesothelioma in humans should be monitored via a registry and appropriate surveillance methods, to detect cases associated with substantial nonindustrial exposure to asbestiform fibers.

Guidelines should be developed for the control of exposure to airborne asbestos fibers during maintenance, renovation, and reconstruction in buildings that contain asbestos and asbestos-bearing shingles, tiles, plaster, etc.

COMBUSTION

Indoor combustion produces a number of contaminants. Among the contaminants that deserve special attention are nitrogen dioxide, carbon monoxide, respirable particles, nitrosamines, and polynuclear aromatic hydrocarbons. The rates of their emission from sources of indoor combustion have not been adequately evaluated. The Committee recommends that controlled chamber experiments be conducted to determine the products and their rates of emission from various types of combustion under various conditions. These experiments should focus principally on gas and electric cooking appliances and supplemental heating systems, such as natural-gas, propane, and kerosene heaters and coal- and wood-burning stoves. Air-venting and air-cleaning systems should be studied as means of reducing indoor concentrations of contaminants.

Indoor concentrations of combustion products have only recently been surveyed. Combustion products are present in many indoor locations, such as restaurants, cafeterias, homes, hotels, buildings with attached garages, and recreational facilities that use gasoline-powered equipment. More comprehensive and systematic surveys are needed to identify the range of combustion-product concentrations encountered indoors and the numbers of people exposed to them. These

studies should determine the population exposure to nitrogen dioxide over both the short and the long term. In addition, the applicability of ambient-air fixed-location monitors for recording nitrogen dioxide and carbon monoxide concentrations indoors and for assessing individual exposures should be studied.

Chemical reactions and rates of removal of emitted gases should be determined. Nitrogen dioxide formation and removal should be examined in detail. This will involve the simultaneous measurement of other gaseous compounds. The effects of reduced air-exchange rates, zone ventilation, and source modification on pollutant reactions should be assessed. As with other indoor contaminants, there is a general need to improve instrumentation. Both nitrogen dioxide and carbon monoxide monitors are available for passive integrating sampling and for continuous monitoring. However, for indoor use, they need to be evaluated with respect to interferences. To evaluate short-term personal exposures, lightweight continuous monitors for oxides of nitrogen need to be developed. Evaluation of personal exposures to respirable particles is currently limited to integrated samples. Lightweight portable samplers or direct-reading monitors that can measure mass concentration over shorter periods are needed.

Nitrosamines can be formed during cooking and smoking. However, very few detailed investigations of the concentrations, mechanisms of formation, and potential control methods have been done.

The polynuclear aromatic hydrocarbons can be formed during high-temperature combustion of organic matter. Some of them have been found indoors as a result of emission from self-cleaning ovens and fireplaces. Pilot studies should be initiated to evaluate the extent of emission of polynuclear aromatic hydrocarbons and their indoor concentrations.

The magnitude and prevalence of decreases in pulmonary function and increases in respiratory tract infection rates among children living in homes with gas ranges and homes with electric ranges need to be determined more accurately, and there are several related issues that require clarification:

- Whether the effects are due entirely to the increased nitrogen dioxide concentration in the gas-stove homes or are influenced by the presence of other combustion effluents from the stoves, such as carbon monoxide, formaldehyde, and particles.
- Whether the effects can be related more closely to peak concentrations or to long-term average exposures.
- Whether the effects of exposures to sidestream cigarette smoke and to nitrogen dioxide are additive or synergistic.
- Whether exposure-response relationships can be developed and, if so, whether they indicate an effective threshold concentration for peak or average exposures.

The influence of reductions in air infiltration rates in existing buildings on indoor concentrations of combustion products needs to be determined. Among the potentially serious health consequences of reductions in infiltration are:

- Creation of greater pressure differentials between the indoors and the outdoors, which could reduce the effectiveness of the venting of combustion sources, e.g., furnaces and water-heaters. Problems in proper venting could cause substantial exposures to carbon monoxide (which could lead to severe intoxication and fatalities), as well as greater exposures to carbon dioxide, nitrogen dioxide, formaldehyde, and particles.
- Increase in chronic exposures to carbon monoxide, nitrogen dioxide, formaldehyde, and particles at low concentrations from unvented combustion sources; this could lead to increases in respiratory infections in children and decreases in their lung function.

CONSUMER PRODUCTS

The use of some consumer products can lead to the release of aerosols and gases indoors. The gaseous compounds of concern are mostly organic vapors. Among the compounds of principal concern are aldehydes and polynuclear aromatic hydrocarbons that evolve from plasticizers; nitrosamines and hydrocines from rubber products, combustion products, and cleaning agents; polychlorinated biphenyls (PCBs) from burnt-out ballasts in fluorescent lights; and a variety of other middle- and higher-molecular-weight organic substances from pesticides. A few studies have noted the presence of many of these compounds indoors. However, no systematic survey has been done. With regard to the evolution of organic molecules from pesticides, organochlorinated pesticides should be examined first, including aldrin, dieldrin, endrin, benzene hexachloride, pentachlorophenol, kepone, chlordane, and DDT. The emphasis should be on the determination of body burdens of these compounds and specifically the relative contributions of inhalation, ingestion, and absorption to body burdens.

The contents of consumer products should be investigated, and the chemical constituents should be tested for their toxic, mutagenic, carcinogenic, and teratogenic properties. In particular, there should be toxicologic studies of solvents, vapors, aerosols, and particulate compounds present in these products. Their synergistic and interactive effects with other indoor pollutants should be tested.

Consumer products whose use involves the release of gaseous and particulate materials to the indoor atmosphere should be so labeled, with their components. Warning labels for consumer products that can seriously pollute the indoors should state that they are to be used only in areas with adequate ventilation and should stipulate the possible consequences of their use when there is inadequate ventilation.

AEROPATHOGENS AND ALLERGENS

Little is known about the sources, concentrations, and survival rates of many aeropathogens in homes and other buildings. Relationships among the incidence of respiratory infections, concentrations of aeropathogens, and air-exchange rates in buildings must be examined. The urgency arises from the recent modifications in building ventilation codes that recommend reduced ventilation rates in residential and commercial structures. A sample of commercial, institutional, and residential buildings should be evaluated for the types and concentrations of aeropathogens under a variety of conditions of occupancy, human activity, ventilation, humidity, temperature, and contaminant control. Special attention should be given to the newer energy-efficient buildings and buildings with drastically reduced ventilation. The potential for infectious contamination from air-cleaning filters, air heat-exchangers, air humidifying systems, and air-conditioning systems deserves special attention.

Other agents in the indoor environment known to produce allergic responses include pollens, household mites, molds, animal dander and excreta, and bacterial spores. Further work is needed to characterize the size distribution of allergen aerosols, their sources, and the conditions that are conducive to their generation. The airborne concentration of allergens in the indoor environment has been determined in only a few instances, and the relationship between indoor concentration and response is poorly understood. Case-control epidemiologic and immunologic studies are needed to clarify exposure-effect relationships. Such studies will require improved instrumental and analytic techniques to facilitate characterization of concentrations of allergens and of the variety of microorganisms in the indoor environment. Synergism of biologic and nonbiologic agents should be explored in animal toxicology studies.

Some acute allergic responses, such as "humidifier fever," are of unknown etiology. The pathologic agents in immunologic and case-control epidemiologic studies need to be identified. The pathogenic process by which repeated small exposures to some allergens often lead to irreversible fibrotic lesions, as in bird-fancier's disease, should be elucidated, and the potential of other, more common indoor pollutants to produce such disease states should be evaluated.

VENTILATION STANDARDS AND CONTROL STRATEGIES

Knowledge of ventilation rates is of primary importance in studies of indoor contaminant concentrations. Given the variety of residential living units and other public and private facilities, it is not surprising that very little information exists to characterize air-exchange rates. Studies should begin to characterize air-exchange rates in existing buildings by building type, geographic location, occupant life styles, building operation, and observed average pollutant concentrations during the different seasons. Smaller-scale

studies over a considerable period are recommended to characterize air exchange and its effects on occupant behavior in a representative number of buildings. This information would help to show the relationships among air-exchange rates, pollutant concentrations, pollutant generation, occupant behavior, and physiologic effects, as well as the effects of energy-conservation programs aimed at reducing air infiltration rates.

With the objective of maintaining indoor air quality, while not adversely affecting heating and cooling costs, there is a need for engineering studies to determine alternative strategies for air dilution of pollutants. Case studies of specific buildings may be useful in determining the efficiency of selected filtration-ventilation schemes. For example, it may be economically preferable to filter (or scrub) recirculated air or a mixture of recirculated and makeup air to maintain indoor air quality. Studies should be done on the effectiveness and energy-conservation implications of pollutant sensors used to activate air-dilution or air-cleaning systems. Engineering studies on both air handling and air treatment systems are encouraged. Although specific buildings may be studied in detail, the application of the findings should be generic; i.e., the lessons learned should be applicable to other buildings.

Standard methods should be developed and applied to evaluate the performance of in-place environmental control systems and components. Improved methods of providing acceptable and efficient air diffusion for thermal and contaminant control should be developed. Life-cycle cost evaluations should be made on the basis of current and projected energy costs to characterize these costs for future use in alternative designs of residences and large buildings. Air-conditioning, heating, and ventilation systems especially should be evaluated, in connection with other building characteristics.

EXPOSURE STUDIES

Proper assessment of indoor contamination needs a perspective that only total-exposure studies can provide. The relative contributions to individual and population exposures of the contaminants encountered indoors, with both indoor and outdoor sources, must be evaluated. For contaminants with multiple entry routes, the contribution of inhalation of indoor pollutants must be compared with all other contributions to body burdens. Only with this evidence can research and control efforts be prudently allocated. Under current conditions, studies of total exposure to many contaminants are limited by the available instrumentation and, to a lesser extent, by analytic methods. Such efforts are expensive. The extrapolation of their results is constrained by the smallness and often the unrepresentativeness of the samples of participants, by the inadequacy of the information on activity patterns in the population at large, and by lack of understanding of the distribution of indoor and outdoor pollutant sources and the pathways that contribute to body burdens. However, studies of total exposure and an understanding of

activity-related concentration data will eventually advance our knowledge of pollutant exposure. This knowledge is a prerequisite to rational allocation of resources for warranted reductions in population exposures.

The Committee urges investigation in the behavioral aspects of indoor environments, specifically the relationships among performance, sense of well-being, contaminant concentrations, and stress. Temperatures, odors, and noise outside preferred ranges can reduce productivity, especially in self-pacing tasks. The relationship between productivity and the quality of the indoor environment needs to be determined. It is recognized that relationships between the behavioral variables and pollutant concentrations may be difficult to establish. Simultaneous measurements of trace organic vapors, water-vapor content, conductivity, noise, light, temperature, and air exchange rates should be pursued.

EDUCATION

Public education offers an effective way of reducing exposure of the population to many contaminants encountered indoors. People informed about the potential for exposure to pollutants from consumer products, tobacco smoke, combustion products, etc., will exercise some control to reduce the pollutant concentrations in their environments. For the most part, their options for controlling these pollutants are limited to source maintenance, ventilation control, and, to some extent, air-cleaning. Information about maintaining a clean indoor environment and assessing indoor spaces for potential contamination before purchasing or renting a structure and a variety of suggestions could be disseminated through health-maintenance organizations, regional health-planning agencies, public-affairs offices, the Environmental Protection Agency, the Department of Housing and Urban Development, the Department of Energy, the Consumer Product Safety Commission, and a variety of other federal and state agencies. The General Services Administration, the armed forces, and the Department of Housing and Urban Development are responsible for many residences and other buildings. Through certification of minimal acceptable occupancy standards, these federal organizations could develop strategies to ensure that indoor spaces under their jurisdiction are free from hazardous concentrations of contaminants.

Professional and trade associations could be instrumental in developing and disseminating information. These associations are encouraged to establish standards for acceptable practice, with respect to manufacturing, designing, building, and using products, equipment, and structures that influence the quality of the indoor air.

IV

SOURCES AND CHARACTERIZATION OF INDOOR POLLUTION

This chapter addresses several chemical pollutants with respect to their sources, concentrations, and indoor-outdoor relationships. In addition, with the aim of characterizing the general quality of the indoor environment, it considers temperature, humidity, unwanted sound, and electromagnetic radiation, such as the radiofrequency, infrared, visible, ultraviolet, and x-ray portions of the spectrum.

In the case of some pollutants, information on health effects is scanty, at best. To the extent possible, the health effects of such pollutants are discussed here. Detailed discussion of the health effects of other pollutants, on which more information is available, is to be found in Chapter VII.

Radioactivity and formaldehyde emitted indoors from building products are discussed in the first two sections of this chapter. Consumer products, a generic source of indoor pollutants of many types, are discussed next. The chapter proceeds with sections on asbestos and fibrous glass (which occur in different forms in many indoor environments), combustion processes (especially of unvented cooking and heating appliances), and tobacco smoke (a highly complex and ubiquitous mixture of pollutants). Several indoor air pollutants can be recognized by their odors. Such odors are often the first indications of deterioration in air quality and may themselves affect people's well-being adversely; hence, they are treated as a distinct category of pollutant in this chapter. Air temperature, radiant temperature, and air velocity and humidity affect the quality of the indoor environment through physiologic and sensory responses, so the thermal environment is also discussed in a separate section. Other physical factors of the indoor environment, such as noise and electromagnetic radiation, are discussed briefly in a final section.

The diversity of subjects discussed in this chapter is evident. Some of the pollutants considered here may be associated with voluntary behavioral patterns, such as tobacco-smoking, whereas others may be related to involuntary and unavoidable exposure, such as exposure to substances emitted from building materials. The reader should not infer any order of priority among the pollutants discussed here. An effort to attach priorities would require judgments on exposures and effects,

and the order of discussion is not intended to indicate the application of such judgment.

RADIOACTIVITY

INTRODUCTION

Radioactivity and ionizing radiation occur naturally throughout the biosphere, both because of the presence of primordial radioactive elements and their decay products in the earth and because of natural processes (primarily cosmic radiation) that produce radionuclides or direct radiation fields. These natural sources expose humans to radiation both outdoors and in buildings. The magnitudes of various contributions to total radiation dose vary from place to place and between outdoors and indoors, and the type of radiation dose depends on the radiation source. At one extreme, the cosmic-radiation field delivers a dose to the entire body; this dose is not affected greatly by the presence of a building and may be characterized primarily on the basis of altitude. At the other extreme, airborne alpha-emitting radionuclides may deliver doses specifically to the lungs, and their concentrations indoors may be strongly affected by the nature of building materials and other sources, such as soil and water, and by building operations, such as ventilation. As an intermediate case, the gamma-radiation field arising from radionuclides that are fixed in place typically exposes the whole body and is affected by radionuclide concentration, proximity, and shielding.

In the discussion that follows, we refer to radioactivity concentrations and radiation fields and, by inference, to radiation doses from sources that are inside and outside the body. Radioactivity is given in curies; 1 Ci = 3.7×10^{10} becquerels, so 1 pCi = 0.037 Bq. Radiation fields can be specified in terms of energy flux; but it is more conventional in the present context to use units of dose rate, in which case the type of radiation has to be indicated. We use the rad as the unit of (absorbed) dose when specifying gamma-radiation fields (1 rad = 0.01 J/kg, so 1 mrad = 1×10^{-5} J/kg). For gamma doses, the dose in rads is numerically equal to the dose equivalent (DE) in rems. A distinction must be drawn between the "tissue dose," that actually received by tissue and therefore including self-shielding by the body, and the "air dose," that deposited in air in the space under consideration.

It is useful to summarize the dose-rate contribution in the United States from radiation arising outside buildings. Three recent summaries are those of the National Council on Radiation Protection and Measurements[34] and the U.N. Scientific Committee on the Effects of Atomic Radiation,[39] which depended heavily on Oakley[38] for U.S. data, and the 1980 BEIR report of the National Research Council.[35] External radiation, that arising from sources outside the body, may be divided into two categories, cosmic and terrestrial. The average tissue dose rate outdoors from cosmic radiation is approximately 28 mrads/yr; the dose rate indoors is slightly reduced by overhead

shielding (the NCRP report assumed a 10% reduction in average exposures). This contribution has a substantial altitude dependence, increasing from about 26 mrads/yr at sea level to about 50 mrads/yr at 1,600 m, the altitude of Denver. The average outdoor population-weighted tissue dose rate from terrestrial radionuclides--due principally to gamma rays from potassium-40, the thorium-232 series, and the uranium-238 series--is approximately 35 mrads/yr. This dose rate varies substantially because of geographic variations in the distribution of these radionuclides. For estimating average terrestrial dose rates, the NCRP assumed that indoor dose rates were 20% lower than outdoor rates. (It also assumed that the tissue dose was 20% less than the air dose.) Internal radionuclides contribute important beta and gamma doses (about 15 mrads/yr to most of the body, primarily from potassium-40) and an important alpha dose (even if that to the lungs from radon and its progeny is excluded). The alpha dose arises primarily from internally deposited uranium-238 and -234, radium-226 and -228, and polonium-210 and varies greatly with body organ. One of the larger contributions, about 3 mrads/yr, is the polonium-210 alpha dose to the cells lining the bone surfaces. However, alpha particles have a greater biologic effectiveness than gamma rays, so the absorbed alpha dose contributes a DE some 10 times greater than that of the same (absorbed) dose of gamma radiation. Table IV-1 shows estimates of various contributions to DE rates, in millirems per year, which are numerically equal to tissue dose rates (in millirads per year) for gamma and beta radiation. For alpha radiation, a quality factor of 10 was assumed (based on relative biologic effectiveness), although 20 is now recommended.[19] The value given for lung dose from inhaled radionuclides assumed a radon-222 concentration in air of 0.15 nCi/m^3 (and slightly less than equilibrium amounts of its radioactive decay products, or progeny). The resulting DE has the largest value in the table. Nonetheless, this value appears more appropriate for outdoor than for indoor air, in which higher radon concentrations are found.

All indoor dose rates from natural radiation sources are affected by buildings, and those from inhaled radionuclides are affected most strongly. The only natural airborne radionuclides of importance are radon and its progeny, principally the series beginning with radon-222, the alpha-decay product of radium-226 (a member of the uranium-238 series). Radon is a noble gas that can move from the site of its formation, giving it a substantial opportunity to reach air that is inhaled by humans. The short-lived decay products of radon--polonium, lead, and bismuth--are chemically active and thus can be collected in the lungs, either directly or through particles to which they attach. The most important dose arises from alpha decay of the polonium isotopes. The decay sequence beginning with radium-226 is shown in Figure IV-1, and, from the biomedical point of view, effectively ends with lead-210, because of its half-life of about 20 yr. Because the alpha energy associated with decays of the short-lived products to lead-210 poses the main risk, progeny concentrations are often expressed as the associated "potential alpha-energy concentration" (PAEC) in air. The unit conventionally used for PAEC is the working

TABLE IV-1

Summary of Average Dose Equivalent Rates from Various Sources of Natural Background Radiation in the United States[a]

Radiation Source	Gonads	Lung	Bone Surfaces	Marrow	GI Tract
Cosmic[b]	28	28	28	28	28
Cosmogenic radionuclides	0.7	0.7	0.8	0.7	0.7
External terrestrial[c]	26	26	26	26	26
Inhaled radionuclides[d]	—	100[e]	—	—	—
Radionuclides in the body[f]	27	24	60	24	24[g]
Totals (rounded)	80	180	120	80	80

[a]Reprinted with permission from NCRP.[34]

[b]With 10% reduction for structural shielding.

[c]With 20% reduction for shielding by housing and 20% reduction for shielding by the body.

[d]Lung only; doses to other organs included in "Radionuclides in the body."

[e]Local DE rate to segmental bronchioles = 450 mrems/yr.

[f]Excluding cosmogenic contributions.

[g]Excluding contribution from radionuclides in gut contents.

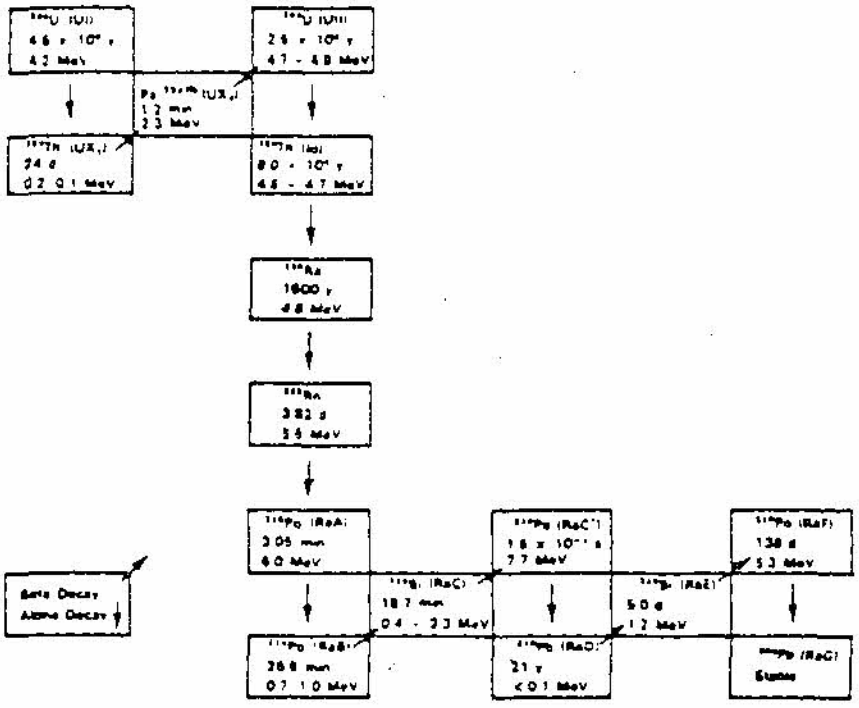


FIGURE IV-1 Principal decay scheme of uranium-238 to radon-222 to lead-206, showing alpha and beta decay; decay energies in millions of electron volts. Reprinted with permission from National Council on Radiation Protection and Measurements.[34(p. 4)]

level (WL), defined as 1.3×10^5 MeV/L, the PAEC if radon-222 at 100 nCi/m^3 is present with equilibrium amounts of its progeny. Dose (and DE) rates may be inferred from the PAEC on the basis of relatively complicated modeling, provided that the progeny particle size distribution and other factors are prescribed.

The character of a building may affect occupant radiation exposure in three principal ways: the building serves as a container for indoor-generated radon and its associated progeny, whether from building materials, underlying soil, or water and gas; the building materials contain natural gamma-emitters (potassium-40, the thorium-232 series, and the uranium-238 series); and the building shields occupants from cosmic or external terrestrial radiation. The last two effects tend to cancel one another. The building structure may, in unusual circumstances, also protect occupants from outdoor radon-progeny concentrations. However, the indoor concentration is ordinarily larger than the outdoor, and outdoor-generated radon usually contributes a small additive term to indoor concentrations. If this term is ignored, the steady-state indoor radon concentration for a fixed indoor radon source strength is inversely proportional to the air-exchange rate, the rate at which the indoor air is exchanged for outdoor air. The air-exchange rate for most U.S. buildings is around 1/h, with 0.5/h to 1.5/h typical for residences (windows closed). The air-exchange rate and other removal mechanisms also affect the ratios of radon-progeny concentration to radon concentration. Lack of removal implies activity ratios of 1, but substantially lower values have been observed. An equilibrium factor (F) is often defined as the ratio of the actual PAEC to the PAEC that would be associated with a specific radon concentration if the progeny were in equilibrium with this concentration.

This section characterizes indoor airborne radionuclides and radiation, summarizes measurements of actual concentrations or radiation fields, briefly indicates control measures, and suggests subjects for further research. The major emphasis is on radon and its progeny. The radionuclides in this decay chain, even at typical outdoor concentrations, cause larger radiation doses to internal organs than all other airborne radionuclides. Furthermore, the radon and progeny concentrations may be substantially higher indoors, particularly in buildings with low air-exchange rates. In addition, building occupants receive external whole-body radiation from radionuclides fixed in building materials and soil, and these doses are also given substantial treatment. This radiation arises principally from several primordial radionuclides--potassium-40 and members of the thorium-232 and uranium-238 decay series--with concentrations of around 0.1 pCi/g or greater in rocks, soil, and derivative building materials. These are also the decay chains in which radon-220, radon-222, and their progeny occur.

SOURCES OF RADIONUCLIDES AND RADIATION

Building Materials

Radionuclide Content. Few measurements and no wide-scale surveys of the radionuclide content of U.S. building materials have been made. Surveys of materials in Europe are summarized in UNSCEAR 1977,[9](p. 50) which gives activity concentrations of potassium-40, radium-226, and thorium-232. As examples, average values for the concrete sample groups examined range from 0.9 to 2.0 pCi/g for radium-226, 0.8 to 2.3 pCi/g for thorium-232, and 9 to 19 pCi/g for potassium-40. By comparison, the ranges for brick are about 50% higher; those for cement are similar, except for potassium-40 (which is 50% less); and those for natural plaster are lower by about a factor of 5.

Available U.S. data (Table IV-2) show concentrations in the same range, assuming that the series radionuclides are sufficiently close to equilibrium to permit comparison. In a number of cases, U.S. workers have examined the radionuclide contents of concrete in the course of selecting materials for low-background facilities for use in radiation-counting;[25] the values obtained are consistent with the European data, although somewhat lower. The observed concentrations are also within the range of values typical for major rock types and soils. Concentrations for building materials not derived from crustal components, such as wood, are much lower.

Measurement programs have recently been initiated to characterize the radionuclide contents of building materials as a basis for understanding the resulting effect on the indoor radiation environment. Kahn et al.[25] have reported measurements of concentrations in various building materials in the Atlanta area; potassium-40, radium-226 progeny, and thorium-232 progeny concentrations for samples of concrete, brick, and tile are given in Table IV-2. Lawrence Berkeley Laboratory has begun to survey concretes and other materials as part of a program on indoor air quality; radionuclide contents for concrete and rock-bed samples from a number of areas are given in the table.[18]

Considerably greater radionuclide concentrations may be found in building materials that contain residues from industrial processes. The principal example of such materials in the United States is concrete blocks incorporating phosphate slag (essentially calcium silicate), a byproduct of phosphate production. As discussed by Roessler et al.,[92] this slag contains most of the radium-226 and uranium-238 found in the phosphate ore. For the electric furnace process used in Florida, concentrations in the ore are about 60 pCi/g, and the slag has similar concentrations. A plant in Alabama (using Florida and Tennessee phosphate ores) sold slag to companies in Alabama, Mississippi, Tennessee, Georgia, and Kentucky. The concrete produced by these companies has radium-226 concentrations estimated, and in some cases measured, to be about 20 pCi/g.[25] Phosphogypsum (essentially calcium sulfate produced by treatment of phosphate ores with sulfuric acid) may also be used for building materials, particularly wallboard. In this treatment, radium-226 follows the

TABLE IV-2

Average Radionuclide Content of U.S. Building Materials

Material	Concentration,[a] pCi/g: Uranium-238 Series[b]	Thorium-232 Series[b]	Potassium-40	Comments
Concrete[29]	0.29-1.32	0.28-1.58	6.6-9.8	Summarized measurements to select counting-room materials
Concrete[25]	1.4	1.5	21	Atlanta area
Brick[25]	1.8	1.8	17	Atlanta area
Tile[25]	1.9	1.1	8	Atlanta area
Concrete[18]	0.2-1.0	0.2-1.0	5-12	Nine metropolitan areas (preliminary values)
Solar rock bed[18]	1.5	1.4	25	New Mexico (preliminary values)
Concrete[49]	0.9-2.0	0.8-2.3	9-19	European concretes[c]

[a]Except where noted, each entry is average for sample group; range is given if several sample groups were examined.

[b]Because various members of decay series were detected, results in each column are directly comparable only if series equilibrium may be assumed.

[c]For comparison.

calcium, leading to tens of pCi/g in the gypsum; but such gypsum has not been used on a large scale in U.S. wallboard. In contrast, concrete that incorporates phosphate slag may have been used in approximately 100,000 homes.[25] Finally, some fly ash from coal-fired power plants has been used in cement production, and this use may continue. Heretofore, it has not been thought to contribute substantially to the radionuclide content of the resulting building material.[25] Emanation measurements on fly-ash concretes are now being performed at Lawrence Berkeley Laboratory.

Radon Emanation. The effective radon-222 generation rate in building materials depends on the radium-226 content, which varies widely, and on the percentage of radon formed that does not remain lodged in the matrix of the material. Radon that is not fixed in place may move through the matrix by diffusion or, if the material contains large air spaces, by convection. Diffusive movement depends on the diffusion length of the material in question and on its thickness. The extent to which these processes occur depends not only on the material's characteristics, but also on environmental conditions--pressure, temperature, and moisture content. A rule of thumb sometimes cited (e.g., UNSCEAR[9]) is that 1% of the radon-222 generated from materials in walls and ceilings escapes into the adjacent air space. However, recent measurements at Lawrence Berkeley Laboratory and elsewhere have indicated that a considerably higher fraction can escape, e.g., from concrete. Ingersoll et al.[18] cited escape-to-production ratios of 0.08-0.25 for radon-222 from concrete. (Radionuclide contents for the sample groups examined are indicated in Table IV-2.)

Of most direct interest for indoor air quality is the actual emanation rate, often given as picocuries per square meter per second and sometimes as picocuries per gram per second. Measurements for various materials give emanation rates over a wide range. For example, European gypsum board and bricks yield radon-222 at about 0.3×10^{-4} pCi/m^2-s, whereas rates for European concretes range from 0.001 to 0.2 pCi/m^2-s.[24, 31] Preliminary measurements of radon-222 emanation rate per unit mass for sample groups of concrete from U.S. metropolitan areas (Table IV-2) give averages that range from 0.4 to 1.2 pCi/kg-h (0.8 pCi/kg-h yields approximately 0.03 pCi/m^2-s for 0.1-m-thick concrete). Several rock samples from solar-heat storage beds averaged 0.5 pCi/kg-h, although radium-226 contents were considerably higher than those for the concrete samples.[18] The resulting indoor radon-222 concentrations depend on the amount of such material in the structure, the interior volume, and the air-exchange rate. For an air-exchange rate of 1/h and a ratio of indoor emanating surface to indoor volume of 0.5 m^2/m^3, an emanation rate of 0.03 pCi/m^2-s corresponds to a radon-222 concentration of about 0.04 nCi/m^3. If the equilibrium factor is 0.5, this would yield a PAEC of about 0.0002 WL. Direct measurement of emanation rates of materials made with industrial byproducts (such as phosphate-slag concrete) is underway, but results are not available. Because these materials may contain 20 times as much radium-226 as a typical concrete, radon-222 contributions

of up to several nanocuries per cubic meter of radon-222 and a corresponding increase in the PAEC could be expected if the same emanation ratio pertains.

Measurements of emanation rate vary by more than an order of magnitude,[19] so it is difficult to use radium content to predict the contribution of a particular material to indoor radon content. For this reason, more comprehensive information on diffusible fraction, diffusion length, etc., and their dependence on material or environmental factors is required before we can characterize building materials on the basis of radionuclide content. If this information becomes available, radionuclide contents may then be helpful in characterizing indoor concentrations on a broad scale, e.g., by geographic area. However, the dependence of diffusion and emanation rates on environmental factors, such as pressure and temperature, and on the moisture content of the material may limit the possibility for such characterization.

In some cases, radon-220 ("thoron") and its progeny, ordinarily present at much lower concentrations than radon-222 and its progeny, may assume importance, particularly when mechanisms exist for transporting emanating radon-220 rapidly into the air space of interest. In comparison with the half-life of radon-222, the much shorter half-life of thoron, 55 s, causes the measured radioactivity in curies to be a characteristic of secondary interest. However, the PAEC still gives a relatively direct indication of possible dose to the lung. One WL of radon-222 progeny has the same PAEC as that associated with progeny in equilibrium with thoron at 7 nCi/m^3. To the extent that uranium-238 and thorium-232, which have similar half-lives, have similar activities in source materials, the PAEC from their progeny, radon-220 and -222, can reach similar values if rapid transport mechanisms exist. This may occur, for example, in solar buildings that sweep air through rock or concrete thermal-storage beds. A few efforts have begun to measure thoron emanation rates, but results are not yet available.

Gamma Radiation. The energies and intensities of photons from decay of natural radionuclides have been well characterized. The external dose from radionuclides in building materials is due to the gamma rays emitted and depends on the geometry of the structure and attenuation by the materials, as well as the gamma-ray energies. A simple expression may be derived for the gamma-ray air dose in a hole in an infinite uniform medium:[25]

$$\dot{X}_\infty = (2.43\ \mu rad/h)(E_U C_U + E_{Th} C_{Th} + E_K C_K),$$

where C_U, C_{Th}, and C_K are the concentrations (in picocuries per gram) of uranium-238 and its progeny, thorium-232 and its progeny, and potassium-40, respectively, and E_U, E_{Th}, and E_K are the average gamma-ray energies per disintegration of the same radionuclides (including disintegration of the progeny for the uranium and thorium series). Using E_U = 1.72 MeV, E_{Th} = 2.36 MeV, and E_K = 0.156 MeV,[25] $\dot{X}_\infty = 4.2C_U + 5.7C_{Th} + 0.38C_K$, in microrads per

hour. The stated dose contributions from the uranium and thorium series are slightly less than those cited elsewhere, e.g., by Krisiuk et al.,[27] who may have used older information on decay schemes. For the radionuclide contents cited in Table IV-2, the three terms in the expression for $\dot{X}_\infty$ contribute comparable amounts. (An analogous expression for the dose from a flat plane is cited in the section on soil.)

For an actual structure, the geometry is complex and varied; in addition, the building materials may attenuate the external radiation dose from other sources. Moreover, radon-222 and its progeny may be present in the material at less than equilibrium values, thereby decreasing the corresponding gamma-ray dose. The radon-222 escape-to-production ratio is most often in the range of low to 0.25, causing a small reduction in the value of $\dot{X}$. The effects of geometry and attenuation cannot be so simply characterized. Dose-rate expressions from various workers, pertaining to a variety of structures, have been summarized.[37] Some of these expressions account for reduction of the dose rate from outdoor sources. Moeller et al.[33] described a computer program suitable for analysis of varied geometries.

The infinite-geometry case yields air dose rates of about 8 μrads/h for a potassium-40 concentration of 8 pCi/g and uranium-238 and thorium-232 series concentrations of 0.5 pCi/g. An infinitely thick slab of such material would contribute about half this dose rate at its surface. As discussed earlier, a typical outdoor tissue dose rate from terrestrial radionuclides is 35 mrads/yr or 4 μrads/h. (Owing to shielding by the body, the tissue dose rate is about 20% less than the air dose rate.)

Soil and Groundwater

Radionuclide Content. Radionuclide concentrations of major rock types and soil have been summarized.[34] U.S. soil values of 0.6, 1.0, and 12 pCi/g have been stated for uranium-238, thorium-232, and potassium-40, respectively, on the basis of 200 measurements of gamma-ray dose rate cited by Lowder et al.[10] These values vary by a factor of around 3 from place to place. Values for crustal rocks[34] typically lie within this same range, but are considerably higher for some formations. For example, the phosphate rocks of Florida contain the uranium-238 series at tens of picocuries per gram, but normal amounts of thorium-232; commercial uranium ore bodies in the United States have uranium-238 concentrations of hundreds of picocuries per gram and higher.

Radon Emanation and Transport. The uranium-238 series, typically present in soils and rocks at concentrations of about 1 pCi/g, includes radium-226, the source of radon-222. The actual radon-222 emanation rate from the ground depends, as for building materials, on the percentage of diffusible radon, diffusion length, and other transport mechanisms (including groundwater) in the soil. A review of available

measurements of radon-222 indicates a mean emanation rate from the soil of 0.42 pCi/m^2-s.[59] Given this value for the ground under a one-story house, and assuming that the emanated radon finds its way into the indoor air, the soil could account for indoor radon-222 at about 1 nCi/m^3 at a typical air-exchange rate of 1/h. Because emanation rates vary by at least a factor of 10 from place to place, this potential contribution can also be expected to vary substantially among U.S. buildings.

The soil as a source of radon-222 can be characterized directly by emanation measurements or, if disequilibrium and transport mechanisms (including groundwater) are known, indirectly by measurements of members of the uranium-238 series. Because of the relative ease of measuring gamma rays, the indirect methods may be more appropriate for large-scale surveys intended to characterize the contribution of soil radon by geographic area. Gamma-ray source measurements may also be less sensitive to changes in pressure, temperature, and moisture content than emanation-rate measurements (see UNSCEAR[59] and NCRP[34]). Moreover, variations in emanation rate may correlate with factors that affect air-exchange rates and may thus complicate assessment of the importance of soil as a source of indoor radon.

The mechanisms by which radon may be transported into buildings have been studied little. Soil-gas measurements, which have yielded results of 100-2,000 pCi/L (Kraner;[26] Scott;[54] and unpublished measurements by Lawrence Berkeley Laboratory), may be relevant to this question, because they may help in characterizing the radon content of air trapped beneath buildings. Emanation rates themselves are useful only for placing an upper limit on the potential of soil as an indoor source. However, a more detailed understanding of radon transport in soil could provide a basis for using emanation data to estimate the amount of radon that may accumulate beneath houses and be transported indoors. Such collection and transport mechanisms may be greatly affected by changes in barometric pressure, soil moisture content, temperature gradients, and wind.

The actual pathway by which radon enters a building from the soil appears to vary substantially with building design and construction practice. In houses with concrete basements that are closed to the outdoors, radon may enter by diffusion through the basement floor, by convection within basement walls, and by movement through cracks, designed openings, or penetrations in either of these components. Even in communities where numerous measurements have been performed, it has not been possible to determine the relative importance of these mechanisms.[1,2] In some mining communities, sealing of cracks has proved relatively successful in reducing indoor radon content, but the effectiveness of this method in general has not been evaluated. The movement of radon from the point of entry to other parts of the building depends on internal construction and building use. Even in buildings with ventilated crawlspaces, the radon concentration in the crawlspace air may be considerably higher than outdoors, and a substantial amount of the radon emanating from the soil may reach the interior space by transport from the crawlspace.

More comprehensive information on how radon is transported is needed for the development of techniques to prevent radon from entering buildings and for establishment of a correlation between the radium-226 content in soil and the indoor radon content attributable to this source.

Gamma Radiation. The gamma dose from radionuclides in soil may be expressed in a fashion analagous to that for building materials; the air dose rate (μrad/h) at 1 m above the ground due to natural emitters uniformly distributed in the soil has been given as $X_{plane} = 1.82C_U + 2.82C_{Th} + 0.179C_K$ for C_U, C_{Th}, C_K in picocuries per gram.[4] More current data on decay schemes may alter this slightly. As noted above, concentrations of natural radionuclides in soil and rock vary from place to place, causing comparable variations in dose rates. The air dose rate is estimated at 2.6 μrads/h on the coastal plain (the Atlantic and Gulf coastal areas), 10.2 μrads/h on the Colorado Plateau, and 5.2 μrads/h on the rest of the contiguous United States (NCRP,[34] based on nuclear-plant site surveys). The materials in a building can provide significant shielding of occupants from gamma rays from local radionuclide concentrations, but the radionuclide content of the materials may more than compensate for this shielding.

Radon from Utilities

Water. Measured concentrations of radon-222 in well water in Maine and New Hampshire average 53,000 and 101,000 pCi/L, respectively.[31] More recent measurements have been performed in Maine.[12,14] Lawrence Berkeley Laboratory has found concentrations of 100-7,500 pCi/L in tapwater from wells or underground reservoirs associated with houses and has correlated use of such water with increases in indoor radon content. Radon-222 in water can quickly transfer to air, with efficiencies of 30-90%, depending on water use;[11] a concentration of 10,000 pCi/L can raise average indoor radon-222 content by about 1 nCi/m^3. It is not known how widespread such water concentrations are, nor how closely they correlate with high radium content in surface soils and rocks.

Natural Gas. Concentrations of radon-222 in natural gas in the Houston area have been found to average approximately 50 pCi/L at STP.[10] Concentrations in distribution lines at various points in the United States were found to average about 20 pCi/L.[22] The resulting concentrations in U.S. residences due to natural-gas combustion have been estimated to be less than 0.1 nCi/m^3, even with unvented burners.

INDOOR CONCENTRATIONS AND RADIATION FLUXES

Airborne Radionuclides

Radon Concentrations. Data from several sources tabulated by UNSCEAR[59] indicated that indoor radon-222 concentrations vary by two orders of magnitude, with average values of about 1 nCi/m^3. Such a large range is not surprising, considering that the studies included various types of buildings, building materials, underlying materials, and ventilation conditions and used many measurement techniques. More recent measurements have confirmed this wide variation.

A wide variation is expected even for conventional housing, because the air-exchange rate typically ranges from about 0.5/h to 1.5/h in such buildings, and further variation in air-exchange rates occurs because of window or door openings and mechanical ventilation systems. The soil under a house can be expected to be the principal contributor to the indoor radon concentration in most cases. As noted earlier, a typical soil emanation rate, if the radon goes into the interior of a house with an air exchange rate of 1/h, would contribute radon-222 at about 1 nCi/m^3. Inasmuch as soil emanation rates and effective capture by the house vary by an order of magnitude and air-exchange rates vary widely, a large range of indoor concentrations would result.

As indicated in Table IV-3, homes monitored in New York and New Jersey were found to have an annual average radon-222 concentration of 0.3-3.1 nCi/m^3 in the living space, with a geometric mean of about 0.8 nCi/m^3.[9] Similar measurements in Austria yielded a geometric mean of 0.42 nCi/m^3.[56] In these studies the mean indoor concentrations were 3-4 times as great as local outdoor concentrations. Spot measurements of homes in the San Francisco area, made during the summer with windows closed and with an average air-exchange rate of 0.4/h, showed concentrations averaging 0.3 nCi/m^3.[15] Spot measurements in Illinois showed a substantial incidence of concentrations greater than 5 nCi/m^3; six of 22 houses had concentrations of 10 nCi/m^3 or more.[53]

High radon-222 concentrations have been found in uranium-mining areas and in buildings that use materials high in radium. In houses monitored in Bancroft, Ontario, 50% of the sample had concentrations greater than 3 nCi/m^3, over 25% had concentrations greater than 7 nCi/m^3, and about 6% had concentrations greater than 15 nCi/m^3.[21] High concentrations have also been found in homes in mining areas in the United States; at Grand Junction, Colorado, PAECs corresponding to radon-222 at up to hundreds of nanocuries per cubic meter have been measured. In a survey of several Swedish houses built with alum-shale concrete, the average radon-222 concentration was 7 nCi/m^3;[58] more recent data showed average concentrations of 15 nCi/m^3 or more for residences built entirely of alum shale.[57]

Radon-222 concentrations of 0.6-22 nCi/m^3 have been found during spot measurements of energy-efficient homes, many of which had low air-exchange rates; these measurements were taken with windows closed, and the air-exchange rates were measured simultaneously.[16] Concentrations and air-exchange rates have also been measured in

TABLE IV-3

Selected Radon and Radon-Progeny Measurements In U.S. Residences[a]

Location	Radon-222 Concentration,[b] nCi/m^3	Progeny PAEC Concentration,[b] WL	No. Residences	Type of Measurement	Comments
Ordinary Areas:					
Tennessee	--	0.008(0.0008-0.03)	15	Grab	Shale area; mostly concrete construction
Boston	0.07(0.005-0.2)	(up to 0.002)	7	Grab and ventilation	Single family; air change rate, 1-6/h
New York-New Jersey	0.8[c](0.3-3.1)	0.004[c](0.002-0.013)	21	Several integrated measurements over year	17 single family, 3 multiple family, 1 apartment building
Illinois	(0.3-33)	--	22	Grab	Wood-frame construction, unpaved crawl spaces (windows closed)
San Francisco area	(0.4-0.8)	--	26	Grab and ventilation	Air change rate, 0.02-1.0/h (windows closed)
U.S.-Canada	(0.6-22)	--	17	Grab and ventilation	Energy-efficient houses; air change rate, 0.04-1.0/h (windows closed)
Special Areas:					
Grand Junction, Colorado	--	0.006[c]	29	Integrated year round	Controls for remedial-action program (which has included houses in range 0.02-1 WL)

Table IV-3 (contd)

Location	Radon-222 Concentration,[b] nCi/m^3	Progeny PAEC Concentration,[b] WL	No. Residences	Type of Measurement	Comments
Florida	--	0.004	28	Integrated year round	Controls on unmineralized soils
	--	0.004(0.0007-0.014)	26	Integrated year round	Controls on unmineralized soils
	--	0.014	133	Integrated year round	Houses on reclaimed phosphate lands
Butte, Montana	--	0.02	56	Integrated year round	Intensive mining area
Anaconda, Montana	--	0.013	16	Integrated year round	Intensive mining area

[a]Data from W. J. Barnes, personal communication, cited in George and Breslin.[9] Single-family residences except where noted.

[b]Averages; values in parentheses are ranges. All measurements are in living space; values in basements are typically higher.

[c]Geometric mean.

conventional houses in England[5] and in houses at Elliot Lake, Ontario;[4,5] the measured radon concentrations were consistent with those observed for conventional houses elsewhere.

Radon-Progeny Concentrations. Radon-progeny concentrations are often measured as potential alpha-energy concentrations (PAEC), given in working levels. Indoor concentrations of radon-222 progeny were measured at the Environmental Measurements Laboratory (EML) in New York City.[8] The concentrations were 0.02 WL in the EML basement and about 0.01 WL in the building's fifth floor, both with progeny activity ratios of polonium-218, lead-214, and bismuth-214 of about 1:0.5:0.3. For 21 New York and New Jersey houses, the mean annual-average PAEC for progeny of radon-222 was about 0.004 WL in the living space, with a range of values from one house to another of 0.002-0.013 WL; equilibrium factors averaged slightly above 0.6 in the living space.[8]

Measurements in Florida houses built on reclaimed phosphate land yielded average radon-progeny concentrations of about 0.01 WL, but the range extended to above 0.05 WL.[13] Houses in Grand Junction, Colorado, in which remedial action has been recommended, had PAECs ranging from 0.02 to 1 WL. Sets of control houses monitored in Florida and Colorado had an average PAEC similar to that in New York and New Jersey (see Table IV-3). Measurements have also been performed in homes in the vicinity of uranium-mining operations.[1,2]

A few measurements of individual radon-progeny concentrations have been made, often to correlate such concentrations with possible removal processes.[17,36] These processes constitute potential control techniques.

Some work has been done on characterizing the particle size distribution of indoor radon progeny, as well as the dependence of concentrations and distributions on various characteristics, including location, particulate mass concentration, air-exchange rate, and air-mixing rate. The fraction of radon progeny that is unattached to particles, as well as the size distribution of attached progeny, was measured at the EML building and in homes.[8,9] Such measurements have also been performed in uranium mines. The diffusion coefficients of radon progeny have been measured,[41] and their interactions with particles have been examined theoretically.[50]

Lawrence Berkeley Laboratory has performed a few measurements of radon progeny in solar homes in New Mexico and found PAECs of about 0.005 WL (J.G. Ingersoll, personal communication).

The simplest models of indoor radon and radon-progeny concentrations use a set of simple equations connecting the indoor radon source strength, outdoor concentrations, and rate of air exchange, assumed to be the only removal mechanism (other than radioactive decay). An example is a computer program of Kusuda,[28] which permits step variations in air-exchange rate. Models may also simulate diffusion of radon into a house,[33] but transport has not been modeled in any comprehensive way. Models have been made of radon-progeny diffusion and attachment processes[50,51] and of the effect of such processes on progeny concentrations and unattached fractions.[17,20,39] However, no realistic models of radon and progeny

behavior in buildings, by which actual concentrations (or the effect of control measures) might be simulated, has been attempted. More experimental information will evidently be required to develop and validate such models.

Gamma-Radiation Fluxes and Shielding Effects from Building Materials

As discussed above, gamma radiation from terrestrial radionuclides may arise from both building materials and nearby soil and rock, although the radionuclide content of these two sources may vary significantly. Moreover, the structural materials shield occupants both from gamma rays from soil and rock and, to a lesser degree, from cosmic rays. As a result, the building may affect external dose rates of occupants in various ways and degrees. Given information on a particular building, the net effect may be calculated in a way similar to that used by Moeller et al.,[33] based on the gamma-ray dose-rate expressions given above and on estimation of shielding effects.

In some cases, the structure may have little effect on terrestrial or cosmic dose rates. Exclusive use of materials that do not contain substantial radioactivity, such as wood, has the effect of shielding the terrestrial gamma flux (tissue dose, about 35 mrems/yr) by about 20 or 30% and has little effect on the cosmic-ray dose (about 28 mrems/yr). A concrete foundation (slab floor or basement) would have no effect on the cosmic-ray dose and, if its radionuclide content were similar to that of surrounding soil or rock, little effect on the terrestrial dose. That is, although concrete substantially attenuates gamma radiation from the soil or rock, it contributes a gamma-ray flux that compensates for this reduction.

However, if a building also uses concrete in the walls and ceilings and has a radionuclide content similar to that of local soil and rock, an approximate doubling of the terrestrial dose rate would occur. As some compensation, concrete walls and ceilings would tend to shield occupants from cosmic rays in many cases by only about 20%, but by larger fractions for large buildings.

Ordinarily, then, building materials with crustal components whose radionuclide contents are similar to those of local soil and rock may increase external dose rates for occupants by up to tens of millirems per year or may decrease cosmic-ray rates by a somewhat smaller amount. For building materials and surrounding soil or rock that contain higher radionuclide contents, the dose-rate differences between outdoors and indoors would be correspondingly larger.

CONTROL TECHNIQUES

From the few available indoor measurements of radon-222 progeny, it appears that variations of 0.01 WL from one building to another, depending on air-exchange rates and on building or ground materials, are not unusual. The full range of values for conventional houses is considerably larger than this, and measures that reduce the

air-exchange rate can be expected to change it further. A progeny concentration of 0.01 WL, if experienced two-thirds of the time, corresponds to an exposure of about 0.3 WLM/yr--less by about a factor of 10 than the occupational limit of 4 WLM/yr. (Exposure of a person to 1 WL for 170 h, a working month, yields one working-level month, or 1 WLM.) But variations in external dose rate due to ordinary building materials are around 10 mrems/yr, less than one-hundredth of the whole-body occupational dose limit of 5 rems/yr. If these occupational limits correspond to similarly valued risks, it appears that the effect of the structure on radon-progeny exposures (given in working-level months per year) is far more important than the effect on external whole-body dose rates (given in rems per year). Health effects are discussed elsewhere, but this simple comparison indicates one basis for emphasizing methods for controlling radon-progeny exposures. Of these methods, only material substitution may be used for control of gamma-ray dose rates, particularly where materials have unusually high radionuclide contents.

Techniques for controlling indoor concentrations of radon-222 or its progeny include measures that decrease radon sources, reduce transport from sources, remove radon or its progeny from indoor air, or exchange indoor air for outdoor air. The easiest technique to implement in many cases is to increase the air-exchange rate--for example, by opening windows or installing fans. For reasons of comfort or energy efficiency, other methods, sometimes equally straightforward, may often be preferable. In general, not enough is known about the cost, effectiveness, and applicability of various measures for a judgment of their importance in the general building stock.

Material Selection or Site Preparation

In construction of a building, the use of materials whose radon-222 emanation rates are low affects the source strength directly. However, in situations where the surrounding soil and rock contribute most of the radon, opportunities for controlling the source strength are limited, especially because the diffusion length of radon-222 is relatively large and radon source strength is not often a criterion for site selection. Attention to building materials or site materials (underlying and surrounding soil) in new construction has a substantial effect in cases where the emanation rate from either of these may be unusually high. Replacing such materials (on a remedial basis) is often difficult or expensive, so other measures may be favored.

Reducing Transport

The principal means of reducing the transport of radon to building interiors are the sealing of materials that have high emanation rates and, for the case of transport from surrounding soil, the plugging of cracks and holes through which air with a high radon-222 content (e.g., soil gas) moves. Materials may be sealed by epoxy resins or other

coatings with up to 90% effectiveness.[3,6] Sealing surfaces, filling holes with impervious materials, and stopping transport by installing plastic or other barriers have proved effective in some cases that required remedial action (see, for example, Atomic Energy Control Board[1]), but they all require integrity of the barrier for long-term reduction of transport. The general applicability or effectiveness of these measures as long-term passive controls is not known. It should be noted that confinement of radon by diffusion or convection barriers also permits buildup of radon and its progeny behind the barrier, causing an increase in gamma radiation from building materials. Nevertheless, this increase appears less important than the associated decrease in airborne radon-222 and its progeny.[7] Transport may also be reduced by ventilating crawlspaces or basements or (in new construction) by designing transport routes that bypass slab floors or basements.

Removal of Progeny from Indoor Air

Methods for removing radon-222 progeny from indoor air include filtration with fiber, electrostatic, or charcoal filters; mixing of indoor air to cause deposition within the structure or ventilation system; and space-charging to remove progeny ions. Filtration systems are effective in reducing airborne particulate mass concentrations. However, depending on the system, they may thereby raise the concentration of unattached progeny ions, especially polonium-218;[13] for some particle size distributions, this would raise the ratio of lung dose to PAEC. Nazaroff[11] observed a substantial decrease in PAEC from operation of the furnace fan (which thereby activated the system's filter), but the unattached fraction was not measured. Holub et al.[17] and Jonassen[23] have performed related experiments on air-mixing, ventilation, and filtration. Finally, in many measurement techniques, charged radon-222 progeny are collected by voltage differentials, but it does not appear that this principle can easily be applied as a control measure.

Exchange of Indoor and Outdoor Air

Use of air-to-air heat exchangers to remove indoor air while conserving potentially lost energy is being investigated by Lawrence Berkeley Laboratory. Preliminary results[36] indicate that this method is effective, in at least one configuration, in reducing radon-222 and progeny concentrations. This method is particularly attractive because it can be applied in both new and existing buildings and because it is effective in reducing concentrations of other indoor contaminants.

RESEARCH NEEDS

Substantial research efforts are needed in three subjects: the characterization of radon sources and of the indoor concentrations and

behavior of radon and its progeny, the development and testing of control techniques, and the modeling of radon and its progeny in structures. These efforts need to be supported by development of measurement instrumentation, followed by an evaluation of indoor concentrations, control measures, and building energy-conservation measures, among other factors. In addition, evaluative efforts will require further work on the health effects of radon, which have not been discussed here.

Programs to characterize building materials by radon emanation rate or radionuclide content should be more widespread and complete. It is even more important to survey soil and groundwater with respect to radionuclide content, radon emanation, and radon transport. A rapid effort should be undertaken to determine the feasibility of geologic or geographic characterization of soil. As part of efforts to characterize materials, attention should be given to the effects of moisture, pressure, and temperature. Community water supplies should also be surveyed.

Studies of indoor radon and progeny concentrations should be undertaken with two major purposes: to learn the range and distribution of radon and its progeny in the building stock, and to understand the behavior of radon and its progeny in buildings. The first purpose requires surveys of many buildings of a variety of types and in various geographic areas. These surveys may be implemented by associating them with other large-scale efforts, such as those for energy-conservation retrofits or for insurance purposes. They may measure either radon concentrations or potential alpha-energy concentrations (PAECs), but the former may be measured more easily and may in fact be preferable, in that an adequate understanding of progeny behavior could be used to infer PAECs in a way that lends itself to generalization.

This interpretative basis must be developed through intensive measurements to characterize indoor radon and progeny behavior. Such intensive work at only a few sites would serve as a basis not only for improving measurement techniques but also for developing control techniques. Particular attention must be given to progeny-particle interactions and removal processes. Results of intensive investigations would be validated by less-detailed field measurements at a larger number of sites. Ultimately, the results would serve as a basis for estimates of health effects.

Many measurement programs will have to be supported by instrumentation development. More convenient portable instruments for field source measurements based on alpha-scintillation techniques or on sodium iodide gamma-ray detectors could be developed. Further work on integrating devices for large-scale surveys of indoor concentrations is warranted, as is development of simple and quick progeny monitors, presumably based on semiconductor detectors. For intensive investigation of progeny behavior at a few sites, more versatile special-purpose systems must be designed to measure infiltration rate, radon, individual radon progeny, particle concentrations, and environmental conditions automatically.

Substantial efforts to develop and study control techniques are required. The effects of techniques to clean the air (rather than control the source) would have to be studied in the manner indicated above for detailed investigations of progeny behavior.

These measurement programs must be accompanied by corresponding modeling efforts. Models that characterize sources (on a geologic and geographic basis) and transport (by site and building type) are needed. Although models for physical processes involving radon progeny have begun to be developed, much more work is needed, especially for understanding progeny-particle interactions and control techniques. Models of indoor-air quality that use the source and progeny models appropriately could then be developed. Finally, the models of indoor-air quality could be combined with models of the building stock to represent current radon and progeny concentrations and the effects of changes in building design and of potential control measures. Models of indoor-air quality and the building stock will be necessary for any indoor air pollutant and for evaluation of potential strategies for controlling indoor-air quality.

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FORMALDEHYDE AND OTHER ORGANIC SUBSTANCES

The infiltration of outdoor air is one source of formaldehyde and other organic substances in the indoor environment, but the primary

sources are in the indoor environment itself--building materials, combustion appliances, tobacco smoke, and a large variety of consumer products. A buildup of formaldehyde may be exacerbated in buildings that have been subjected to energy-efficiency measures intended to reduce infiltration and, thus, energy consumption. Emission rates for formaldehyde and other organic pollutants emitted in the indoor environment are generally unknown. Analytical techniques have been applied mostly in measuring concentrations in indoor air. Very little work has been done in measuring specific source strengths or ranking emission sources, except on a broad relative basis.

FORMALDEHYDE

In general, indoor formaldehyde concentrations exceed those outdoors. The contribution of formaldehyde in outdoor air to indoor air appears to be minor.

A recent National Research Council report deals in great detail with the sources and effects of formaldehyde and other aldehydes.[18] The reader is referred to that report for a more comprehensive treatment than is feasible here.

Sources and Emission Rates

Insulation. Urea-formaldehyde (UF) foam is used as thermal insulation in the side walls of existing buildings, especially single-family residential buildings. UF foam is a convenient substance for retrofitting existing buildings, because it is injected directly into wall cavities through small holes that are then sealed up.

UF foams were developed in 1933 and first used as an insulating material in the 1960s. UF foam has been used for thermal insulation in Europe for many years, but is relatively new in the United States. In the early 1970s, interest in and use of this material increased as the cost of energy mounted and resulted in increased demand for residential insulation. A production peak occurred in 1977, when the demand for insulating products created shortages of other insulating materials, such as cellulose and fiberglass.[19] Approximately 170,000 houses were insulated in 1977. The need for efficient thermal insulation in housing has increased dramatically in the last few years. About 150,000 houses a year are now being insulated with UF foam. Industry representatives reportedly believe that more than 200,000 homes will be insulated with UF foam during 1980.[19]

Installation involves mixing partially polymerized UF resin with a surfactant (foaming agent) and an acid catalyst under pressure that forces air into the mixture to create a foam. The foam hardens within minutes and cures and dries completely within a few days. Building codes in the United States, concerned with the fire-safety aspects of UF-foam insulation, rate it as a combustible material. The codes require that UF foam, when used on the inside of buildings, must be protected by a thermal barrier of fire-resistant material. In England

and Holland, UF insulation materials are certified for use only in masonry cavities of buildings.

If the ingredients of UF-foam insulation are improperly formulated or mixed, formaldehyde may be released into the building. Long et al.[12] enumerated some of the factors that affect the release of formaldehyde from UF foam:

- Excessive formaldehyde in the resin-concentrate solution.
- Excessive acid catalyst in the foaming agent.
- Excess foaming agent (surfactant).
- Foaming during periods of high humidity and high temperature.
- Foaming with cold chemicals (optimal temperature, 50-80°F).
- Improper use of vapor barriers.
- Improper use of foams (in ceilings, etc.).

Owing to the diversity of time factors and the complexity of their interrelationships, the quantity and rate of formaldehyde relesase from a house insulated with UF foam is difficult to predict.

Particleboard and Plywood. The superior bonding properties and low cost of formaldehyde polymers make them the resins of choice for the production of building materials, especially plywood and particleboard. Among the various formaldehyde resins used in building materials--urea-formaldehyde, phenol-formaldehyde, and melamine-formaldehyde--urea-formaldehyde resin is the most common adhesive used in indoor plywood and particleboard. Plywood is composed of several thin sheets of wood glued together with UF resin. Particleboard is made by saturating small wood shavings with UF resin and pressing the resulting mixture, usually at high temperature, into the final form. Particleboard can emit formaldehyde continuously for a long time (several months, or even years). In buildings in which these wood products are used for partition walls or furniture, formaldehyde may reach concentrations sufficient to cause eye and upper respiratory irritation. In cases of extensive use of these products where air-exchange rates are low, the concentration can reach 1 ppm or more. This is due in part to the high surface-to-volume ratio of particleboard and plywood used as building materials. The emission rate depends on a number of factors--the original manufacturing process, quality control of fabrication, porosity, ambient temperature, humidity, cutting of the board for final use, etc.

UF resins contain some free formaldehyde; in addition, the resin may hydrolyze and release free formaldehyde at high temperature and humidity. The phenol-formaldehyde resins used for wood products that require greater moisture resistance (i.e., exterior plywood) do not release formaldehyde as readily as products bound with UF resin. Phenol-formaldehyde resins are not generally used for indoor wood products, because of their higher cost.

Combustion Appliances. Recent studies have reported on combustion-generated indoor air pollutants, specifically contaminants from gas stoves and heating systems in residential buildings.

Laboratory studies have shown that gas stoves emit substantial quantities of aldehydes and that formaldehyde is the major component of the aldehydes measured (Schmidt and Götz;[17] G. W. Traynor, Lawrence Berkeley Laboratory, personal communication). Reported formaldehyde emission from a single gas stove under test conditions has been measured as approximately 15,000 and 25,000 μg/h for each top burner and the oven, respectively (Traynor, personal communication).

Tobacco Smoke. Tobacco smoke is a source of several chemicals, including several aldehydes other than formaldehyde. It may contribute formaldehyde to the indoor environment. The smoker's exposure to these chemicals results principally from smoke inhaled directly into the lungs (mainstream smoke). The smoke that is not directly inhaled into the lungs enters the space surrounding the smoke (sidestream smoke). It is the sidestream smoke that is the major contributor to indoor pollution.

Analysis by Osborne et al.[16] indicated that acrolein was an important component of tobacco smoke; this finding was confirmed by Jermini et al.,[3] whose studies were conducted on a smoking machine in an environmental chamber. Formaldehyde and acetaldehyde have also been identified in cigarette smoke.

Harke et al.[7,8] measured concentrations of nicotine, carbon monoxide, acrolein, and other aldehydes (expressed as acetaldehyde) in the air of an unventilated room in which a series of experiments with a smoking machine had been performed. Substantial concentrations of all four compounds were observed with this extremely low ventilation.

Other Sources. Several products that are potential sources of formaldehyde emission are mentioned below. Because there is no information on the rates or quantities of such emission, it is not known which are important sources.

UF resins are used in the paper industry to increase the wet strength of various grades of paper. Paper products typically treated with UF resins are grocery bags, wax paper, facial tissues, napkins, paper towels, and disposable sanitary products.

Formaldehyde polymers are used extensively in the manufacture of floor coverings and as carpet backing. UF resins are used in the textile industry as binders for pigments, fire retardants, or other materials to cloth and to impart stiffness, wrinkle resistance, and water repellency to fabrics.

Fertilizers and pesticides used for commercially grown plants also may use aldehydes and, theoretically, could contribute to the aldehyde content of ambient air locally.

Urea-formaldehyde fertilizers are used not only to obtain a more uniform release rate than is possible with soluble sources of nitrogen but also to minimize the hazards of water pollution by nitrates leached out of the soil. These compounds have been used with field crops, turfgrass, pine seedlings, and geranium. The extent of their indoor use and the amounts and rates of formaldehyde release are unknown.

Formaldehyde is used in numerous places, such as biologic laboratories, hospitals, and hobby and craft areas.

Concentrations

Indoor monitoring data for U.S. homes are few; there are limited monitoring data for European homes, particularly in the northern European countries, and they show higher indoor formaldehyde concentrations than in the United States. Table IV-4 summarizes recent monitoring data. Most of the measurements of organic substances in the indoor environment have been made on aldehydes--specifically on formaldehyde. Studies of the indoor-outdoor relationships of formaldehyde show that indoor concentrations generally exceed outdoor.

There have recently been several studies measuring indoor formaldehyde concentrations in which emission was from particleboard and plywood furnishings and UF-foam insulation in houses. Measurements in Denmark,[2] Sweden (J. Sundell, personal communication; T. Lindvall, personal communication), West Germany (B. Seifert, personal communication; M. Deimel, personal communication), and the United States (P.A. Breysse, personal communication) have shown that indoor concentrations often exceed 0.1 ppm and may even exceed the then-established 8-h time-weighted average safe exposure limit (3 ppm) for workroom air.[23] In the 23 Danish houses, the average formaldehyde concentration (the predominant source was particleboard) was 0.62 mg/m^3 (about 0.5 ppm), and the range was 0.08-2.24 mg/m^3 (about 0.07-1.9 ppm).[2]

As a result of occupants' complaints, formaldehyde was measured in more than 200 mobile homes in the United States; the concentrations reported ranged from 0.03 to 2.4 ppm (about 0.04-2.9 mg/m^3) (Breysse, University of Washington, personal communication). A study of formaldehyde concentrations in new office trailers with air-exchange rates as low as 0.16/h found formaldehyde concentrations of 0.15-0.20 ppm,[5] in contrast with outdoor concentrations of less than 0.01 ppm.

Formaldehyde concentrations build up in mobile homes, not only because of emission from some building materials used in their construction, but also because mobile homes are often more tightly constructed than conventional homes, thus decreasing ventilation.

Aldehydes (measured by the MBTH method) were monitored in a study of 19 homes across the United States.[10] Indoor concentrations of aldehydes were always higher than outdoor concentrations, typically by a factor of 6 and quite often by a factor of about 10. Figure IV-2 shows the data collected in this study from a gas-cooking home with one smoker. Although the source strengths were not determined in this study, the highest concentrations were observed in the two mobile homes; in general, the plywood and particleboard appeared to be the primary sources.

In a more recent study, formaldehyde and total aliphatic aldehydes (formaldehyde plus other aliphatic aldehydes) were measured at several energy-efficient research houses at various geographic locations in the United States.[11] Results showed that, at low infiltration rates (<0.3 air change per hour, or ach), indoor formaldehyde concentrations often exceeded 0.1 ppm, whereas outdoor concentrations typically remained at 0.016 ppm (20 μg/m^3) or less. Typical air-exchange rates for single-family residential buildings are between

TABLE IV-4

Summary of Aldehyde Measurements in Nonoccupational Indoor Environments[a]

Sampling Site	Concentration,[b] ppm		Method of Analysis
	Range	Mean	
Danish residences	1.8 (peak)	--	Unspecified
Netherlands residences built without formaldehyde releasing materials	0.08 (peak)	0.03	Unspecified
Residences in Denmark, Netherlands, and Federal Republic of Germany	2.3 (peak)	0.4	Unspecified
Two mobile homes in Pittsburgh, Pa.	0.1-0.8[b]	0.36	BTH bubblers
Sample residence in Pittsburgh, Pa.	0.5 (peak)[b]	0.15	MBTH bubblers
Mobile homes registering complaints in state of Washington	0-1.77	0.1-0.44	Chromotropic acid (single impinger)
Mobile homes registering complaints in Minnesota	0-3.0	0.4	Chromotropic acid (30-min sample)
Mobile homes registering complaints in Wisconsin	0.02-4.2	0.88	Chromotropic acid
Public buildings and energy-efficient homes (occupied and unoccupied)	0-0.21	--	Pararosaniline and chromotropic acid
	0-0.23[b]	--	MBTH bubblers

[a]Reprinted from National Research Council.[15](p. 5-13)

[b]Formaldehyde, unless otherwise indicated.

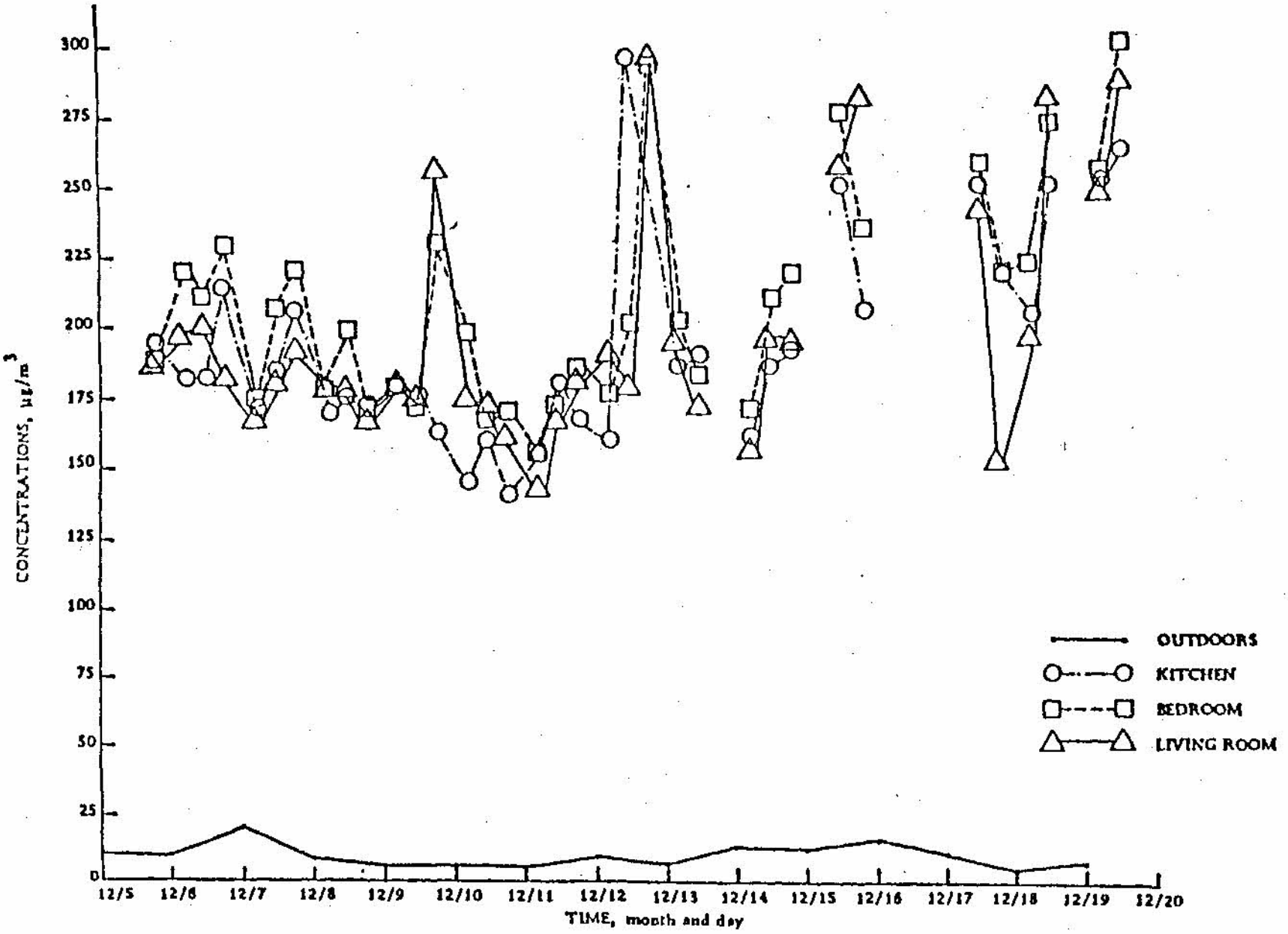


FIGURE IV-2 Diurnal variation in indoor and outdoor aldehyde concentrations at single-family house in Chicago. Reprinted from National Research Council.[15](p. 5-14)

0.6 and 1 ach. Figure IV-3 is a histogram showing the frequency of occurrence of formaldehyde and total aliphatic aldehyde concentrations measured at an energy-efficient house with an average air-exchange rate of 0.2 ach. Data taken at an energy-efficient house in Mission Viejo, California, are shown in Table IV-5. As shown, when the house did not contain furniture, formaldehyde content was 80 $\mu g/m^3$; when furniture was added, formaldehyde rose almost threefold. A further increase was noted when the house was occupied, very likely because of such activities as gas cooking. When occupants opened windows to increase ventilation, the formaldehyde content dropped substantially. Although high, aldehyde contents observed in the majority of the energy-efficient dwellings monitored were generally below 200 $\mu g/m^3$.

Indoor and outdoor formaldehyde and aldehyde concentrations were found to be about the same at a public school in Columbus, Ohio, and a large medical center in Long Beach, California, and were well below 0.1 ppm (120 $\mu g/m^3$). Both buildings have high ventilation rates; this probably explains the low indoor concentrations, essentially equivalent to outdoor concentrations.

Because many of the data reported from these field-monitoring studies involved houses whose occupants had complained of indoor air quality, these findings may not be representative of all homes. However, when data from a random sample in Wisconsin are compared with those from the Washington mobile-home sample, most of the differences in aldehyde concentrations can be explained by differences in the age of the homes. The mobile homes in the complaint sample are much newer than those in the random sample. Tabershaw Associates[21] analyzed the complaint data for mobile homes in Washington, and found that there was no statistically valid relationship between the severity of symptoms reported by occupants and the concentration of formaldehyde and that, regardless of the actual exposure concentration, all persons in the mobile homes reacted in substantially the same manner.

Foreign (particularly Danish and Swedish) houses monitored for formaldehyde appear to have much higher concentrations than U.S. houses. These findings probably represent differences in construction and, hence, cannot be considered as representative of U.S. houses.

Andersen et al.[2] formulated a mathematical model that estimates the indoor air concentration of formaldehyde. Although use of Danish studies may not be appropriate for U.S. houses, the treatment of Andersen et al. illustrates the many variables that must be considered. By conducting climate-chamber experiments, Andersen et al. found the equilibrium concentration of formaldehyde from particleboard to be related to temperature, water-vapor concentration in the air, ventilation, and the amount of particleboard present. From this work, a mathematical model was established for room-air concentration of formaldehyde.

When this mathematical formulation was applied to the room-sampling results, a correlation coefficient of 0.33 was found between the observed and predicted concentrations--not a particularly good predictive ability. The authors then modified the adjustable constants by calculating them for each room on the basis of monitoring results. The modified values led to a correlation coefficient of 0.88--a considerable improvement in predictive ability.

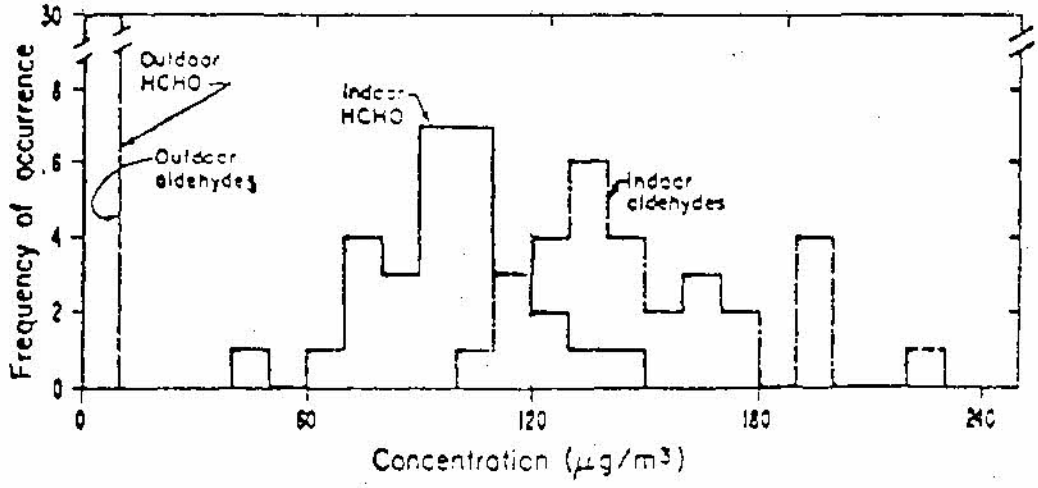


FIGURE IV-3 Indoor and outdoor formaldehyde and other aldehyde concentrations at single-family house in Maryland. Histogram showing the frequency of occurrence of formaldehyde and total aliphatic aldehydes at an energy-efficient house with 0.2 ach. Reprinted from National Research Council.[15(p. 5-17)]

TABLE IV-5

Indoor and Outdoor Formaldehyde and Aliphatic Aldehyde Concentrations Measured at a Single-Family House (California)[a]

Condition	Number of Measurements	Sampling Time	Formaldehyde ($\mu g/m^3$)[b]	Aliphatic Aldehydes ($\mu g/m^3$)[c]
Unoccupied, without furniture	3	12	80 ± 9%	90 ± 16%
Unoccupied, with furniture	3	24	223 ± 7%	294 ± 4%
Occupied, day[d]	9	12	261 ± 10%	277 ± 15%
Occupied, night[e]	9	12	140 ± 31%	178 ± 29%

[a]Reprinted from National Research Council.[15](p. 5-18)

[b]Determined using pargrosaniline method (120 $\mu g/m^3 \approx$ 100 ppb). All outside concentrations <10 $\mu g/m^3$.

[c]Determined using MBTH method, expressed as equivalents of formaldehyde. All outside concentrations <20 $\mu g/m^3$.

[d]Air exchange rate = 0.4 ach.

[e]Windows open part of time; air exchange rate significantly greater than 0.4 ach and variable.

Formaldehyde release from interior particleboard occurs at a decreasing rate with an increase in product age. Eventually the rate of formaldehyde evolution decreases to an imperceptible point. The time necessary for this phenomenon to occur (perhaps several years) depends on the atmospheric conditions to which the board has been subjected, as well as the degree of cure of the resin. The more unstable groups degrade first, followed by the more stable free methylol groups.

A 1977 field study in which field tests and a mathematical model were used to determine the half-life of formaldehyde in particleboard typically used in Scandinavian home construction reported it to be about 2 yr when the ventilation rate in the home is 0.3 ach (C.D. Hollowell, personal communication). Suta has analyzed the effect of home age on formaldehyde concentrations in Danish houses. These data give the following relationship of formaldehyde concentrations as a function of house age when no corrections are made for other pertinent factors, such as the amount of particleboard in the home, temperature, humidity, and ventilation: $C = 0.50e^{-0.012A}$, where C = formaldehyde concentration (ppm) and A = home age (mo). On the basis of this formula, the half-life of formaldehyde concentration is 58 mo. The difference between half-life values derived from test data and those from house-monitoring may result partly from the fact that particleboard is often added to older homes for repair and improvement.

Monitoring data for the 65 randomly selected mobile homes in Wisconsin show a similar decrease in formaldehyde concentration with increasing home age (M. Woodbury, personal communication). In these studies, the reported formaldehyde half-life was 69 mo, which is quite similar to that found in the Denmark study. Monitoring data on 45 complaint mobile homes in Wisconsin also showed a decrease in formaldehyde concentration with increase in home age; the indicated half-life in this sample was 28 mo. When these data are combined, the formaldehyde half-life becomes 53 mo, or approximately 4.4 yr.

Residences are not all expected to have the same formaldehyde concentration. As suggested earlier, there is variation even among homes of the same age, depending on the amount and type of particleboard, plywood, and UF-foam insulation used in the construction, as well as on temperature, humidity, and ventilation. Therefore, monitored concentrations from a sample of similar homes will be characterized by a frequency distribution that can be approximated by a known statistical distribution, which, in turn, can be used to estimate the range of human exposures to formaldehyde in the residential environment.

The average atmospheric formaldehyde concentration appears to be approximately 0.2 ppm in both mobile homes and homes insulated with UF foam. Little information is available on conventional houses that do not contain UF-foam insulation or that were not designed to be energy-efficient. The average formaldehyde concentration in conventional houses appears to be 0.01-0.1 ppm and may be only slightly higher than outdoor concentrations. Houses containing more

particleboard would fall on the high side of this concentration range, and houses with no particleboard would fall on the low side.

Outdoor atmospheric formaldehyde concentrations are generally much lower than 0.1 ppm in U.S. cities. Examples of annual average concentrations are 0.05 ppm for Los Angeles,[1,20,22] 0.004-0.007 ppm for four New Jersey cities,[4] 0.04 ppm for Wisconsin cities (Hanrahan, personal communication), and less than 0.03 ppm in Raleigh, North Carolina, and Pasadena, California.[6] Formaldehyde concentrations at four Swiss locations ranged from 0.007 to 0.014 ppm; these concentrations are about one-fifth the corresponding indoor Swiss concentrations.[25] A mean value of 0.004 ppm has been reported for mainland Europe.[3]

Thus, formaldehyde concentrations in mobile homes and for homes insulated with UF-foam resin are considerably higher than those of the corresponding outdoor atmosphere.

Control Techniques

Several measures have been used in attempts to correct problems associated with formaldehyde release from building materials, including:

- Ventilation (opening doors and windows).
- Mechanical ventilation coupled with the use of heat exchangers.
- The use of impregnated charcoal in furnace or air-conditioning filters.
- The evaporation of household ammonia in closed and overheated rooms to neutralize formaldehyde, followed by ventilation.
- Injection of ammonia into insulation through holes in walls to neutralize formaldehyde.
- Spraying of air filters or floors with a specified odor absorbent provided by the manufacturer.
- Use of a "masking agent" available from the manufacturer.
- Application of vinyl wallpaper or a low-permeability paint to interior walls.
- Removal of all or some of the insulation from the home.

Although many manufacturers claimed that they had successfully used these remedial measures, no studies have shown that the measures will reliably control the release of formaldehyde gas.

OTHER ORGANIC SUBSTANCES

In addition to formaldehyde, many other organic contaminants can be present in indoor environments. Very little work has been done to identify or measure organic contaminants that may be harmful. Nevertheless, these compounds may provide a partial explanation for complaints registered by people in indoor environments where it is determined that formaldehyde and other indoor-pollutant concentrations are low or undetectable.[13]

The experimental tasks associated with characterizing organic contaminants in indoor environments are formidable. The contaminants are usually present as complex mixtures of many compounds at low concentrations. Enough work has been done to outline broadly the nature of the problem and the available information.

Sources and Emission Rates

Four major sources of organic contaminants can be identified. People emit one category of organic contaminants termed "bioeffluents." Building materials can also emit organic contaminants. The other two categories, personal consumer products (including insecticides, pesticides, and herbicides) and tobacco-smoking, are discussed later in this chapter.

Bioeffluents. Humans, through normal biologic processes, emit a category of organic substances known as "bioeffluents." Wang[24] studied bioeffluents in a school auditorium seating over 400 people. Many organic contaminants were observed, but the major ones associated with people were methanol, ethanol, acetone, and butyric acid. As an example, the absolute concentrations and the emission rate per person during a class lecture are shown in Table IV-6. Emission rates of bioeffluents increased sharply during a class examination, considered by Wang to be a period of stress. The findings of Wang were corroborated in part by Johansson,[10] who studied two schoolrooms in Sweden. He observed that acetone and ethanol were associated with the presence of schoolchildren. No emission rates were reported.

Building Materials. Such products as adhesives, paints, and sealants contain solvents and other agents that can be released during and immediately after application. The health hazards associated with these short-term releases of organic contaminants are acknowledged in the warning labels regarding the use of adequate ventilation, which are commonly applied to these products.

Less well understood is the potential for long-term emission of organic contaminants from building materials. Slow release of residual solvents and other agents (e.g., catalysts, surfactants, and plastic monomers) is one possibility, as is the gradual production of contaminants by degradation (e.g., air oxidation, photoinitiated reactions, and retropolymerization reactions).

Preliminary data indicate that concentrations of organic compounds in new buildings are generally higher than outdoor concentrations.[13,18] Figure IV-4 shows comparative gas chromatograms of an indoor air sample and an outdoor air sample taken simultaneously at the same building. Classes of compounds consistently observed in indoor air include hydrocarbons, alkylated aromatic compounds, and chlorinated hydrocarbon solvents. Table IV-7 lists organic compounds identified in several office buildings at concentrations at least 5 times greater than outdoor concentrations.[18]

TABLE IV-6

Average Concentrations and Emission Rates of Organic Bioeffluents in a Lecture Class (389 People at 9:30 a.m.)

Bioeffluent	Concentration, ppb	Emission Rate, mg/day per person
Acetone	20.6 ± 2.8	50.7 ± 27.3
Acetaldehyde	4.2 ± 2.1	6.2 ± 4.5
Acetic acid	9.9 ± 1.1	3.6 ± 3.6
Allyl alcohol	1.7 ± 1.7	19.9 ± 2.3
Amyl alcohol	7.6 ± 7.2	21.9 ± 20.8
Butyric acid	15.1 ± 7.3	44.6 ± 21.5
Diethylketone	5.7 ± 5.0	20.8 ± 11.4
Ethyl acetate	8.6 ± 2.6	25.4 ± 4.8
Ethyl alcohol	22.8 ± 10.0	44.7 ± 21.5
Methyl alcohol	54.8 ± 29.3	74.4 ± 5.0
Phenol	4.6 ± 1.9	9.5 ± 1.5
Toluene	1.8 ± 1.7	7.4 ± 4.9

[a]Data from Wang.[24]

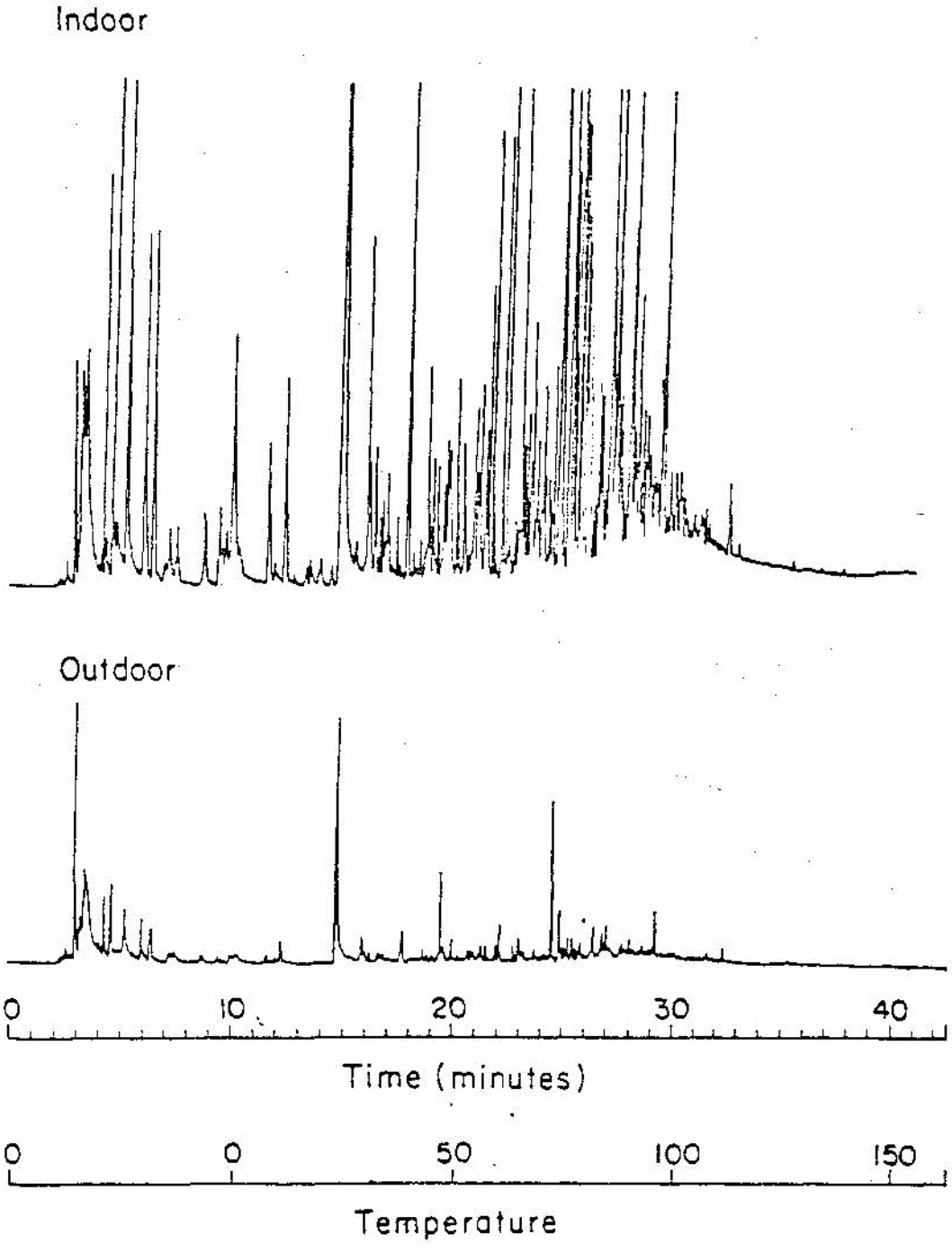


FIGURE IV-4 Comparison of chromatograms of samples taken inside and outside an office for trace organic substances. From C. D. Hollowell, personal communication.

TABLE IV-7

Organic Substances Detected in Offices[a]

Substance	OSHA Permissible Exposure Limit, ppm
Hydrocarbons:	
n-Hexane	500
n-Heptane	500
n-Octane	500
n-Nonane	--
n-Undecane	--
2-Methylpentane	--
3-Methylpentane	--
2,5-Dimethylheptane	--
Methylcyclopentane	--
Ethylcyclohexane	--
Methylcyclohexane	500
Pentamethylheptane	--
Aromatics:	
Benzene	1
Xylenes	100
Toluene	200
Halogenated hydrocarbons:	
Trichloroethane	350
Trichloroethylene	100
Tetrachloroethylene	100
Miscellaneous:	
Hexanal	--
Methylethylketone	200

[a]Data from Schmidt et al.[18]

In general, concentratons of specific organic compounds in nonindustrial indoor environments are well below occupational exposure limits established by OSHA. The health hazard from the effects of organic compounds at concentrations observed in indoor environments cannot now be assessed. It is important to note that OSHA criteria were established for the industrial environment, where high exposures to single compounds are encountered. The possibility cannot be overlooked that cumulative exposure to several compounds at low concentrations, or synergistic effects, may explain the complaints of building occupants.

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CONSUMER PRODUCTS

A wide array of activities and products that affect air quality can be found in contemporary residential spaces. The full range is often wider than is found in most occupational or public spaces. We are concerned here with the air-pollution aspects of such activities and consumer products.

Pollutants emitted into the indoor environment by consumer products are usually dissipated by dilution and surface deposition. For a given amount of such a pollutant, the rate of discharge, the volume of the space, the ventilation rate or infiltration rate, and the presence of occupants determine the severity and duration of human exposure. The specifics of control strategies are discussed in Chapter IX, but it will be clear that even an identical activity could produce radically different exposures in different spaces, depending on the application and efficacy of control strategies.

Consumer products introduce pollutants into the indoor air in a number of forms and ways. For example, spray paint and sprayed-on oven cleaners introduce an aerosol of the chemical products whenever they are used. There are many spray products; it has been estimated that the average U.S. residence at any given time contains 45 aerosol sprays.[12] Aerosols can also be produced indirectly, as in grinding, sanding, cleaning, and some hobby activities. Other consumer products and the activities associated with them introduce pollutants by evaporation or sublimation of solvents or active ingredients, such as paint solvents, cleaners, bleaches, and disinfectants. Still other products--e.g., plastics, paints, and textiles with artificial fibers and conditioners--release, or "outgas," small amounts of volatile chemicals over long periods. The association of emission with these types of products or activities is shown in Table IV-8. The table represents a compilation of kinds of emission encountered in a search

TABLE IV-8

Types of Emission of Indoor Air Pollutants Associated with Various Activities and Consumer Products

Activity or Product	Intentional Aerosol Production	Unintentional Aerosol Production	Evaporation or Sublimation	Unintentional Outgassing
Cleaning	X	X	X	X
Painting	X		X	X
Polishing	X		X	X
Stripping		X	X	
Refinishing	X		X	X
Hobbies and crafts	X	X	X	X
Deodorizer	X		X	
Insecticide	X		X	
Disinfectant	X		X	
Personal grooming product	X		X	X
Plastic				X

of the literature and in lists in consumer catalogs; it is clearly incomplete.

AEROSOL-PRODUCING PRODUCTS

Pressurized aerosol cans have found widespread use in a great variety of applications. Aerosol cans typically contain a propellant gas under a relatively low pressure--about 40 psi (3 kg/cm^2)--with a vapor pressure at normal room temperature that allows some of the propellant to be in equilibrium in the liquid phase. Until recently, dichlorodifluoromethane and trichlorofluoromethane (fluorocarbon-12 and fluorocarbon-11) were used widely as propellants, but their effect on the atmospheric ozone layer has led to a prohibition of this application in the United States. Propane, butane, nitrous oxide, methylene chloride, and others are currently used as propellants. Most propellant gases are biologically inactive or active only at high concentrations. Some are extremely flammable and could reach explosive concentrations in enclosed spaces.

Active ingredients vary from product to product, and a complete list of propellants and active ingredients rarely appears on a can. Cleaning agents include sodium or potassium hydroxide in oven-cleaners, ammonium hydroxide in window-cleaners, and tetrachloroethylene and petroleum-derived solvents in spot-cleaners. Spray paints often contain toluene, xylene, methylchloride, and other volatile organic substances, as well as pigments and a vehicle. Dravnieks[16] measured the organic chemicals present in indoor air of a high-rise apartment in which aerosol products had been used. The use of a scented oven-cleaner released at least 13 organic chemicals into the residential space. An unscented aerosol deodorant and a scented aerosol furniture polish released similarly large numbers of chemical species throughout the residence.

Cote *et al.*[7] surveyed the composition of a range of aerosol products for propellants and active ingredients. Mokler *et al.*[23] reported that under "worst reasonable conditions" some aerosol products introduce respirable particles smaller than 6 μm in aerodynamic diameter at a concentration of over 50 mg/m^3--10 times the threshold limit value for daily average exposure in an industrial environment for biologically inactive nuisance dust.[3] The same investigators found that the conditions of discharge did not have a great effect on the size characteristics.[24] There was no evidence of animal toxicologic effects after a series of studies of cosmetic aerosols.[19]

Marier and co-workers[21] exposed 20 human subjects to a number of aerosol products daily for four consecutive weeks. These products included deodorants, hair spray, frying-pan spray, room-freshener, insect repellent, window-cleaner, insecticide, furniture polish, bathroom-cleaner, and a depilatory. All products were used according to the manufacturer's instructions; after 3 wk of exposure, all subjects were evaluated for cardiac function, respiratory function, and hematologic and clinical biochemical characteristics. None of the tests showed evidence of toxic effects, and no fluorocarbon was found

at any time in any blood sample. All the spray products used fluorocarbon as propellant.

In an epidemiologic study of 3,800 people in Tucson, Arizona, Lebowitz reported in 1976 that he found respiratory effects associated with frequent use of aerosols.[17] In a followup study 5 yr after the first one, he found that use of aerosol products was lower by a factor of 10, and the association between aerosol use and respiratory effects was no longer seen.[18]

Stewart et al.[32] evaluated physiologic responses to different propellant gases (isobutane, propane, fluorocarbon-12, and fluorocarbon-11). They were inhaled at concentrations of 250, 500, and 1,000 ppm for periods of 1 min to 8 h. None of the subjects showed a decrease in pulmonary function or a change in cardiac rhythm as a result of these exposures.

Deliberate inhalation of some aerosol sprays often leads to serious consequences, or even death. Lipid sprays[10] have been shown under such circumstances to lead to acute functional and anatomic disintegration of alveolar surfactant. The resulting alveolar collapse can cause fatal hypoxemia. In one preliminary study,[29] the statistical association found between the indoor use of spray adhesives and congenital malformations was sufficient to warrant further study.

PARTICLES PRODUCED AS A BYPRODUCT

The major production of particles in the indoor environment is undoubtedly due to tobacco-smoking and food-preparation (which are considered elsewhere, as is malfunctioning heating equipment). In the course of cleaning of floors and furniture, there is likely to be a resuspension of dust particles that had previously settled. Maintenance and cleaning activities have been identified as a source of asbestos fibers in spaces that contain accessible asbestos. Resuspension of settled dust in the absence of ventilation or air-cleaning effectively increases the exposure of occupants.[26]

Even minimal exposures to asbestos fibers in hobby materials[6] have been found to result in identifiable asbestos bodies in the lung, and the asbestos fibers found in mesothelioma have been ascribed to minimal exposures in the pursuit of hobbies.[5]

Hobby and home craft activities can cause substantial production of particles.[22] The use of lead glazes by potters[14] can lead to high lead intakes, and an unknown fraction of this intake will be from inhalation. The use of solder and flux in stained-glass fabrication and jewelry-making results in the aerosolization of lead, cadmium, and flux.[15] The effects of inhalation of lead and cadmium are known better from occupational exposures, but some residential exposure must be expected.

Some phases of woodworking result in the production of airborne wood dust from sawing, grinding, and sanding. Acheson and co-workers studied the risk of nasal cancer in woodworkers in the furniture industry[1] and concluded that exposure to wood dust, rather than varnish or polish, caused an increased risk of nasal cancer. For

occupational exposures, the risk of nasal cancer was as much as 500 times the normal incidence.

PRODUCTS AND ACTIVITIES ASSOCIATED WITH EVAPORATION OR SUBLIMATION

Solvents are used in the indoor environment for a variety of purposes--in clothing- and furniture-cleaners; as carriers for polishes, paints, and varnishes; and as chemical strippers in the refinishing of furniture--and they are inherent in many adhesives, personal grooming products, disinfectants, and deodorizers. Most of the chemical species involved are used in the occupational environment, and threshold limit values have been adopted for that environment.[2,3] Table IV-9 contains a nonexhaustive list of substances that evaporate or sublimate from solvents and household products and their occupational exposure limits.[3]

The containers of many of the products containing substances listed in Table IV-9 do not list their constituents or concentrations, although hazard warnings are generally provided. The indoor air concentrations are rarely measured; even when they are, there is disagreement concerning the typical conditions. An example of this is provided by two reports on methylene chloride. The first deals with exposures to methylene chloride in paint-removers in home workshops.[31] One person developed acute myocardial infarction in three separate episodes after the use of methylene chloride for furniture-stripping in a basement workshop. The last exposure was fatal. In the case of a healthy younger man, it was accidentally discovered that he had a high carboxyhemoglobin (COHb) concentration (6-8%) on each of several mornings after 2-h exposures to paint-removers at home on the previous evening. Controlled exposures of volunteers to methylene chloride at 50, 250, and 986 ppm for 3 h produced COHb at concentrations of 2%, 4.5%, and 15%, respectively, measured 1 h after the end of exposure. In the second study,[30] volunteers were exposed to methylene chloride at 450 ppm for 26 min; the authors considered that a typical exposure for the use of spray paint with methylene chloride as propellant. They found "a clinically insignificant increase" in COHb 7 h after exposure. These two reports represent a range of reactions to methylene chloride--from nearly undetectable to accidentally detected to fatal--that depends on individual vulnerability, conditions of use, and criteria used for detection. Measurements of actual concentrations of chemicals associated with household products are scarce; where they are reported, they can be very high, as in a report of benzene at 130 ppm found in a double garage during furniture-stripping.[39]

The use of mercury compounds as fungicides in latex paints gives rise to long-term emission of mercury into the indoor spaces in which such paints have been applied. Taylor[35] used a radioactive tracer and found that 20-25% of the mercury was lost in the first 90 d after application. Foote[11] measured background ambient atmospheric mercury concentrations in a number of homes at slightly over 2 ng/m^3 and in rooms recently painted with latex paint at up to 3,000 ng/m^3. In

TABLE IV-9

Threshold Limit Values of Various Substances[a]

Chemical	TLV-TWA,[b] mg/m^3	TLV-STEL,[c] mg/m^3	Source
Acetone	2,400	3,000	Lacquer solvent
Ammonia	18	27	Cleaner
Benzene	30	--	Adhesive, spot cleaner, paint remover
Carbon tetrachloride	65	130	Spot cleaner, dry cleaner
Chlorine	3	9	Cleaner
Methanol	260	310	Paint, spot cleaner
Trichloroethane	1,900	2,380	Cleaning fluid
Methylene chloride	700	870	Paint remover
Ozone	0.2	0.6	Copying machine, electrostatic air cleaner
Trichloroethylene	535	800	Dry-cleaning agent
Turpentine	560	840	Paint, finish
Xylene	435	655	Solvent, paint carrier, shoe dye
Toluene	375	560	Solvent, paint carrier, dry cleaning

[a]Data from American Conference of Governmental Industrial Hygienists.[3]

[b]Threshold limit value--time-weighted average.

[c]Threshold limit value--short-term exposure limit.

rooms painted 3 yr before, he found concentrations of 68 ng/m^3. The threshold limit value adopted by the American Conference of Governmental Industrial Hygienists[3] for 8-h workdays is 50,000 ng/m^3. Sibbett et al.[28] found room-air concentrations of around 1,000-2,000 ng/m^3 in rooms with fresh latex paint--also well below the TLV and not likely to produce clinical symptoms. In comparison, spilt mercury from broken clinical thermometers[11] produced mercury-vapor concentrations in room air of about 5 µg/m^3, continuous exposure to which might produce clinical symptoms.

Residential or commercial use of insecticides, pesticides, and herbicides both inside and outside has the potential for contaminating the indoor environment. Some organochlorinated or organophosphated pesticides have specific agricultural applications, but others are used widely in urban areas by both private property-owners and municipalities. The organic-based compounds can find their way indoors by various routes. Some are applied directly indoors for rodent and insect control. The long-term effectiveness of many of these compounds is achieved by prolonged sublimation. Various studies have found many of these compounds indoors.[8 9 13 16] In spite of the widespread use of residential and commercial insecticides, pesticides, and herbicides, no systematic survey has been done to identify indoor concentrations for the numerous compounds likely to be present.

SOME MECHANISMS OF BIOMEDICAL EFFECTS

The wide variety of the chemicals in consumer products makes it difficult to anticipate all the possible adverse health effects. Some classes of chemicals have common characteristics that cause them to attack particular organ systems.

Various environmental chemicals have structural similarities that suggest that they may have similar effects on the myocardium; these chemicals have a lung-tissue half-life that could represent a long-term hazard. Some examples are polyhalogenated hydrocarbons used as insecticides and industrial chemicals, such as polychlorinated and polybrominated biphenyls (PCB and PBB). These may produce sudden death. The polyhalogenated hydrocarbons bind to estrogen receptors and are estrogenic in animal systems. This may increase cholesterol and triglyceride concentrations and so increase the risk of coronary heart disease (CHD), of mortality from CHD, or of CHD-related death.[35] Myocardial fibers may also be damaged by toxic agents, such as ozone.[27] Coronary-arterial-disease mortality has also been shown to be related to concentrations of suspended particulate matter in the external environment.[27] Thus, there may be many sources of cardiotoxic agents in the indoor environment.*

Organophosphorus pesticides, such as parathion, lead to clinical symptoms resembling strong cholinergic stimulation due to a nonreversible blockade of cholinesterase and leading to an accumulation of endogenous acetylcholine.[37] This accumulation disrupts the transmission of impulses from nerve fibers to muscles.[36] (In the case of parathion, skin may be the route of entry.) Chronic effects on

cerebrospinal fluid may also occur. Other disturbances of the nervous system may occur through exposure to such chemicals as PCB, which may be ingested and may be stored in fatty tissue. PCB inhibits growth in cultured cells and interferes with the activity of a variety of enzymes.[36] Pesticides occur in indoor environments through the spraying of pesticides or herbicides and through the contamination of items brought into the home, including foods and flowers.[25]

Solvents, especially chlorinated hydrocarbons, may damage the kidneys and liver.[38] Although the skin acts as an effective barrier, serious toxicologic effects may result from exposure of the skin to such substances as some organic phosphates, lead compounds, and acid. Dermatitis--especially in the form of dry, scaly or fissured reactions--can be caused by recurrent contact with solvents, emulsifying substances, dehydrators, or detergents. Acidic or alkaline gases and aerosols are readily dissolved in the aqueous protective film of the eye, on the mucous membranes of the nose and mouth, and on skin that is moist with sweat. Such exposure may also erode teeth and change hair structure.[33]

These mechanisms have been demonstrated; because the exposures described can occur in nonoccupational indoor spaces, their potential impact should be considered. However, the importance of such exposures is still difficult to evaluate.

SUMMARY AND CONCLUSIONS

Among the sources of pollutants in the indoor air that are due to consumer products or hobby or craft activities, many can harm exposed occupants. Such products usually bear labels with hazard warnings and instructions for use that, if followed carefully, will reduce pollutant exposures to a point that is presumably acceptable for healthy users. Willful abuse, as in the case of direct inhalation of aerosol products or careless use of solvents in enclosed spaces, can result in acute or delayed disorders or even death. The prohibition of fluorocarbons as propellants in aerosol spray products has resulted in substitution of other propellants that may be found to be toxicologically less desirable. The recognition of the carcinogenicity of vinyl chloride and benzene has resulted in the banning of these chemicals from consumer products, but a number of chemicals with serious toxic potential continue to be used.

Many consumer products are used only intermittently by a given person, and those in different households are likely to use different products for a given purpose. For safety, most products are formulated to avoid acute discomfort or irritation and because such acute effects will reduce marketability. Risks of long-term or delayed adverse health effects are not as likely to become apparent to the consumer and are not as likely to be incorporated in hazard warnings on products.

If the constituents of the consumer products are known, it may be possible to use a combination of occupational threshold limit values and likely exposure concentrations and exposure durations in making assessments of the impact of consumer products on indoor air quality.

Disclosure of product composition, assessment of acute and chronic consequences, and labeling with composition, directions for use, and hazard warnings specific for a particular formulation amount to one of several strategies that should be evaluated.

Monitoring or surveillance techniques now in use by such responsible agencies as the Consumer Product Safety Commission, the Environmental Protection Agency, and the Centers for Disease Control are more likely to discover acute consequences than delayed adverse health effects. The assessment of the human exposure and adverse health consequences due to the storage and use of consumer products is made difficult by the irregular, sporadic, and highly variable exposures, scarcity of measurements, and limited knowledge about composition of many of the products. Except for studies of accidental poisoning, epidemiologic assessments are almost completely impossible, owing to the episodic and irregular nature of exposures. It will be necessary to rely on knowledge of and experience with the use of the constituents of consumer products in the workplace. Assessment of the impact of consumer products on nonoccupational indoor air quality must, then, be based on constructed risks and potential exposures.

Labeling of consumer products with lists of constituents, instructions for safe use, and hazard warnings is often inadequate, and in any case it may be disregarded by the user and is ineffective when the products are handled by children.

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ASBESTOS

"Asbestos" is a collective term for the fibrous or asbestiform types of various minerals. Characteristics of flexibility, strength, and durability have brought these mineral fibers into numerous and varied applications involving potential exposure of large populations. Both widespread use and increasing investigations of the health effects of asbestos exposure have created intense interest in asbestos as an environmental contaminant. The health effects and toxicologic impact of mineral fibers are covered in detail in Chapter VII.

Because the asbestiform minerals have been used in numerous construction materials, consumer products, and appliances, the nonoccupational environment has become an area for investigation of asbestos contamination and human exposure. A potential for contamination from some types of these materials in structures during construction, renovation, demolition, and even normal use has been demonstrated. Repair and maintenance of household appliances, furnaces, stoves, and asbestos-cement pipes can also result in release of fibers into the air.

DEFINITION OF ASBESTOS

"Asbestos" is applied to chemically varied, naturally occurring mineral silicates of the serpentine and amphibole classification that are separable into fibers that are flexible and incombustible and usually have large length-to-diameter ratios. These asbestos, or asbestiform, mineral fibers have high tensile strength and desirable thermal and electric insulating properties and resist chemical degradation. Asbestos minerals with commercial and exposure importance are serpentine chrysotile and the amphibole group of asbestos minerals: amosite, crocidolite, anthophyllite, and actinolite-tremolite.[6 52]

IMPORTANT CHARACTERISTICS OF ASBESTIFORM MINERAL FIBERS

The characteristics of durability, airborne lifetime, and fiber dimension are especially important in determining the potential of exposure and biologic effect.

Durability

Asbestiform fibers retain physical integrity in nearly all uses and applications and within human tissue.[14 52]

Potential to Remain Airborne

This potential strongly affects exposure probability. Settling velocity depends heavily on fiber diameter and to a lesser extent on fiber length.[4 36] Settling in still air in a 3-m-high room, a fiber 5 μm long and 1 μm in diameter will remain airborne for approximately 4 h. A fiber of the same length with a 0.1-μm diameter will remain airborne for up to 20 h.[36] Such settling times could be prolonged in turbulent air, and, like other suspended particles, fibers can be transported by air currents. Disruptive mechanical forces cause predominantly longitudinal cleavage of fibers or fiber bundles into a larger population of particles with smaller diameters and increased persistence in the air.[3] However, the extent of fiber cleavage by natural forces in the environment is unknown.

Fiber Dimensions

The deposition and retention of fibers in the respiratory tract depend on fiber dimensions, breathing conditions, and airborne fiber concentration. Most fibers retained in human lungs are shorter than 5 μm, and have diameters less than approximately 2.5 μm,[12 14 26 40] but some thin fibers up to 200 μm have been found in lung samples.[40]

ASBESTOS PRODUCTION AND APPLICATION

Uses

The characteristics of durability, flexibility, strength, and resistance to wear bring the asbestiform minerals into thousands of applications. They are used in roofing and flooring products, textiles, papers and felts, friction materials, filters and gaskets, cement, panels, pipes, sheets, coating materials, and thermal and acoustic insulation.[38] Asbestos production began late in the nineteenth century, when it was used as thermal insulation for steam engines. Worldwide production is now nearing 5 million tons/yr, with chrysotile the principal fiber type.[42]

Production

Approximate consumption of asbestos in the United States was 600,000 tons in 1979[42] and is expected to be 400,000-900,000 tons in 2000. Over 90% of the asbestos used in the United States is imported, and over 90% of the imported asbestos is Canadian chrysotile. Over 70% of the asbestos is used in the construction industry.[2]

Spray Application

Of all the uses in the construction industry, the spray application of asbestos onto structural surfaces is the most important in the development of potential contamination situations. Such sprayed material is usually friable or susceptible to damage and disintegration by hand pressure. Sprayed material has been applied extensively to steel work to retard deformation during fire and to other structural surfaces for thermal and acoustic insulation, decoration, or condensation control.

Spray application of asbestos fibers began in the 1930s and allowed the rapid covering of irregular surfaces without the use of mechanical support or extensive preparation. Early spray applications in the United States were mainly for decoration and acoustic insulation. In 1950, the Underwriters Laboratories approved the use of sprayed asbestos where concrete had been required for prevention of deformation of steel from fire in multistory buildings. This approval brought about an intense use of sprayed asbestos material in new construction.[28,29]

However, evidence of the health hazards of asbestos exposure was accumulating.[22] In 1972, the New York City Council banned asbestos spray application because of the health hazard to spray operators, other construction workers, and the general public.[21] After failure of attempts at on-site asbestos-contamination control,[30] the EPA, in 1973, banned sprayed asbestos application for structural insulating or fireproofing.[48] Decorative materials and some heavy mix materials were not included in the ban. In July 1978, the EPA banned spray-on application of materials, except those in which the asbestos fibers are encapsulated with a bituminous or resinous binder during spraying and that are not friable after drying.[47] A rough estimate of the total amount of asbestos-containing materials sprayed over the 28-yr period is 500,000 tons.[36] Although the spraying of asbestos-containing materials in construction ceased, such friable material in existing structures remains a widespread asbestos-fiber source with potential for indoor contamination (see Lumley;[17] Sawyer;[31] and H. V. Brown, UCLA, personal communication).

ASBESTOS CONTAMINATION OF THE ENVIRONMENT

Fiber release depends on both material cohesiveness and the disruptive energy applied. The majority (85% or more) of asbestos in current use is immobilized in strong binding materials, such as cement or tiles; however, any asbestos-containing materials will release fibers when sufficiently disrupted, and the hard materials will liberate fibers if ground, sanded, or cut. The remaining asbestos--including that in insulation, troweled asbestos plaster, and pipe lagging--releases fibers upon minor disturbance.[36] These friable materials are the most important source of asbestos contamination in structures. Friable materials can be found on open and visible ceilings, walls, and structural members and on hidden surfaces accessible to maintenance, renovation, or ventilatory air flow.

The proportion of asbestos in such material is generally 10-30% by weight, but may vary from trace amounts to nearly 100%.[36] Other fibrous components include fibrous glass, mineral wools, and cellulose. Friable materials can also contain vermiculite, talc, perlite, diatomaceous earth, organic fibers, clays, quartz, gypsum, and various adhesives.

Environmental contamination from asbestos-containing materials can occur in three general ways: fallout, contact disruption, and reentrainment of previously released but settled fibers.[35] Fallout is, except for very friable material, negligible. Contact disruption and reentrainment are activity-dependent and can result in substantial contamination and exposure.

Fibers enter occupied spaces at a relatively low rate, depending on material friability and exposed surface area. Variations in the fallout rate are due to structural vibration, air movement, and changes in cohesiveness. Fallout can result in the accumulation of surface deposits of fibers over long periods; such accumulations are then available for later disturbance and reentrainment.

Any asbestos-containing material will release fibers if the energy applied to it is adequate. Contact may be intentional during demolition, renovation, or vandalism, unavoidable during maintenance, or accidental during routine activity. Fiber release depends on probability and energy of contact. Contamination is episodic and local and can be intense.[36]

The disturbance of released and accumulated fibers can cause repeated cycles of settling and resuspension. Such reentrainment contamination may occur after any disturbance, but can be important in custodial activities.[36]

ENVIRONMENTAL SAMPLING FOR ASBESTOS

Analysis of Bulk Materials

Identification of asbestos is relatively simple with mineralogic specimens that are generally uniform in type and composition. With samples of construction materials, identification is more difficult. The amount of asbestos may be small, and construction materials may contain other fibrous components with a variable collection of nonfibrous components.

The primary method for asbestos identification in bulk materials is polarized-light microscopy (PLM). X-ray diffraction (XRD) is used for quantitative analysis of fiber type.[18 31] Transmission electron microscopy has only limited application.[36]

The petrographic microscope is a transmitted-polarized-light instrument widely used for identification and characterization of substances based on their optical and crystallographic properties. The techniques are established, and the equipment is inexpensive. However, a high degree of skill and experience is required of the microscopist.[18 31 36]

X rays diffracted by crystalline material produce a characteristic pattern. The technique usually yields information with a high degree of diagnostic reliability and a printed record. It is usually used to confirm results of petrographic microscopy. X-ray diffraction requires a large investment in equipment, references, mineral standards, and technical expertise. X-ray diffraction of bulk construction material cannot define particle shape and may fail to detect concentrations of asbestos much below 5%. Moreover, other silicates or crystalline phases can interfere with asbestos identification.[18, 31]

Specific and accurate fiber identification can be achieved by examination of the structure of individual particles, especially in conjunction with electron diffraction or energy-dispersive x-ray analysis. The extrapolation of precise electron-microscope data, however, to bulk sample information is inefficient and costly. Its use in identification is usually confined to resolving ambiguities raised by petrographic microscopy and x-ray diffraction. The most important use of such analytic techniques is the identification and analysis of inorganic particles in tissue.[14, 36]

Errors in asbestiform-mineral analysis have potentially serious consequences and are not uncommon. False-negative results will lead to a continuation of unnecessary environmental contamination, and false-positive results can precipitate unnecessary action. Errors arise from analyst inexperience and from the use of phase-contrast microscopy, rather than the appropriate polarized-light instrument.[37]

Measurement of Airborne Asbestos

A pump is used to draw a measured volume of air through a membrane filter. The pump and filter are either stationary or carried on a person, with the sampling orifice in the respiratory zone. Common sampling rates are 2.0 L/min for personnel monitoring and 10 L/min for general environmental sampling. Sampling times vary from minutes to many hours, depending on anticipated fiber concentrations. Filter segments can be examined by various methods and observers for comparison or verification and can be stored indefinitely.[15]

Estimation of the amount of asbestos collected on the sampling filters is performed by one of two methods: counting fibers by optical microscopy with a phase-contrast light microscope and counting fibers by electron microscopy.

The standard technique for fiber enumeration is specified by the National Institute for Occupational Safety and Health (NIOSH) for determination of airborne asbestos in occupational settings.[15, 19] Air is pumped through a membrane filter (effective pore size, 0.8 μm). A filter segment is examined with a microscope that has phase-contrast illumination at a magnification of 400-450. Particles longer than 5 μm and with a length-to-width ratio (aspect ratio) greater than 3 are counted. Results are presented as the numbers of fibers per milliliter of air.[15] This method enumerates only particles of defined aspect ratio and length. It is not capable of accurate identification of asbestos. Both the resolution limits of the

optical microscope and the 5-μm length limit preclude enumeration of some smaller fibers, which may be present and in numbers greater by an order of magnitude or more.[9 10] However, the short fibers that are not counted may be less hazardous than the longer ones that are.[33 51]

Electron microscopy is the definitive method for both identifying and counting small asbestos particles. When it is combined with selected-area electron diffraction (SAED) and energy absorption, accurate identification of a particle is possible.[13 27] Laboratories vary in techniques of sample preparation, magnification, and mass estimation, and comparison of results has been discouraging.[16 36] Furthermore, there are no standards for interpretation of exposure data derived this way. Investigation of asbestos contamination by electron microscopy is both expensive and extremely time-consuming. Results are usually given in nanograms per cubic meter for mass estimations, but can also appear as fibers per milliliter where only enumeration is performed.[13 27 36]

Other Contamination Assessment Methods

The most relevant estimate of asbestos hazard is based on the concentration of airborne, respirable asbestos fibers in the immediate environment of building occupants. Under the usual conditions and with the standard NIOSH optical-microscopy technique, this task is difficult. The technique was originally intended for use in areas of recognized contamination, such as asbestos production facilities. The airborne contaminant was known, and relatively high concentrations were readily measured. In this setting, the optical technique is effective and appropriate. However, in the assessment of exposure situations in other, nonoccupational structures, it has become apparent that it is not entirely satisfactory.[17] The optical-microscopy method is truncated in its limit of resolution both physically and by regulation. The 5-μm lower limit of counted particles will preclude the enumeration of many small fibers in the sample environment.[10] Fiber emission is nearly always local, sporadic, and activity-related. Routine air-sampling commonly fails to describe this contamination situation.[16 17]

An alternative approach to assessment by air sampling involves a subjective observational ranking system to provide guidance in potential contamination situations.[11 49] This process evaluates relevant contamination factors that can contribute to total contamination potential. Rating systems are an approach to a complex process involving fiber aerodynamics, material characteristics, structure effects, and human activity. However, they are not exact, are subject to variations in factor estimation, and cannot be easily evaluated with existing methods. They do not provide a precise benchmark for selecting appropriate corrective actions, but can provide consistent guidance in assessment and evaluation.

An adequate system should reflect factors that influence material fallout, direct material disturbance, and reentrainment of fibers. These modes of contamination are functions of material characteristics,

structure configuration, and user activities. One example is a system that considers eight factors that influence exposure.[11] It is predictive to the extent of estimating the probability of contamination and exposure potential. The factors are:

- Total asbestos content of material.
- Friability of material.
- Condition of material.
- Extent of damage of material by water.
- Accessibility of material to activity and contact.
- Surface area of material exposed.
- Activity or movement in environment.
- Plenum or other airstream effects.

The factors are individually weighted and used in a formula to generate a single score. Contamination evaluation by this method will provide guidance in two ways: (1) It indicates corrective action; scores exceeding a given value indicate that hazard potential is substantial. (2) It establishes relative priority, the most useful feature of the scaling system; the higher the score, the greater the need for corrective action, and scores can be used to establish a logical sequence of corrective actions within indoor spaces or buildings.

ASBESTOS AIR DATA

Studies have been performed in structures containing various types of asbestos material (classified as either friable or bonded) and under a series of activity conditions. Tables IV-10 and IV-11 list airborne-fiber concentration data obtained by optical microscopy (with the NIOSH standardized technique[13]) and electron microscopy. Nearly all the data were collected in nonoccupational settings in apartment buildings and private homes and in offices and schools. Both surveillance and reenactment studies are included.

The airborne-fiber concentrations determined by optical microscopy range from zero in background, quiet, and some routine activity to over 100 fibers/ml in stripping dry-spray-applied ceiling material. Fiber contamination has an expected relationship to activity and proximity.

The data in Table IV-11 obtained by transmission electron microscopy are expressed as nanograms per cubic meter. Thus, they do not distinguish between fibrous and nonfibrous asbestos, nor between long and short fibers. Estimation of the health significance of mass-concentration data from Table IV-11 is impossible. There is a lack of methodologic standardization, exposure standards, and applicable epidemiologic information that relates fiber number concentration to mass concentration. The electron-microscopy results vary from zero to nearly 2,000 ng/m^3. There is a progression in contamination from background through routine and custodial activity.

Exposure probabilities can be estimated to some degree by consideration of modes of contamination and activity in a building.

TABLE IV-10

Airborne Asbestos in Structures: Optical Data

Main Mode of Contamination	Activity	Mean Count, fibers/ml	n	Range or SD	References
Friable asbestos-containing material:					
Mixed fallout	Background, city	0.0	42	0.0	32,33
	Quiet conditions	0.0	65	0.0	32,33
Reentrainment	Routine activity:				
	Dormitory	0.1	10	0.0-0.8	[a]
	Schools, general	0.0	79	0.1	32,33
	Offices	0.0	60	0.0-0.6	32,33
	Dry sweeping, floor	1.6	5	0.7	33
	Dry dusting near face	4.0	6	1.3	33
	Bystander to cleaning	0.3	3	0.3	33
	Heavy dusting	2.8	8	1.6	36
	Laundry (contaminated clothing)	0.4	12	0.0-1.2	33
Contact with material	Maintenance:				
	Relamping	1.4	2	0.1	33
	Plumbing	1.2	6	0.1-2.4	32
	Cable movement	0.4	24	0.2-6.4	32
Mixed: contact reentrainment	Renovation:				
	Ceiling repair	17.7	3	8.2	32
	Track light	7.7	6	2.9	33
	Hanging light	0.3	12	0.8	33
	Partition construction	3.1	4	1.1	33
	Pipe lagging	4.1	8	1.8-5.8	36
Contact	Ceiling damage by vandalism	12.8	5	8.0	32
	Stripping dry ceiling material	82.2	11	22.4-117.0	33
	Stripping wet (amended water) ceiling material	1.2	96	5.2	32,33[a]
	Drilling, machining	3.4	7	1.0-5.8	32
	Abatement by encapsulation	0.0	28	0.7	32

Table IV-10 (contd)

Main Mode of Contamination	Activity	Mean Count, fibers/ml	n	Range or SD	References
Bonded asbestos-containing material:					
Contact	Stripping cementitious by wet method (amended water)	0.1	26	1.0	32
	Machining:				
	Sanding tiles	1.2	2	1.2-1.3	20
	Sanding concrete	7.2	6	2.1	32
	Cutting concrete	6.3	14	2.3	32
	Grinding concrete	0.3	6	0.2	32
	Sanding taping	5.3	11	1.3-16.9	30

[a]H. V. Brown, personal communication.

TABLE IV-11

Airborne Asbestos: Electron-Microscopy Data

Location and Activity	Mean, ng/m^3	n	Range, ng/m^3	Reference
Urban outdoors:				
48 U.S. cities	<10	187	--	23
N.Y. City	17	22	2-65	25
N.J. schools	14	3	3-30	25
Indoors:				
Friable-asbestos, structural surface:				
Office building	79	3	40-110	36
N.Y. City schools	99	5	9-135	24
Mass. schools	151	5	38-260	24
N.J. schools	217	27	9-1,950	25
Office buildings	2.5-200	116	0-800	24
Custodial activity:				
N.J. apartment	296	1	--	36
Conn. school	643	2	186-1,100	36
N.J. school	1,950	1	—	25

Quiet or background conditions may represent the usual extent of activity in areas with low probability of either contact or reentrainment of asbestos. Under these conditions, contamination is low. Routine activities in a structure containing sprayed-asbestos surfaces will not usually result in detectable fiber concentrations. Routine activity can result in intense contamination in some situations. A school population's routine activity in a building with accessible asbestos surfaces may cause environmental contamination. Increased fallout, occasional contact, and reentrainment may all contribute to the highly variable fiber concentrations found under these conditions.

Custodial work can cause disturbance and reentrainment of accumulations of asbestos fibers. Reentrainment can be high during custodial activity, depending on cleaning methods. Maintenance work may involve direct contact with asbestos surfaces. Such activities may result in marked fiber dissemination.[33, 56]

Uncontrolled removal of sprayed-asbestos surfaces during renovation not only causes high fiber concentrations for the duration of the work, but also increases the released-fiber burden in the structure. In such cases, exposures involve the renovation worker and the routine building-user as well.[33] Before a decision on building renovation to remove asbestos, the potential contamination during and after renovation must be evaluated. Both contact and reentrainment release mechanisms are involved, and very high concentrations occur during actual contact.

STANDARDS

Estimation of the hazard associated with the airborne fiber concentrations of Table IV-10 can be only approximate. No exposure standards to evaluate hazard have been developed for the general indoor environment. The only existing standard is that of the Occupational Safety and Health Administration (OSHA). Other recommendations for occupational exposure limits are those of NIOSH and the American Conference of Governmental Industrial Hygienists (ACGIH). Although these apply to and were intended for only occupational exposures, their consideration in the general indoor environment may have some merit:

- The standard optical-microscopy method is used, and the air data of Table IV-10 are comparable with the standard limits.
- The occupational exposure limits represent a distillate or summary of both exposure and epidemiologic information. Recently proposed changes reflect additional relevant epidemiologic evidence.

The use of the occupational exposure limits is considered by some investigators to be acceptable for approximating the exposure hazard.[34]

Table IV-12 outlines the occupational exposure limits from 1972 to the present. Comparison of the data in Table IV-10 with the exposure limits of Table IV-12 demonstrates that some activities can exceed

TABLE IV-12

Occupational Asbestos Exposure Limits

Limit	Time-Weighted Average (8 h/d), fibers/ml	Ceiling, fibers/ml
OSHA original, 1972[46]	5.0	10.0
OSHA present, 1976[45,46]	2.0	10.0
OSHA proposed, 1975[43]	0.5	5.0
NIOSH revised, 1977[41]	0.1	0.5
ACGIH adopted, 1980:[1]		
Amosite	0.5	--
Chrysotile	2.0	--
Crocidolite	0.2	--
Other forms	2.0	--

present time-weighted average (TWA) OSHA limits. Occasional events--such as removal, renovation, and vandalism (contact-mode categories)--exceed the 15-min excursion limit of 10 fibers/ml.

The present TWA and ceiling limits were set by the 1972 regulations to become effective in 1976.[16] The more recent limits, the 1975 proposed and the 1977 revised recommended, reflect the increasing awareness in asbestos-disease epidemiology and are more stringent. With each successive regulation, the range of activities that could be considered hazardous becomes more inclusive. The activities remaining outside the 1977 limits are only in categories of quiet and nonspecific routine. Occupational standards can potentially be exceeded during renovation, maintenance, and custodial activities that disturb applied material or accumulated fibers.

The optical-microscopy data indicate that contamination can sometimes exceed concentrations considered hazardous. Exposures occur in existing structures, and the population involved is large and varied in age, occupation, and behavior.

Children attending schools that contain friable asbestos material constitute a population of special concern. The schoolchildren population differs from other nonoccupational populations in age, population density, and behavior. Any exposure would occur early in their life, leaving a long period for development of asbestos-related diseases. A large number of students can be exposed at one time to asbestos that is released from asbestos-containing materials in the school building. The school population is also very active. Friable asbestos-containing materials can be damaged during routine activities and as a result of capricious behavior. Many cases of badly damaged asbestos-containing materials have been found in schools.[17]

REGULATIONS

Most asbestos handling, control, and disposal in the structural environment are subject to regulation by the EPA and the OSHA. Other federal agencies that regulate asbestos in various settings are the Mine Safety and Health Administration (MSHA), Consumer Product Safety Commission (CPSC), Food and Drug Administration (FDA), and Department of Transportation (DOT).

In accordance with section 112 of the Clean Air Act ("National Emission Standards for Hazardous and Air Pollutants"), the EPA promulgated regulations on asbestos in 1973 (40 CFR 61, Subpart B, "National Emission Standard for Asbestos").[18]

These regulations apply to the renovation or demolition of friable asbestos materials and to the spraying of asbestos. They specify procedures for removal and stripping of friable sprayed-asbestos fireproofing and insulation materials. The required work practices include EPA notification, material-wetting, containment, container labeling, and disposal of the removed material in an approved landfill. Fiber concentrations are not specified, but the regulations require that there be no visible emission outside the structure.

Actions taken by the CPSC have banned the use of asbestos textiles in general-use garments, asbestos in artificial-fireplace materials, and asbestos-containing spackling and taping compounds.[7,8] Voluntary actions by manufacturers controlled the use of asbestos in hand-held hair-dryers.

CONTROL OF CONTAMINATION POTENTIAL

Where a potentially hazardous situation has been identified in a structure, asbestos-contamination control or elimination methods are indicated.[36,39] There are four approaches to corrective action: preventive management (a specific management system initiated to prevent disturbance of asbestos-containing material, with no direct action taken on the material itself), removal and disposal by burial, encapsulation (asbestos-containing material is coated with a sealant), and enclosure (asbestos-containing material is separated from the building environment by barriers, such as sealed suspended ceilings).

The corrective methods can be used separately or in combination, and each has its own advantages. Removal eliminates the source of exposure to asbestos. Both enclosure and encapsulation are containment methods; because the asbestos material remains in the building, enclosure and encapsulation should be considered as temporary control methods, for use until the building is renovated or demolished.

The surface of asbestos-containing material can be damaged--causing the friable fragments to be released--by inadvertent or uninformed maintenance, repair, or renovation. A management system should be implemented to control any activity by either structure personnel or contractors. Any necessary work would be performed under controlled conditions to protect involved personnel and other building users and to prevent contamination of the building environment. Renovation or demolition should include elimination of friable asbestos-containing materials under safe conditions and protection for the worker, the building users, and the community.

Corrective action for surfaces considered very hazardous should be planned with priority appropriate to their contamination potential. Removal is the ultimate solution and will end contamination potential. Encapsulation with either penetrating or bridging sealants will enhance the cohesiveness of the material, eliminate fallout, and protect from minor damage. The technology of encapsulating asbestos has recently been the subject of intense interest, and effective sealants are now available. Management and control should be initiated for all areas that contain friable asbestos material, no matter what other action is planned. An asbestos management system can be implemented immediately, has low cost, and is highly effective in exposure control.[37,39]

SUMMARY

Asbestos is a widespread component of the structural environment. Release of asbestiform mineral fibers from structural components

depends on the cohesiveness of the asbestos-containing material and the intensity of the disturbing force. Asbestos-containing material that is friable is most readily released in structures. High-energy or machine disruption is necessary for the release of fibers from hard or bound asbestos-containing materials. Durability and aerodynamic capability have combined to produce a persistent and important contaminant for human exposure. Contamination can be due to slight fiber release in fallout, relatively great release by contact or direct material disruption, or reentrainment of fallen and accumulated fibers. Most contamination is episodic, activity-related, and local. Documented concentrations have been compared with existing and proposed occupational standards. Extensive disruption of asbestos-containing material results in substantial hazard potential. Removal, renovation, and demolition of friable material and machining of hard asbestos-containing material result in a high degree of environmental contamination in the vicinity of the disturbance. Maintenance work or custodial care involving either friable material or accumulated fibers can cause airborne contamination that should be considered hazardous.

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FIBROUS GLASS

Fibrous glass is a man-made inorganic fiber with widespread application and distribution in the fabrication, textile, and construction industries. It is used in thermal insulation (for

structures, appliances, and pipe), acoustic insulation, textiles, plastic-material reinforcement, tire cord, yarns, matting, and filters. It is a common component of the structural environment. Although the production of fibrous glass is a half-century old, interest in potentially serious adverse health effects has occurred fairly recently.

DEFINITION

The term "fibrous glass" generally includes particles composed of glassy material with a length-to-width (or length-to-diameter) ratio that exceeds 3. The composite elements of the glassy material form an amorphous structure and are not well ordered or crystalline, as in the asbestiform minerals. Contemporary conventional fibrous-glass production blends silica sand, limestone, and soda ash as raw materials in a continuous process. Before 1950, glassy fibrous materials, commonly known as "mineral wools," were commonly produced by melting the slag of ore-smelting processes (slag wool) or naturally occurring rocks (rock wool). Mineral-wool production began serious development in this country after 1920 and reached a peak in the 1950s, before modern continuous glass processes reduced the use of slag and rock as primary materials.[13,16]

There are two major categories of fibrous-glass products: continuous-filament glass and glass wool.

Continuous-filament glass is used in textiles and fabrics; as reinforcement in plastics, rubber, and paper; and in numerous other applications. It is produced by extruding molten glass through dimensioned orifices to yield fibers with fairly well-defined diameters. Continuous glass fibers can be selectively sized to provide the strength, hardness, or thermal properties desired for the intended application. Most continuous-filament operations produce fibers roughly 6 µm in diameter, with some glass reinforcing fibers having diameters of over 10 µm (see Pundsack;[13] Smith;[16] and W. Rietze, Johns Manville Corporation, personal communication).

The most important example of glass wool is the fibrous-glass thermal-insulation material used extensively in construction and equipment fabrication. Contemporary glass-wool production is a continuous processing of raw materials through melting, fiberization, and packaging. Fiberization devices use rotors or high-velocity gas jets either alone or in combination to produce particles of a desired dimension range. The diameter is important in thermal-insulation production, because the effectiveness of the material varies inversely with the fiber diameter. The fiber population produced in this way does not have the well-defined diameter of the continuous-filament process. A glass-wool fiberization system produces a range of sizes following a frequency distribution characteristic of the process.[19] Most commercial fibrous-glass insulation has mean fiber diameters in the range of 4.0-9.0 µm. For special applications, a small percentage of glass wool is produced at mean diameters of 1.0 µm or less.[13,16]

CONCERN OVER POTENTIAL ADVERSE HEALTH EFFECTS

The increasing concern over the potentially serious health effects of fibrous-glass exposure is a consequence of a number of factors:

- Interest in the carcinogenicity of asbestiform mineral fibers has raised questions concerning a possible similar effect of other fibrous materials, including fibrous glass.[17]
- The fibrogenicity and carcinogenicity of fibrous glass had been demonstrated in animal inoculation studies in which mesotheliomas were produced by intrapleural and intraperitoneal implantation of fibrous glass.[19 22]
- Some studies of the mechanism of carcinogenicity associated with fibers have indicated that fiber dimensions are more important than physical or chemical properties.[18] The demonstration of the apparent influence of particle size and shape indicated the possibility of common mechanisms and effects among fibrous materials, specifically asbestos and fibrous glass.
- Glass fibers 1.0 μm or less in diameter are termed "microfibers." In animal studies, fibers of pathologic importance are in the microfiber range, with diameters less than approximately 0.5 μm.[19] Fibers considered respirable have diameters of approximately 3.5 μm or less.[18 20] Furthermore, the aerodynamic capability and potential for respiration increase mainly as a function of decreasing fiber diameter.[20 21] This implicates microfibers as the most suspect in pathologic importance, with respect to cellular effect, respirability, and aerodynamic capability.[4] Microfibers are being intentionally produced for special applications, and there is incidental microfiber production in some glass-wool processes.
- The use of fibrous glass is widespread and increasing. There is heavy consumption of fibrous glass by the construction industry, including friable materials that readily release fibers in the structural environment. The substitution of fibrous glass for asbestos and the demand for fibrous-glass insulation products for energy conservation will increase the use of these friable materials. The forms, uses, and distribution of fibrous-glass materials implies a substantial impact if the material has marked adverse health effects.

There is concern that human exposure to fibrous glass may cause disease. However, studies of mortality and morbidity and radiographic examinations have failed to demonstrate discernible hazard in occupational populations with exposure to fibrous-glass particles.[8 9 12 23]

Glass microfibers could be expected to have more airborne persistence, respirability, and cellular effect. However, no human epidemiologic data support the concept of microfiber pathogenicity. The industrial use of microfibers is relatively small, and there have been few accumulated years of human exposure.[4] However, in the older mineral-wool production, with a wide distribution of fiber dimensions, there has been a substantial population of particles that meet the microfiber definition. In the mineral-wool industry, the accumulated

exposure experience is large in numbers and years.[13] The health significance of microfiber exposure in human populations is not known.

IMPORTANCE OF CHARACTERISTICS OF FIBROUS GLASS

The potential for environmental contamination and exposure is influenced by the dimensions of fibrous-glass particles. Both airborne persistence and respirability of the environmental contaminant are less than those of asbestos, because of the relatively large diameters of the glass particles.

Emission rates of fibrous glass depend on the proximity of the source material, general characteristics of cohesiveness and friability, and the intensity of the force causing the disruption. Studies of the general environment, including space ventilation systems, have demonstrated extremely low concentrations, less than 3 fibers/L.[3] Glass-fiber concentrations in occupational and production environments vary widely with the nature of the production process. Most studies have shown normal production-facility concentrations well below 1 fiber/ml as measured by the OSHA-NIOSH standard method for airborne asbestos.[5-7,10] As would be anticipated from aerodynamic considerations, studies in microfiber production facilities have documented airborne concentrations orders of magnitude higher than those in conventional fibrous-glass processes.[9,10]

The more friable forms, such as thermal insulation, are potential sources for environmental contamination. Studies of concentrations of airborne fibrous particles during removal of friable insulation material have shown high concentrations of airborne fibrous glass in the vicinity of worker activity. During the removal of friable spray-applied material (20% chrysotile asbestos and 70% fibrous glass), fiber counts in excess of 100 fibers/ml were encountered.[15]

ANALYSIS

The polarized-light microscope can be an effective instrument for identification of fibrous glass in construction materials. Characteristic shape, transparency, and lack of birefringence distinguish fibrous glass from asbestos mineral fibers. Modern fibrous glass usually appears as isotropic particles of fairly uniform diameter, rod-like appearance, and high length-to-diameter ratio. Some mineral-wool products may have highly variable dimensions, teardrop shapes, and spherical glass "shot" of relatively large diameters.[11,14]

STANDARDS

A TLV/TWA of 10 mg/m^3 for fibrous glass or dust has been listed by the ACGIH.[24] This had been listed as a nuisance particle, with the occupational exposure limit for fibrous glass given as 30 x 10^6 particles/ft^3, or 10 mg/m^3 of air.[1] In a recent industry-wide

survey, concentrations of airborne particulate matter were generally less than 2.5 mg/m^3.[5]

CONTROL

In consideration of the uncertainties of carcinogenicity, relevance of the experimental tumor-production studies in animals, and the characteristics of glass-fiber exposures of people, it appears prudent to reduce microfiber exposure to the lowest possible point permitted by available technology.

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COMBUSTION SOURCES

Whenever unvented combustion takes place indoors or venting systems attached to stoves, boilers, or heaters are malfunctioning, a wide range of combustion products can be discharged directly into the indoor atmosphere. This section summarizes essential information pertaining to indoor sources of combustion products and their effects on indoor air quality. The emphasis is on residential buildings, where research efforts on indoor air quality have been concentrated, but results of limited studies of combustion-generated pollution in other types of buildings are also presented.

In general, the data presented here represent isolated situations whose characteristics are highly specific to the site and the combinations of activities that produced the effects; therefore, transfer of these findings to other situations must be done only with extreme care.

Smoking (which is discussed elsewhere), is the most widely encountered source of combustion products indoors. Besides smoking, the primary sources of combustion byproducts in residential buildings are usually space heaters, gas stoves, and gas water heaters. Exhaust from automobiles in attached garages can also be a source of combustion byproducts in buildings, as can wood fires, oil and kerosene lamps, and candles.

The major pollutants associated with indoor combustion are carbon monoxide, nitric oxide, nitrogen dioxide, aldehydes and other organic compounds, and fine particles. These combustion products usually occur in low concentrations, compared with the major combustion products--carbon dioxide and water vapor. Inefficient combustion from unvented or poorly vented space heaters, fireplaces, and lamps can also emit carcinogenic hydrocarbon particles. Carbon dioxide and water vapor are also produced as a result of normal metabolic processes of building occupants and add to the burden associated with gas appliances. Humans produce 30-60 g of carbon dioxide and a similar amount of water vapor per hour. An unvented space heater rated at 10,000 Btu/h, or about 2,500 kcal/h, produces around 750 g of carbon dioxide per hour. Accordingly, depending on occupant density, space limitations, and the extent of ventilation and infiltration, carbon dioxide concentration in the indoor atmosphere can rise substantially above the normal range of 0.03-0.06%. Respiration is affected when the concentration of carbon

dioxide in the air rises above 1.5%, and concentrations above 3% can cause headache, dizziness, and nausea. Above about 6-8%, carbon dioxide causes stupor to the degree that exposed persons are unable to take steps for self-preservation.[4] The Threshold Limit Value in the occupational environment is 0.5%[1]--a value which has also been applied to submarine crews, whose incidence of illness increased after long-term exposure to concentrations of 0.5-1%.[19 22 25] In the residential environment under occupied conditions, carbon dioxide concentrations are typically 0.07-0.20%.

RESIDENTIAL BUILDINGS

Space Heating

There are many documented cases of health problems and even deaths resulting from excessive carbon monoxide released by unvented or improperly vented heating systems;[2 16 18 27] however, few systematic studies have provided detailed measurements of indoor air-quality problems in such houses. Most space heating in U.S. houses is by externally vented heating systems (central furnaces or space heaters). When the heating system is properly designed, maintained, and functioning, combustion products that could directly affect indoor air quality do not enter the indoor environment. However, if a negative pressure develops in the interior space or if there is a faulty exhaust system (e.g., a cracked heat exchanger or blocked flue), there can be direct and serious degradation of indoor air quality.

Some space heating of homes is generally provided by unvented gas and kerosene heaters. This type of heating is more commonly used in rural areas and warm climates, such as the southern United States, and is especially dangerous, because it emits its combustion products directly into the living space.

In their study of indoor air quality in several homes in Rotterdam, Biersteker and associates determined that sulfur dioxide concentrations indoors are not normally affected by heating systems that are kept in good condition.[3] However, in one older home with a faulty heater, the indoor concentration was 3.8 times the outdoor concentration.

In a 1969-1970 study of indoor-outdoor air quality in the United States, Yocom and co-workers selected four homes, two public buildings, and two office buildings for analysis.[28 29] One of the two homes with coal heating had an antiquated central heating system with a leaky flue. Sulfur dioxide in this home approached 1 ppm and carbon monoxide exceeded 50 ppm over periods of 1 h and longer, coinciding with periods when coal was added to the fire and the fire was stoked.

As part of a pilot study to assess indoor air quality in buildings, Hollowell and co-workers showed that a gas-fired heating system in one home, although vented to the outside, produced higher nitric oxide and nitrogen dioxide concentrations indoors than outdoors.[9] In this work and in the earlier work of Yocom et al.[28 29] and Biersteker et al.,[3] no attempt was made to measure the emission rates of the

combustion source or the rates at which pollutant gases entered the indoor environment.

In a 1973-1974 study of indoor sources of air pollutants, Cote and co-workers determined the emission rates of carbon monoxide, nitric oxide, and nitrogen dioxide from an unvented gas-fired space heater.[6] Table IV-13 shows the emission data obtained.

Yamanaka and co-workers measured nitrogen oxides from various unvented and vented space heaters commonly used in Japan.[26] Results on both radiant and convection types of unvented kerosene heaters, as well as on various water heaters and gas stoves, were reported. Nitrogen dioxide emission from the radiant kerosene-fired space heater averaged 46 μg/kcal (0.011 g/MJ); that from the convection type averaged 251 μg/kcal (0.060 g/MJ).

In all the studies noted, the purpose was to measure emission of "typical" units. The units tested were not necessarily representative of the entire class of devices from which they were selected, nor was there any attempt to conduct an exhaustive study of their combustion characteristics.

Homeowners in many parts of the country are returning to the use of wood as a heating fuel, because of the increasing cost of oil and natural gas. This trend is especially strong in the northeastern states, which have depended largely on oil as a heating fuel. (The price of No. 2 fuel oil increased by a factor of 6 or more between 1965 and 1980.)

Wood stoves and fireplaces are vented to the outdoor atmosphere, but a number of circumstances can cause combustion products to be emitted to the indoor atmosphere: improper installation (e.g., insufficient stack height), cracks or leaks in or poor fitting of stovepipe, negative air pressure indoors, downdrafts, and accidents, as when a log rolls out of the fireplace. Although much is known about the combustion products of fuels used for space heating, little is known about the impact of the emission from wood stoves and fireplaces on indoor air quality--a subject urgently in need of investigation. Combustion products of wood are highly irritating to the eyes, nose, and respiratory system and thus provide a warning to occupants that combustion products are present. Table IV-14, from the work of Duncan and co-workers, shows the types of pollutants associated with wood burning.[7]

A field monitoring program designed to compare indoor and outdoor pollution in 10 residences and two office buildings was undertaken in the Boston metropolitan area.[13] Three of the monitored residences used either a wood stove or a fireplace in the course of the sampling period. Increased indoor concentrations of total suspended particles (TSP), respirable particles, and benzo[a]pyrene were observed during periods of wood-burning. The average indoor TSP concentrations during days with wood-burning were about 3 times the corresponding concentrations during days without wood-burning. Indoor 24-h benzo[a]pyrene concentrations during days with wood-stove use were an average of 5 times higher than those during non-wood-burning periods. The authors concluded that the increased indoor concentrations of TSP,

TABLE IV-13

Pollutant-Gas Emission from Unvented Gas-Fired Space Heaters[a]

Operation	Heat Input, kcal/h	Pollutant Emission Factors, µg/kcal			Pollutant Emission Rates, mg/h		
		NO	NO_2	CO	NO	NO_2	CO
Low flame, steady state	2,800	76.4	46.4	632	214	130	1,770
High flame	6,200	135	43.8	319	837	272	1,982

[a]Based on Cote et al.[6]

TABLE IV-14

Emission from Residential Wood-Fired Stoves[a]

Substance Emitted	Emission, lb/cord[b] Range	Average
Criteria pollutants		
Particles	3-93	30.3
SO_x	0.5-1.5	0.7
NO_x	0.7-2.6	1.6
Hydrocarbons	1-146	41.6
Carbon monoxide	300-1,220	598.3
Noncriteria pollutants		
Polycyclic organic materials	0.6-1.22	0.9
Formaldehyde	0.3-1	0.8
Acetaldehyde	0.1-0.3	0.4
Phenols	0.3-8	3.3
Acetic acid	5-48	21.1
Aluminum	--	1.3
Calcium	--	10.2
Chlorine	--	0.1
Iron	--	0.7
Magnesium	--	2.0
Manganese	--	1.6
Phosphorus	--	1.0
Potassium	--	3.6
Silicon	--	1.6
Sodium	--	0.7
Titanium	--	0.02

[a]Based on Duncan et al.[7]

[b]The relationship used to convert from lb/ton to lb/cord was: 1 lb/ton = 1.65 lb/cord.

respirable particles, and benzo[a]pyrene attributed to wood-burning may have long-term health implications.[18]

Gas Stoves: Pollutant Emission Rates and Concentrations

The early pilot studies of Yocom and co-workers, reported in 1974, showed that, on the basis of relative indoor-outdoor concentrations of carbon monoxide, unvented gas stoves definitely contribute to the deterioration of indoor air quality.[28]

A brief study by the EPA showed that peak nitrogen dioxide concentrations up to 1 ppm (about 1,880 $\mu g/m^3$) and 1-h averages of 0.25-0.50 ppm (about 470-940 $\mu g/m^3$) are reached in a closed kitchen with no external ventilation.[8]

Wade and co-workers in 1973-1974 studied four homes equipped with gas stoves to determine the concentrations of nitric oxide, nitrogen dioxide and carbon monoxide and their impact on indoor air quality.[24] Sampling was carried out for 2-wk periods in each home, simultaneously at four sampling locations--three indoors and one outdoors. Table IV-15 presents the principal data from one home included in this study. The following were the main conclusions of the study:

- Emission from gas stoves contributes nitrogen dioxide, nitric oxide, and carbon monoxide to the indoor atmosphere of houses where such stoves are used. Kitchen concentrations of these gases responded rapidly to stove use and, for a given house during a given season, there was a rough correlation between average nitrogen dioxide concentrations and average stove use.
- Nitrogen dioxide and nitric oxide were produced in roughly equal amounts in the homes where testing was conducted. Indoor concentrations of these pollutants were invariably higher than those outside.
- Normal stove operations frequently resulted in nitrogen dioxide concentrations in the kitchens averaging over 100 $\mu g/m^3$ for the 2-wk sampling periods.
- Comparison of samplings carried out in the spring-summer of 1973 and the fall-winter of 1973-1974 showed that in the colder weather, when the house was closed up more often, pollutant concentrations were more uniformly distributed in the various rooms of the house than in the warmer months.
- A diffusion experiment conducted in one of the houses showed that the half-life of nitrogen dioxide was only one-third that of carbon monoxide and nitric oxide, indicating that nitrogen dioxide decays through reaction or adsorption, in addition to normal dilution from air exchange. This effect was observed in some of the other houses by comparing the relative concentrations of nitrogen dioxide and the other pollutants in various parts of the house.

Moschandreas and his associates carried out a 2-yr air sampling program to characterize the indoor residential air environment.[12] Indoor air quality was monitored for continuous periods of

TABLE IV-15

Summary of Indoor and Outdoor Concentrations of Pollutant Gases at a Suburban Home near Hartford, Connecticut[a]

Sampling Period	Pollutant Gas	Average Concentration, $\mu g/m^3$					Indoor: Outdoor Ratio	
		Kitchen over Stove (1)	Kitchen 1 m from Stove (1A)	Living Room (2)	Bedroom (3)	Outside (4)	1:4	2:4
Spring-summer, 1973	NO_2	--	100	61	52	44	—	1.39
	NO	--	102	64	65	26	—	2.46
	CO	--	4,490	4,070	4,170	3,480	--	1.17
Fall-winter, 1973-1974, first half	NO_2	67	60	55	--	50	1.34	1.10
	NO	136	134	94	--	63	2.16	1.49
	CO	4,190	3,520	3,230	--	1,670	2.51	1.93
Fall-winter, 1973-1974, second half	NO_2	110	67	--	49	46	2.39	--
	NO	134	131	--	102	65	2.06	--
	CO	4,790	4,210	--	3,820	2,310	2.07	--

[a]Based on Cote et al.[6] House specifications: split level, two bedrooms, centrally located, well-ventilated kitchen, 2,000-ft^2 living area, gas-fired stove and central heating system; occupied by two (smoking) adults and two teen-aged children; home 6 yr old at time of tests.

approximately 14 d in each of five detached dwellings, two semidetached dwellings (townhouses), six apartment units, two mobile homes, and one school. Three of the dwellings were referred to as "experimental," because they were designed to conserve energy. The remaining dwellings were referred to either as "conventional" or by structural type. In addition, the residences were divided according to their cooking and heating fuel. These structures are in five metropolitan areas: Baltimore, Washington, D.C., Chicago, Denver, and Pittsburgh. The dwellings in Baltimore, Washington, D.C., and Chicago were monitored twice to obtain seasonal variations. Conclusions from the study pertaining to pollutants associated with combustion products (carbon monoxide, nitric oxide, and nitrogen dioxide) follow:

> Indoor CO concentrations are generally higher than corresponding outdoor levels in all residences monitored. High indoor concentrations may be attributed to . . . indoor CO emission sources, such as gas-fired cooking appliances, attached garages, faulty furnaces, and cigarette smoking. . . .
>
> The complexity of the dynamics involved in the establishment of an indoor-outdoor relationship is clearly illustrated in the interpretation of the data base generated for NO. From the perspective of NO indoor variation and under real-life conditions, three types of indoor environments have emerged: 1) houses with electric cooking and heating appliances; 2) houses that are heated by gas furnaces, yet serviced by electric cooking appliances; and 3) houses that are furnished with gas cooking and heating equipment. In houses equipped with gas cooking appliances, observed indoor NO levels are consistently higher than observed outdoor levels. Houses with gas furnaces but electric cooking appliances display higher NO indoor levels than outdoor levels, most of the time. However, there are time intervals interspersed throughout the monitoring period during which the observed NO outdoor levels surpass corresponding indoor levels. Indoor NO concentrations in totally electric homes are almost always lower than corresponding outdoor concentrations. . . .
>
> The residential environment often provides a shelter from high outdoor NO_2 levels. The three classes of residences identified in the interpretation of the NO data also manifest themselves in the study of NO_2. The data base collected for this project indicates that the hourly average indoor concentrations of NO_2 are almost always lower than the corresponding ambient levels in totally electric houses. Houses equipped with gas furnaces and electric cooking appliances also shelter their occupants, but to a lesser extent during peak ambient NO_2 levels. Totally gas residences do not appear to provide such protection. . . .

With the large data base generated by the study, Moschandreas and Stark[11] formulated, documented, and validated a numerical prediction model (see Chapter VI). A series of numerical simulations with the model showed that, under some conditions involving gas appliances, indoor carbon monoxide, nitric oxide, and nitrogen dioxide concentrations increase substantially. Under these same conditions (with the oven in use for 2 consecutive hours), carbon monoxide may reach concentrations over 35 ppm.

The influence of gas stoves on indoor nitrogen dioxide content was confirmed by Palmes and associates, who used integrating "personal" monitors.[15] Melia and associates used this same type of sampler in an epidemiologic study to show that nitrogen dioxide concentrations were significantly higher in homes with gas stoves than in those with electric stoves.[16]

Puxbaum measured nitric oxide and nitrogen dioxide in a kitchen and an adjoining room in a home with a gas stove and gas hot-water appliance and compared indoor and outdoor concentrations. He also compared the burning of natural and "town" gas and found that the concentrations of both nitrogen dioxide and nitric oxide were significantly higher during the natural-gas runs than during the town-gas burning.[17]

In their 1973-1974 study, Cote and co-workers measured rates of pollutant emission from gas appliances.[6] Table IV-16 presents their data from various burner configurations and operating modes for a new and an old stove. (These stoves produce low but continuous emission from the pilot light, whereas current trends are toward low-heat-input gas pilot lights and non-gas ignition systems, which will reduce this source of indoor air pollution.)

More recently, Traynor and co-workers studied, in detail, emission from a gas-fired stove.[23] Table IV-17 summarizes some of the data from this work. In the case of nitrogen dioxide, there was generally good agreement between the Cote and Traynor studies. No laboratory tests on electric stoves were carried out for comparison in these studies, although, in an earlier field study, Hollowell et al. did observe small increases in kitchen ozone concentrations with electric-stove use.[5]

Both Cote et al. and Hollowell et al. measured gaseous pollutant emission from top burners while water-filled pans of different materials were being heated. Cote et al. noted some minor effects on gas-flame emission, especially with a low flame.[6] For example, carbon monoxide concentrations were higher when an aluminum pan was in place, and nitrogen dioxide concentrations were marginally lower with all types of pans under low-flame conditions. Hollowell and co-workers, however, could find no appreciable differences in gaseous emission, regardless of the types of pans used.

In the study by Cote and co-workers, the efficiency of an exhaust hood in removing stove-generated pollutants was determined.[6] Removal efficiencies varied from 4% with the hood fan off to 49% with the fan at its highest speed setting. (The low removal efficiency with the fan off was the result of natural ventilation through the hood.) A recirculating hood with activated charcoal, as might be expected, had

TABLE IV-16

Summary of Pollutant Emission of Gas Appliances for Typical Operating Conditions (No Pans in Place)[a]

Appliance	Operation	Heat Input Rate, kcal/h	Pollutant Emission Factors, µg/kcal			Pollutant Emission Rates, mg/h		
			NO	CO_2	CO	NO	NO_2	CO
Older gas stove with cast iron burners	Pilot lights	150	45.3	54.6	419	6.8	8.2	62.9
	1 burner, high flame	2,700	92.6	51.8	382	250	140	1,031
	3 burners, high flame	6,780	117.0	72.8	475	793	494	3,220
	Oven, steady state	2,200	91.4	73.1	530	201	161	1,166
Newer gas stove with pressed-steel burners	Pilot lights	100	4.7	18.6	842	0.5	1.9	84.2
	1 burner, high flame	3,500	130.0	79.0	510	455	277	1,795
	3 burners, high flame	10,200	138.0	65.6	315	1,408	669	3,213
	Oven, steady state	2,200	77.9	50.4	1,620	171	111	3,564

[2]Based on Cote et al.[6]

TABLE IV-17

Summary of Gas-Stove Emission[a]

Pollutant	Oven[b] Emission, μg/kcal[d]	Oven[b] No. Experimental Runs	Top Burner[c] Emission, μg/kcal[d]	Top Burner[c] No. Experimental Runs
Gases:				
CO	950 (650-1,600)	6	890 (720-1,090)	4
CO_2	200,000 (195,000-205,000)	6	205,000 (196,000-217,000)	3
NO	29 (14-50)	11	31 (21-47)	4
NO_2	62 (44-74)	11	85 (69-100)	4
SO_2	0.8 (0.5-1.0)	11	0.8 (0.6-0.9)	4
HCN	1.8 (1.6-2.3)	3	0.07	1
HCHO	11.4 (9.9-14.2)	5	5.2 (2.0-12.0)	5
Particles (<2.5 μm):				
Carbon	0.13 (0.05-0.24)	9	0.90 (0.86-0.96)	4
Sulfur (as SO_4)	0.01	9	0.05 (0.01-0.08)	4
Total respirable mass	--	--	1.7 (1.0-2.6)	3

[a]Based on Traynor et al.[23]

[b]Oven operated for 1 h at 180°C (350°F).

[c]Operated with water-filled cooking pots.

[d]Ranges in parentheses.

little effect on gaseous pollutants; however, such a device may partially control organic gases and vapors responsible for odors and aerosols released during cooking. The effect of kitchen ventilation on carbon monoxide, nitric oxide, and nitrogen dioxide was also included in the study by Traynor et al.,[23] and their data on nitrogen dioxide are presented in Figure IV-5.

Other Indoor Combustion Sources

Several other combustion sources can affect indoor air quality. Although they tend to be site-specific and not as common as domestic heating and gas-stove operation, several are worthy of mention.

Water Heaters and Clothes Dryers. In two homes with gas water heaters, Traynor observed that indoor nitrogen dioxide concentrations were greater than outdoor (G. W. Traynor, Lawrence Berkeley Laboratory, personal communication). Initially, passive monitors were used to measure average indoor concentrations over 1 wk. Both homes were found to have increased indoor concentrations and were investigated in more detail with a continuous analyzer. In both cases, nitrogen dioxide entered the living space from the flue collar on top of the water heater, despite the fact that the appliances were designed to vent the combustion products outside. Similar considerations apply to gas clothes dryers.

Automobiles. Driving a car into or out of a basement or attached garage can strongly affect indoor air quality, depending on the configuration of the house and the routes for entering and leaving the garage. In a number of documented cases, occupants have inadvertently left a car running in a basement garage from which the resulting carbon monoxide drifted into the associated house or apartment and caused sickness or death of occupants. The most critical situation would occur in a basement garage during cold weather, when the "stack effect" of a heated house tends to draw air in from the garage and distribute the pollutants captured in the garage throughout the house. A variant of this situation is an apartment house or office building with a basement garage that has stairwells or elevator shafts that can distribute pollutants throughout the building.

Little information is available on the impact on indoor air quality of automotive exhaust emitted in garages. Yocom and associates[28] sampled one house with an attached garage and concluded that the design of the house (split-level) caused automotive emission from the garage to have a greater impact on carbon monoxide concentration than the gas stove.

Charcoal Broiling. Charcoal cooking is usually an outdoor activity; however, it is sometimes done in a fireplace, and some fireplaces even include a charcoal cooker. Depending on how such cooking is carried out and how well the fireplace draws, the resulting emission can enter the indoor environment. There appear to be no data

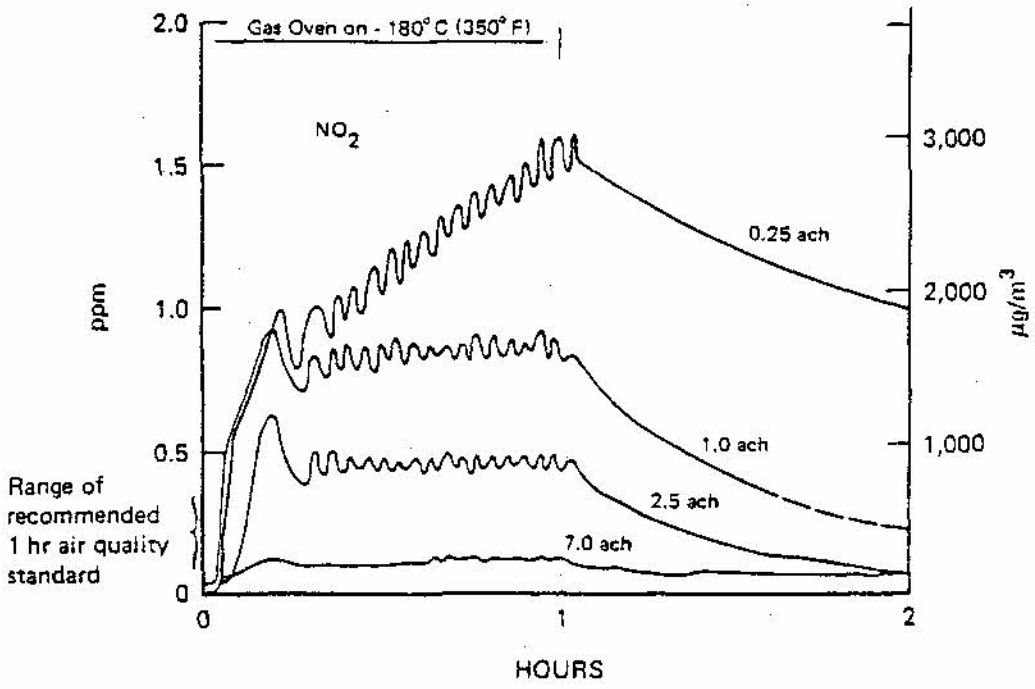


FIGURE IV-5 Nitrogen dioxide concentrations in test kitchens reported by Traynor et al.[23] as functions of use of gas oven with different kitchen exhaust rates.

on the direct impact of charcoal broilers on indoor air quality, but a recent study by Brookman and Birenzvige on exposure to air pollutants from "domestic combustion" sources provides some idea of how great these exposures might be.[5] They used personal samplers to measure hourly carbon monoxide and particulate matter in the outdoor environment while people were operating gas lawn mowers and chain saws and cooking with charcoal. A sampler adjacent to a charcoal cooker recorded that hourly carbon monoxide exposures ranged from 8 to 38 ppm. Analysis of the particulate filters for polynuclear aromatic organic matter showed that it was below the detection concentration of the screening method used.

Hobbies. A variety of hobbies involve combustion processes, for example, heating and soldering with an LPG torch and brazing or welding with an oxyacetylene torch. Depending on the location, extent, and duration of such activities, they could have an impact on indoor air quality. Under some circumstances, the exposure of those pursuing such hobbies to harmful contaminants could be substantial.

COMMERCIAL BUILDINGS

Only limited information is available on the effects of combustion sources on indoor air quality in commercial buildings. Nevertheless, any of the sources mentioned above can affect the quality of indoor air in commercial buildings as they do in residential buildings.

Spengler and co-workers determined carbon monoxide concetrations in several enclosed ice-skating rinks in Massachusetts[20] where gasoline-powered ice resurfacing machines are used; the concentrations were found to exceed 50 ppm regularly.

Large public garages where traffic congestion commonly occurs in confined spaces constitute another example of indoor air-quality problems in commercial buildings. There are many documented cases of drivers and passengers who experienced acute health effects (presumably from carbon monoxide) at times of heavy garage traffic. The most critical situation occurs at the end of the workday or at the completion of a major sporting event, when hundreds of cars line up with their engines running. Stankunas and associates carried out a study for the American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) to measure carbon monoxide in several parking garages.[21] They developed a model for calculating in-garage carbon monoxide concentrations on the basis of such variables as ventilation volume, outdoor ambient carbon monoxide concentration, initial carbon monoxide concentration in the garage, and garage volume.

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TOBACCO SMOKE

Nearly everyone is exposed to tobacco smoke at some time or other. Indirect exposure (i.e., exposure of nonsmokers) is referred to as "passive exposure," "passive smoking," or "involuntary smoking." The extent of the exposure is determined by the number of smokers one

associates with, their smoking habits, and the characteristics of the environment in which exposure occurs. Actual population exposures to tobacco smoke are therefore quite variable. For some, passive exposure to cigarette, pipe, or cigar smoke may occur routinely at work, in transit, or at home. For others, passive exposure may result only from infrequent encounters with smokers in public facilities.

Over 2,000 compounds have been identified in cigarette smoke; many are established carcinogens that appear primarily in the particulate phase. It is reasonable to assume that passive tobacco-smoke exposure is many people's principal source of exposure to many of these compounds.

Despite the overwhelming evidence on health effects on smokers, the impact of tobacco-smoke exposure on nonsmokers is not well documented. (The health effects of gases and particles emanating from tobacco combustion are summarized in Chapter VII.) Tobacco smoke irritates the eyes, nose, and throat and is annoying to nonsmokers, even in the presence of "adequate" ventilation. Annoyance increases with increasing smoke contamination and increasing dryness of air. Aside from the irritation and annoyance that it causes, smoking in confined spaces increases annoyance from odors and particle accumulation. Aldehydes and ketones produced by burning tobacco give rise to odors. Particles that adsorb and release organic vapors can be odor sources long after the tobacco is extinguished, and the lingering odors can be smelled by those not desensitized.

This section discusses indoor exposure to pollution resulting from cigarette-smoking. The factors of concern here are the number of people exposed to cigarette smoke, the composition of the gases and particles emitted, and the concentrations of pollutants encountered.

BACKGROUND

The number of people exposed to passive smoking, who might also be termed "involuntary smokers," is not known. However, given the number of people who smoke in the United States, some involuntary inhalation of tobacco combustion products from smoke-contaminated atmospheres by nonsmokers is unavoidable. Passive exposure to tobacco smoke will inevitably occur in any number of public or private activities.

In 1978, an estimated 54 million persons smoked 615 billion cigarettes. The prevalence of regular cigarette-smoking in the adult population declined from 42% in 1964 to 33% in 1978. Figure IV-6 plots the annual consumption, from 1950 to 1978, of cigarettes and filter-tip cigarettes per person aged 18 and over. The adult per capita consumption for 1978 is estimated at 3,965, which is the lowest recorded consumption since 1958. Surveys show that fewer men are smoking each year, but more women are smoking, particularly teen-agers. Figure IV-7 presents the proportion of U.S. men, women, and teen-agers that reported being regular smokers in 1974 and 1975. This figure implies that one of every three persons between the ages of 17 and 64 regularly smokes cigarettes. Association with adult males 35-44 yr old increases the likelihood that a person will be passively

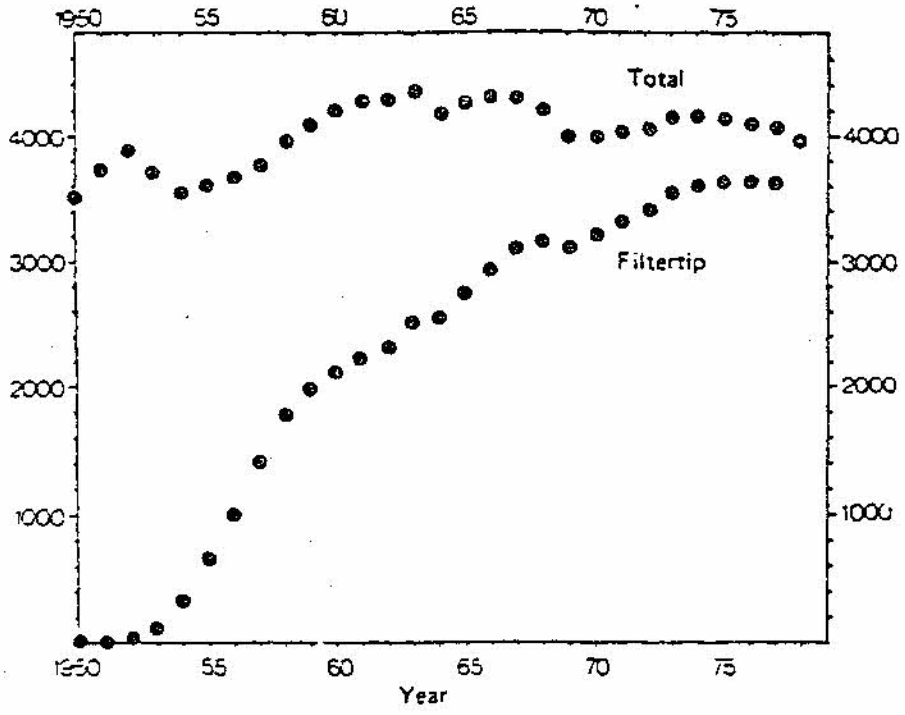


FIGURE IV-6 Annual consumption of cigarettes and filter-tip cigarettes per person aged 18 yr and over, 1950-1978. 1978 per capita consumption of cigarettes was 3,965, the lowest since 1958. Open circle, preliminary estimate. Reprinted from U.S. Department of Health, Education, and Welfare.[31(p.A-5)]

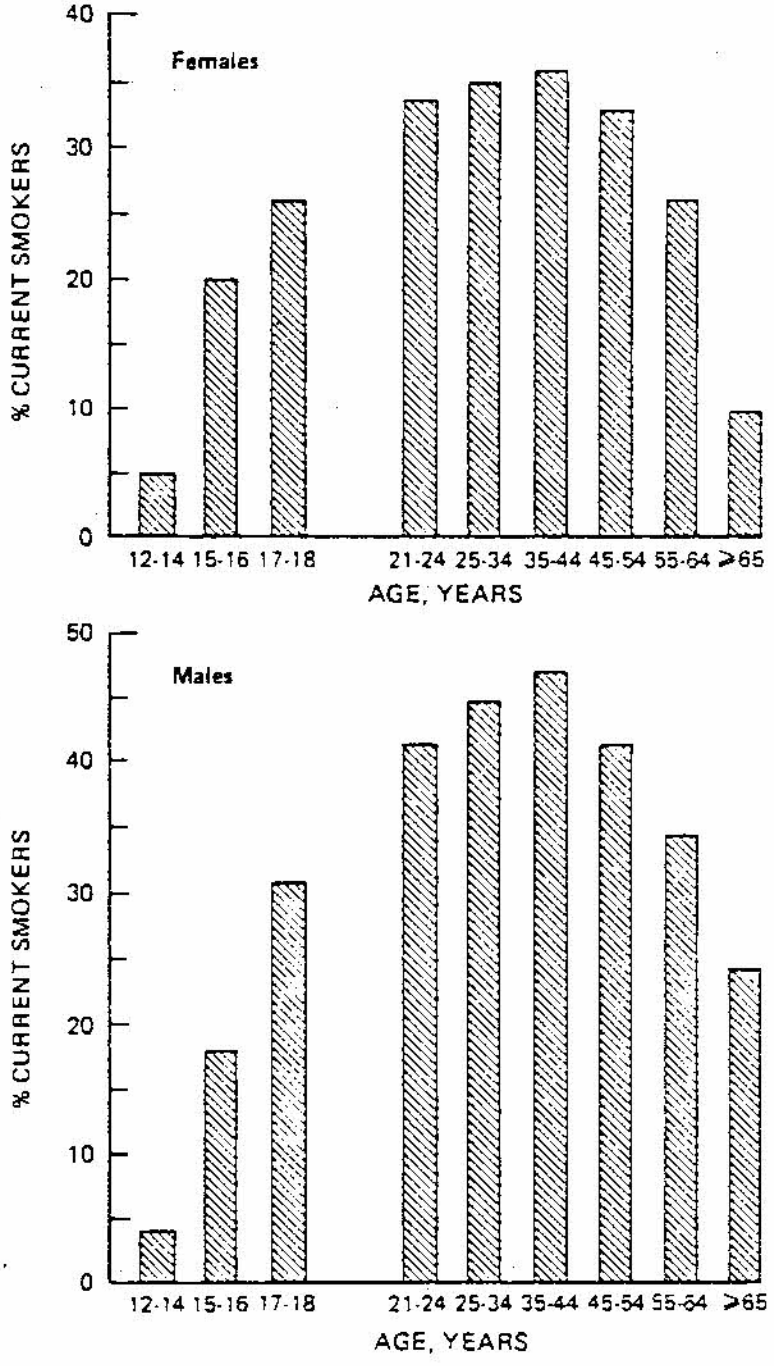


FIGURE IV-7 Percentage of regular smokers in population. Teenagers, 1974. Adults, 1975. Data from U.S. Department of Health, Education, and Welfare.[31](pp. A-10, A-14)

exposed to cigarette smoke. In 1974, the percentages of girls and boys 15-16 yr old who smoked were essentially the same. The percentage of teen-aged boys smoking, however, has been dropping since 1970, whereas the percentage of teen-aged girls smoking has increased dramatically.

Because cigarette smoke is ubiquitous, passive exposure to it will be encountered in offices, industrial facilities, homes, public sporting events, restaurants, transportation facilities, and innumerable other locations. Professional and technical workers have the lowest percentage of cigarette-smokers; laborers, craftsmen, and other "blue-collar" workers have the highest. Of the males in "blue-collar" occupations, 47% were smokers in 1975, whereas 36% of the males in "white-collar" employment were smokers. Females showed the opposite relationship; 34% of the "white-collar" females and 32% of "blue-collar" female workers smoked. In the 1975 survey, 40% of the women in the sample worked outside the home; of these, 33% were cigarette-smokers, compared with 27% of the housewives.

Information on passive exposure of nonsmokers to tobacco smoke has never been systematically obtained, except in a limited number of epidemiologic investigations (see Chapter VII). Data on populations exposed at home, at work, or in other locations must be estimated from surveys on smoking prevalence and epidemiologic studies. The National Center on Smoking and Health (NCSH) has conducted the most extensive surveys on American smoking habits. Marital status, educational achievement, and income are demographic variables associated with differences in smoking prevalence. NCSH information indicates that divorced or separated persons smoke more than those in any other marital-status group. For men, the highest prevalence occurs in middle-income and high-school-educated groups. For women, there is a more direct relationship between income and smoking, with more smoking in the higher-income groups.

The demographics of smoking prevalence do not indicate exactly what groups are passively exposed to tobacco smoke or where or when they are likely to be exposed. We do not have information on the percentage of the 80 million workers in the United States who are employed in an environment free of tobacco smoke. We do not know what percentage of America's 80 million residential units have smokers, but the results of some surveys are summarized below.

Some 54 million adult Americans smoke (33%), so we can infer that the number of homes and other residential units with smokers is substantial. Summaries of prevalence of homes with smokers taken from a study on air-pollution health effects support this contention. Table IV-18 summarizes the response to a questionnaire in the children's study. On the basis of 8,493 questionnaire responses obtained across six cities (75% response rate), 70% of the homes reported having at least one smoker. The percentages reporting at least one smoker ranged from 63% in rural Portage, Wisconsin, to 76% in a middle-income community in St. Louis. Lebowitz and Burrows[13] reported that 53.8% of the children in their Tucson study had smokers in their homes. Schilling et al.[25] reported that an average of 63% of the sampled homes in two Connecticut towns had smokers. Substantial regional variations in the percentage of homes with smokers may be expected,

TABLE IV-18

Percentage of Homes Reporting One or More Smokers[a]

Location	No. Responses	Proportion of Homes with Smokers, %
St. Louis, Mo.	1,922	76.1
Steubenville, Oh.	1,808	74.2
Kingston-Harriman, Tenn.	810	71.8
Watertown, Mass.	838	69.8
Topeka, Kans.	1,663	63.3
Portage, Wis.	1,452	62.5
Tucson, Ariz.	676	53.8
Two towns, Conn.	376	63.3

[a]Data from Ferris et al.,[8] Lebowitz and Burrows,[17] and Schilling et al.[25]

owing to geographic variations in social, demographic, and religious variables that are associated with differences in smoking prevalence.

Further stratification of the responses to the question of parental smoking by level of parental education confirms earlier NCSH surveys. In the Harvard six-city study,[8] of the homes in which neither parent had a high-school education, over 80% had at least one smoker. In homes in which one parent had graduated from college, only 50% had smokers.

CONTAMINANTS IN SMOKE

A distinction can be made between mainstream smoke and sidestream smoke in the contaminants evolved from tobacco combustion. Both smokers and nonsmokers are, of course, exposed to sidestream smoke. Mainstream smoke is undiluted and is pulled through the tobacco into a smoker's lungs. Sidestream smoke is directly from the burning tobacco. Depending on smoking behavior, burning temperature, and type of filter, the composition of mainstream smoke exhaled by a smoker varies substantially. From 50% to more than 90% of water-soluble compounds are removed from the mainstream smoke by the smoker, depending on the depth of inhalation and the time of breath-holding. The insoluble compounds show equal variability over a range of 20-70%.

A typical cigarette-smoker inhales mainstream smoke 8-10 times, for a total of 24-30 s of a total 12-min burn time for a cigarette.[19] There is no dispute that the concentrations of almost all constituents are far greater in mainstream than in sidestream smoke. However, given approximately 24 to 1 disparity in burning time (i.e., the sidestream smoke is produced during 96% of the total smoking time) and the difference in combustion conditions, it is not surprising that sidestream smoke is more important to the involuntary, passive smoker. The sidestream smoke can also be enriched in many compounds. Sidestream smoke and mainstream smoke have been characterized by many investigators.[2-5, 6, 18, 19] The mean particle size of fresh mainstream smoke is slightly greater than that of sidestream smoke. Exhaled-smoke particles are larger, on the average, than those in fresh mainstream or sidestream smoke, because of water absorption and coagulation. The same processes modify the size distribution of smoke particles as they age. If particles begin with a mass mean diameter less than 0.2 μm, they grow by agglomeration and water absorption to a mass mean diameter approaching 1 μm within minutes.

The passive smoker by no means receives a lung dose of smoke equivalent to that of the smoker. Several investigators have estimated the exposure of the nonsmoker in a smoke-filled environment as one-hundredth to one-tenth of the smoker's exposure.[12, 13, 22, 23] These estimates were based primarily on measurements of carbon monoxide, suspended particulate matter, or nicotine concentration extrapolated from reported values for mainstream smoke. Substantial evidence indicates that many substances are increased in sidestream smoke. Therefore, direct comparison on the basis of particle mass or concentration of a specific gas may not adequately describe a passive smoker's exposure to cigarette smoke.

The sidestream-to-mainstream ratios (s:m) of specific compounds range from 0.7 to 46. The ratios of vapor-phase compounds vary more than those of particulate-phase compounds. Although the composition of mainstream smoke from nonfiltered cigarettes is quite different from that from filtered cigarettes, the compositions of their sidestream smoke are essentially the same. Table IV-19 presents a compilation of the concentrations of some substances found in cigarette smoke, with s:m ratios. Values are given in mass per cigarette. Unless otherwise noted, the values refer to nonfiltered cigarettes. Sidestream cigarette smoke, because of the length of the burn and the burn temperature, is a more important source of local air contamination with many substances--such as carbon monoxide, nicotine, ammonia, and aldehydes--than mainstream smoke.

INDOOR CONCENTRATIONS OF PARTICLES AND VAPORS FROM CIGARETTE SMOKE

Cigarette-smoking in enclosed areas increases concentrations of particles and gases. Increased concentrations of carbon monoxide, nicotine, nitrosamines, and benzopyrene are among the most frequent. Pollution measurements of air contaminated with tobacco smoke can be conveniently divided among controlled chamber experiments and actual exposure conditions. In many of the controlled-setting experiments, carbon monoxide concentrations exceeding the 1-h NAAQS of 35 ppm have been reported (see summary table on chamber studies of cigarette-smoke exposure in U.S. Surgeon General's Report, Smoking and Health[11]). Without ventilation, indoor carbon monoxide concentrations are proportional to the amount of tobacco burned and inversely proportional to room volume. These studies indicate that carbon monoxide concentrations of 50-100 ppm can be obtained and that increased ventilation substantially reduces concentrations (see Figure IV-8). Chapter 11 of the 1979 Surgeon General's report tabulates the results of many of the chamber experiments (Table IV-19).

The experiments performed in controlled environments generally involved heavier smoking than is normally encountered. Of more interest are the observations of carbon monoxide and other pollutants in normal indoor locations. Several studies are summarized in Table IV-20. In general, the carbon monoxide concentrations were less than those in the controlled experiments, probably because of the heavier smoking. Taverns, bars, nightclubs, and restaurants have been the more frequently reported locations in assessments of the air-quality impact of cigarette-smoking. Concentrations less than 35 ppm for an hourly average have been reported, primarily because of mechanical or natural ventilation. However, studies have indicated that the 8-h NAAQS of 9 ppm could be exceeded in public facilities that permit cigarette-smoking. Elliot and Rowe[7] reported that carbon monoxide concentrations in public assemblies of 2,000-14,000 people were between 9 and 25 ppm--up to 4 times higher than background.

As a more direct measure of exposure, several investigators have measured COHb in nonsmokers' blood after their exposure to cigarette smoke. The results were as expected: modest increases in COHb.

TABLE IV-19

Composition of Mainstream and Sidestream Smoke

Characteristic or Compound	Concentration, mg/cigarette[a] Mainstream Smoke (1)	Sidestream Smoke (2)	Ratio, 2:1	Reference
General characteristics:				
Duration of smoke production, s	20	550	27.5	19
Tobacco burned	347	411	1.2	15
Particles, no. per cigarette	1.05×10^{12}	3.5×10^{12}	3.3	24
Particles:				
Tar (chloroform extract)	20.8	44.1	2.1	16
	10.2[b]	34.5[b]	3.4	16
Nicotine	0.92	1.69	1.8	16
	0.46[b]	1.27[b]	2.8	16
Benzo[a]pyrene	3.5×10^{-5}	1.35×10^{-4}	3.9	10
	4.4×10^{-5}	1.99×10^{-4}	4.5	18
Pyrene	1.3×10^{-4}	3.9×10^{-4}	3.0	10
	2.70×10^{-4}	1.011×10^{-3}	3.7	18
Fluoranthene	2.72×10^{-4}	1.255×10^{-3}	4.6	18
Benzo[a]fluorene	1.84×10^{-4}	7.51×10^{-4}	4.1	18
Benzo[b/c]fluorene	6.9×10^{-5}	2.51×10^{-4}	3.6	18
Chrysene, benz[a]anthracene	1.91×10^{-4}	1.224×10^{-3}	6.4	18
Benzo[b/k/j]fluoranthrene	4.9×10^{-5}	2.60×10^{-4}	5.3	18
Benzo[e]pyrene	2.5×10^{-5}	1.35×10^{-4}	5.4	18
Perylene	9.0×10^{-6}	3.9×10^{-5}	4.3	18
Dibenz[a,j]anthracene	1.1×10^{-5}	4.1×10^{-5}	3.7	18
Dibenz[a,h]anthracene, ideno-[2,3-ed]pyrene	3.1×10^{-5}	1.04×10^{-4}	3.4	18
Benzo[ghi]perylene	3.9×10^{-5}	9.8×10^{-5}	2.5	18
Anthanthrene	2.2×10^{-5}	3.9×10^{-5}	1.8	18
Phenols (total)	0.228	0.603	2.6	13
Cadmium	1.25×10^{-4}	4.5×10^{-4}	3.6	27
Gases and vapors:				
Water	7.5[c]	298[d]	39.7	13
Carbon monoxide	18.3	86.3	4.7	32
	—	72.6	—	26
Ammonia	0.16	7.4	46.3	20
Carbon dioxide	63.5	79.5	1.3	20
NO_x	0.014	0.051	3.6	20
Hydrogen cyanide	0.24	0.16	0.67	31
Acrolein	0.084	—	—	31
	—	0.825	—	26
Formaldehyde	—	1.44	—	26
Toluene	0.108	0.60	5.6	32
Acetone	0.578	1.45	2.5	32
Polonium-210, pCi	0.04-0.10	0.10-0.16	1-4	7

[a]Unless otherwise noted. [b]Filtered cigarettes.
[c]3.5 mg in particulate phase; rest in vapor phase.
[d]5.5 mg in particulate phase; rest in vapor phase.

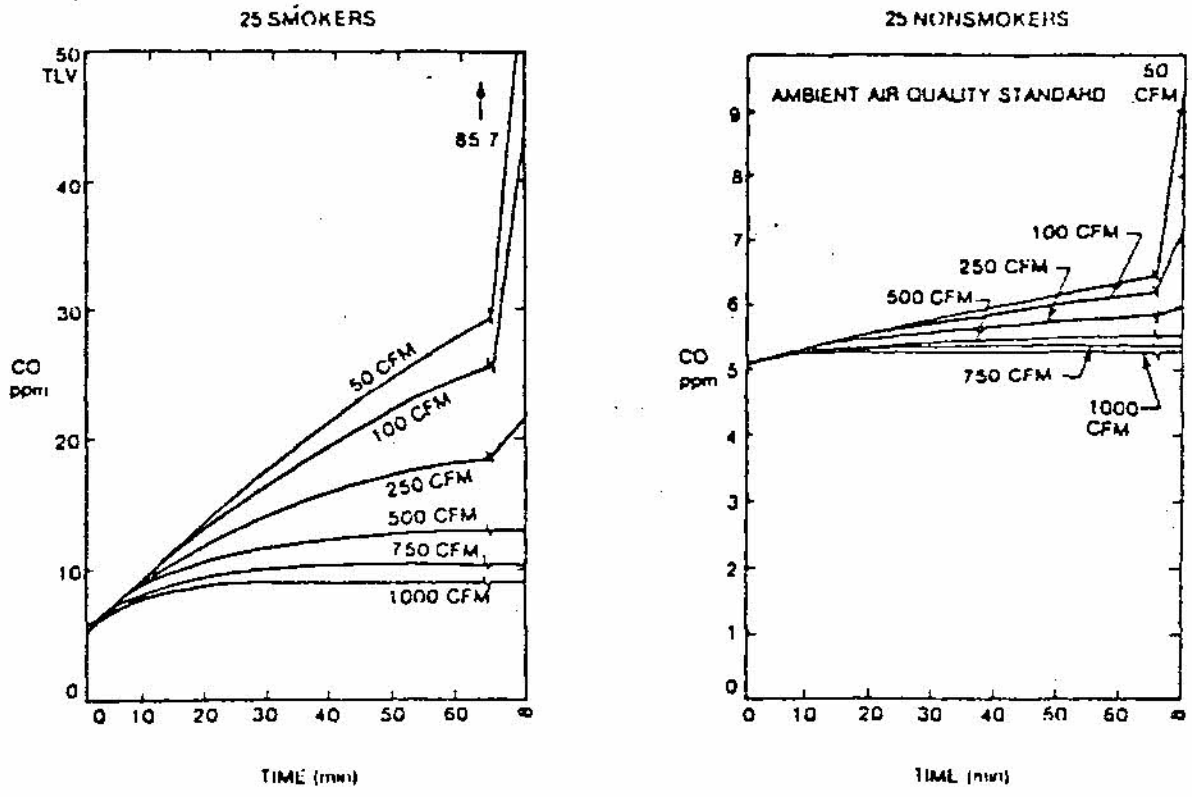


FIGURE IV-8 Calculated buildup of carbon monoxide under various conditions of ventilation and smoking. Calculated for a room of 3,000 ft^3 with 25 smokers on the left and for 25 nonsmokers on the right. TLV is the threshold limit value for carbon monoxide (50 ppm). CFM is ventilation in cubic feet per minute. Reprinted with permission from Galuškinová.[9]

TABLE IV-20

Measurement of Constituents of Tobacco Smoke Under Natural Conditions[a]

Reference, Location, and Dimensions	Ventilation	Amount of Tobacco Burned	Level of Constituent	
			Smoking Section	Other Control Section
Brunnemann and Hoffmann			dimethylnitrosamine	
Train 1 (Bar Car)	--	--	.13 ng/l	--
Train 2 (Bar Car)	--	--	.11 ng/l	--
Bar	--	--	.24 ng/l	--
Nightclub	--	--	[.09 ng/l]	--
Cano, et al.				
Submarines 66 m^3	Yes	157 cig per day	<40 ppm CO, 32 $\mu g/m^3$ nicotine	--
		94-103 cig per day	<40 ppm CO, 15-35 $\mu g/m^3$ nicotine	--
Chappel and Parker				
General public places	--	--	3.5 ppm CO	2.0 ppm CO
Government offices	--	--	2.5 ppm CO	2.5 ppm CO
Restaurants	--	--	4.0 ppm CO	2.5 ppm CO
Night clubs and taverns	--	--	13.0 ppm CO	3.0 ppm CO
Cuddeback, et al.				
Tavern 1	6 air changes per hour	--	12.5 ppm CO, .33 mg/m^3 TPM	--
Tavern 2	[natural (~1-2/hr)]	--	17 ppm CO, .98 mg/m^3 TPM	--
Elliott and Rowe				
Arenas	--	--	14.3 ppm CO, .367 mg/m^3 TPM	3 ppm CO, .068 mg/m^3 TPM

TABLE IV-20 (continued)

Reference, Location, and Dimensions	Ventilation	Amount of Tobacco Burned	Level of Constituent	
			Smoking Section	Other Control Section
Galuskinova				
Restaurant	--	--	.0002-.0046 mg/m^3 benzopyrene	--
Godin, et al.				
Ferry boat compartments	--	--	18.4 ± 8.7 ppm CO	3.0 ± 2.4 ppm C
Theater	--	--	3.4 ± 0.8 ppm CO	1.4 ± 0.8 ppm C
Harke				
Office building	air conditioned	--	<5 ppm CO	--
Office building	not air conditioned	--	<5 ppm CO	--
Room 78.3 m^3	--	3 smokers	15.6 ppm CO	--
Harke and Peters				
Automobile	35 km/hr speed, no ventilation.	4 cig	24.3 ppm CO	--
	80 km/hr speed, no ventilation.	4 cig	12.1 ppm CO	--
	30 km/hr speed, no ventilation.	4 cig	21.4 ppm CO	--
	30 km/hr speed, air jets open.	4 cig	15.7 ppm CO	--
	3 km/hr speed, air jets open & blower on.	4 cig	12.0 ppm CO	--

TABLE IV-20 (continued)

Reference, Location, and Dimensions	Ventilation	Amount of Tobacco Burned	Level of Constituent: Smoking Section	Level of Constituent: Other Control Section
Hinds and First			nicotine:	
Commuter train	--	--	.0049 mg/m^3	--
Commuter bus	--	--	.0063 mg/m^3	--
Bus waiting room	--	--	.001 mg/m^3	--
Airline waiting room	--	--	.0031 mg/m^3	--
Restaurant	--	--	.0052 mg/m^3	--
Cocktail lounge	--	--	.0103 mg/m^3	--
Student lounge	--	--	.0028 mg/m^3	--
Lefcoe and Inculet				
House	--	1 cig	48 x 10^6 particles per cubic foot	.9 x 10^6 particles per cubic foot
Szadkowski, et al.				
Offices	--	--	2.7 ppm CO	--
Sebben, et al.				
Night clubs	--	--	13.4 ppm CO [6.5-41.9]	9.2 ppm CO
Restaurants	--	--	8-28 ppm CO	--
Bus	--	--	7.3 ppm CO [6-14]	6.2 ppm CO
Slavin and Hertz				
Conference room	8 air changes per hour	--	8 ppm CO	1-2 ppm CO
	6 air changes per hour	--	10 ppm CO	1-2 ppm CO

TABLE IV-20 (continued)

Reference, Location, and Dimensions	Ventilation	Amount of Tobacco Burned	Level of Constituent: Smoking Section	Level of Constituent: Other Control Section
Seiff				
Intercity bus	15 air changes per hour	23 cig burning continuously	33 ppm CO	--
		3 cig burning continuously	18 ppm CO	--
U.S. Dept. Transportation, et al.				
Airplane flights:				
Overseas--100% filled	15-20 air changes per hour	--	2-5 ppm CO, <.120 mg/m^3 TPM	--
Domestic--66% filled	--	--	2 ppm CO, <.120 mg/m^3 TPM	--

[a]Reprinted from U.S. Department of Health, Education, and Welfare.[31](pp. 11-16--11-20)
cig = cigarettes; -- = unknown; TPM = total particulate matter.

Harke[11] showed a COHb increase from 0.9% to 2.1% after a 2-h exposure to carbon monoxide at 30 ppm. Aronow[1] reported similar results in patients exposed to smoke from 15 cigarettes smoked over 2 h in a 30.8-m^3 room without ventilation. It is possible that passive smoking was the reason that a portion of the nonsmoking population was reported by Stewart et al.[39] (in a national COHb survey) to have COHb of over 1.5%. But, given the range of carbon monoxide concentrations reported in smoke-filled indoor locations, it is unlikely that cigarettes alone contribute significantly to increased COHb in the nonsmoking population. Because carbon monoxide is slow to be removed from the environment, reducing fresh-air supplies to office buildings or public facilities that permit smoking would necessarily increase carbon monoxide concentrations.

Several other constituents of tobacco smoke have been measured indoors, including nicotine, acrolein, benzo[a]pyrene, nitrosamines, and aldehydes.

Under heavy-smoking conditions, acrolein is the only gaseous substance that has been shown to exceed threshold limit values established for industrial environments. Acrolein is found at 1-20 ppb in bars and restaurants; even to nonsensitive persons, these concentrations can cause annoying odors and eye and nose irritation.

The indoor concentrations of benzo[a]pyrene where smoking occurs are more ambiguous. Galuškinová[9] reported concentrations of 0.2-4.6 $\mu g/m^3$ in restaurants described as "smoky." These high concentrations may have been due to cooking; Elliot and Rowe[7] reported concentrations of 7-22 ng/m^3 in the presence of total suspended particles at 224-480 $\mu g/m^3$ in public arenas with smokers.

The nicotine concentration in air is an excellent indicator of cigarette smoke. A typical cigarette contains 2 mg of nicotine. Hinds and First[12] measured nicotine at 1-10.3 $\mu g/m^3$ in a number of public facilities, including cocktail lounges, transportation waiting rooms, trains, and buses. The submarine contains a unique environment for observing human exposure to cigarette smoke. Cano et al.[5] found nicotine in the urine of nonsmokers when the submarine environment had nicotine at 15-35 $\mu g/m^3$, and the nonsmokers' urinary nicotine concentration was only 1% of that of the smokers. It appears unlikely that the threshold limit value (500 $\mu g/m^3$) for exposure to nicotine in industrial environments would be exceeded in indoor locations with ventilation, although only a few studies have been reported.

Total suspended particles (TSP) and the fractions of respirable suspended particles (RSP) have been measured in the indoor environment in the presence of tobacco smoke. TSP concentrations of 50-400 $\mu g/m^3$ have been reported for integrated samples taken in public arenas, lounges, bus stations, and airplanes (see Table IV-20).

There are few reported measurements of RSP in the vicinity of tobacco-smoking. Repace and Lowrey[21] reported on 2-min RSP samples taken in 20 indoor environments where smoking is permitted (outdoor measurements were also reported). The indoor RSP concentrations ranged from 86 to 697 $\mu g/m^3$. These results are consistent with residential measurements of 24-h RSP concentrations reported by Spengler et al.[29] Daily indoor concentrations of RSP frequently

exceeded 200 $\mu g/m^3$ in homes with cigarette-smokers. Aggregating the data obtained from a study of 69 homes in six cities reveals that the indoor and outdoor concentrations in the 38 nonsmoking homes are essentially equivalent (24 $\mu g/m^3$ indoors versus 22 $\mu g/m^3$ outdoors). In the 22 homes with only one cigarette-smoker, the mean concentration indoors was 43 $\mu g/m^3$; the nine homes with two or more smokers had a mean concentration of 75 $\mu g/m^3$. These data, collected over a 3-yr period, are presented in Figure IV-9, which shows monthly mean RSP concentrations outside and inside homes without smokers, with one smoker, and with two or more smokers. The data clearly illustrate the contribution of cigarette-smoking to indoor particle concentrations. (The effects of pollution control on indoor concentrations of particles generated from tobacco-burning are discussed in Chapter IX.)

In recent investigations of personal exposures to respirable particles by Spengler et al.,[28] passive smoking was shown to be an important source. Volunteers in Topeka, Kansas, carried portable monitors for 12-h periods on 15 sampling days. The mean RSP concentration of samples where participants reported passive cigarette-smoke exposure for some time during the day was 40 $\mu g/m^3$. The nonsmoking, nonexposed participants had an overall mean concentration of approximately 22 $\mu g/m^3$, and the outdoor concentrations averaged less than 15 $\mu g/m^3$.

CONCLUSIONS

Tobacco-smoking indoors can contribute to or cause increased concentrations of respirable particles, nicotine, carbon monoxide, acrolein, and many other substances in the smoke. Many experimental and "real-life" measurements have demonstrated that where ventilation is low or nonexistent, indoor pollutant concentrations can exceed ambient-air quality standards and industrial standards. The indoor concentrations have been shown to depend on the number of smokers, how the tobacco is smoked (cigarette, pipe, or cigar), the room volume, the volume of fresh-air makeup, the efficiency of the air-cleaning apparatus, and the effectiveness of air mixing in the room. The absorbing characteristics of building and furnishing materials can affect the concentration. It has been demonstrated that the use of makeup air (for ventilation) that has lower concentrations of the contaminants effectively lowers the concentrations of carbon monoxide and other pollutants in tobacco smoke. Nevertheless, high respirable and total suspended particle concentrations have been noted even in the presence of "adequate" ventilation. Daily concentrations of respirable particles can exceed proposed ambient-air quality standards for TSP in homes with smokers.

Although it needs to be documented, tobacco smoke may be the most important source of exposure of nonsmoking populations to benzo[a]pyrene, nicotine, and other compounds in nonindustrialized areas. It must be noted, however, that there are other important sources, both indoors and outdoors, of many of the pollutants produced

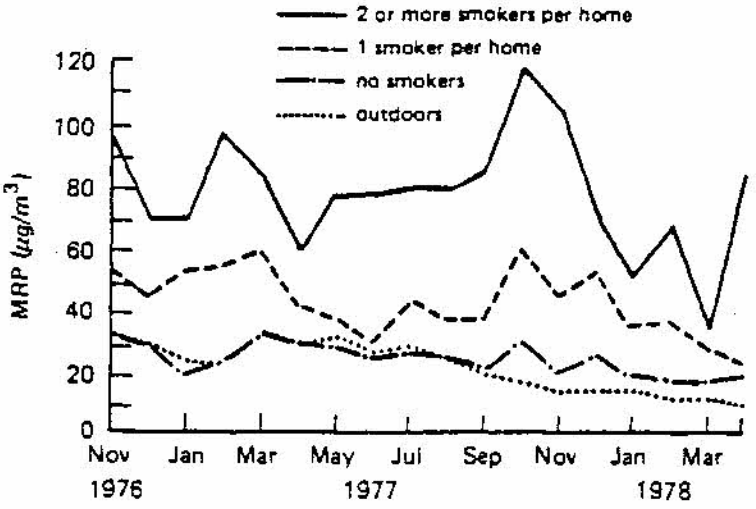


FIGURE IV-9 Monthly mean respirable particle concentrations. O, outside. X, inside nonsmoker homes. 1, inside homes with one smoker. Solid circles, inside homes with two or more smokers. Sample represents 80 homes across six cities (approximately 10-15 homes per city). Reprinted with permission from Spengler et al.[29]

by the burning of tobacco. And it is important to point out that direct exposure of a smoker is an order of magnitude greater than the passive exposure of a nonsmoker.

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ODORS

Some substances in the indoor environment make their presence known primarily by their ability to evoke odor sensations. These substances generally arise from humans or their activities. A room full of people, for example, will invariably have "occupancy odor." A characteristic odor like this generally emerges from a mixture of many organic substances, each present at a low concentration. Some of the individual constituents might cause greater concern if present alone at much higher concentrations; as building blocks of a composite, relatively benign odor, they receive attention only as odorants. Thus, the smell of the indoor environment is a measure of environmental quality. The remarkable sensitivity of olfaction encourages this approach. That is, treating low-concentration organic contaminants on the basis of their olfactory impact usually places stringent requirements on the quality of the indoor air. With some notable exceptions (e.g., the presence of carbon monoxide or mercury vapor), an indoor environment that is odorless contains air of healthful quality.

Completely odorless conditions occur indoors only rarely. Weak odors may go unnoticed or may be tolerated, particularly after persons have had the opportunity to remain in a space for a while. The lability of olfaction, evident in this phenomenon of adaptation, may impair the credibility of the nose as an air-quality indicator. Its sensitivity, often as good as or better than that of the most sensitive instruments, offers some compensation for its functional instability.

This section deals with how odors arise, how human beings perceive them, and how their status as "perceived" contaminants determines the means to cope with them. The stringent air-quality standard effectively imposed by high olfactory sensitivity makes odorants a target of particular interest in any effort to keep indoor air quality high in an energy-conscious society.

SOURCES

Occupancy Odor

Most buildings exist to hold people, in some cases many people. With the exception of churches, structures built specifically to hold many people were relatively rare until the nineteenth century. Before then, people avoided crowded places because of possible contamination--it was believed that crowded places, with their odors, served as breeding grounds of disease. The odors were often blamed for the spread of infection.[27] This attitude makes some sense, in view of the imperceptibility of the actual agents of contagion. Even at the end of the nineteenth century, many people still found it difficult to accept the notion that contagion is primarily fingerborne, rather than airborne. Belief in the airborne route carried no evident penalty for the layman, merely some inconvenience. Hence, the notion that bad-smelling air indoors signaled unhealthful conditions could carry on undisturbed. Through the burning of incense, churches had long seemed to give credence to the idea that a good (i.e., pleasant) odor would purify the air and thereby protect against illness. Such a practice actually reflected a predominant view of pre-nineteenth-century medicine.[53]

Only in the second half of the nineteenth century did the toxicity of the body effluvia responsible for occupancy odor receive scientific attention. In a common type of laboratory experiment on the matter, animals breathed air previously breathed by other animals, or received liquid injections of condensed organic materials from previously breathed air. In the experiments on "rebreathing," the animals sometimes developed infections or other difficulties; but, despite such occurrences, experimenters could not point indisputably to any harmful effects of the organic materials in previously breathed air.[12] Experiments on injections of condensed materials yielded essentially the same result: no consistent hazard. At the close of the nineteenth century, the issue seemed more or less settled. On the basis of both a review of available literature and their own experiments, Billings *et al.*[12] concluded that

> [it is] very improbable that the minute quantity of organic matter contained in the air expired from human lungs has any deleterious influence upon men who inhale it in ordinary rooms, and, hence, it is probably unnecessary to take this factor into account in providing for the ventilation of such rooms. . . . The discomfort produced by crowded, ill-ventilated rooms in persons not accustomed to them is not due to the excess of carbonic acid, nor to bacteria, nor, in most cases, to dusts of any kind. The two great causes of such discomfort, though not the only ones, are excessive temperature and unpleasant odors. . . . The cause of the unpleasant, musty odor which is perceptible to most persons on passing from the outer air into a crowded, unventilated room is unknown; it may, in part, be due to volatile products

> of decomposition contained in the expired air of persons having decayed teeth, foul mouths, or certain disorders of the digestive apparatus, and it is due, in part, to volatile fatty acids given off with, or produced from, the excretions of the skin, and from clothing soiled with such excretions. It may produce nausea and other disagreeable sensations in specially susceptible persons, but most men soon become accustomed to it, and cease to notice it, as they will do with regard to the odor of a smoking-car, or of a soap factory, after they have been for some time in the place. The direct and indirect effects of odors of various kinds upon the comfort, and perhaps also upon the health, of men are more considerable than would be indicated by any tests now known for determining the nature and quantity of the matters which give rise to them. (pp. 24, 26-27)

This statement would prompt little dispute today.

A quarter-century after the experiments of Billings and colleagues, a New York State commission focused on ventilation requirements for occupied classrooms.[66] Tests of such varied functions and indexes as comfort, body temperature, intellectual performance, motivation, respiration, metabolism, condition of the nasal mucosa, frequency of colds, blood pressure, hematocrit, appetite, and rate of physical work uncovered no cause for medical concern under normal conditions of occupancy. This 8-yr effort reinforced notions that control of occupancy odor should figure prominently in indoor-air quality control, but the justification had to rest on grounds of comfort, rather than on grounds of health. The first truly quantitative studies of ventilation requirements started with the premise that ventilation primarily must control occupancy odor.

Tobacco Smoking. Throughout the twentieth century, the air in occupied rooms has commonly been smoky. Mainstream cigarette smoke contains approximately 3,000 gaseous constituents,[76] and these and associated particulate matter may both constitute health hazards for occupants. To add insult to injury, tobacco smoke forms the most annoying and persistent indoor odor nuisance.[54] A survey of professional ventilating engineers placed it well ahead of the next two most disturbing indoor odorous contaminants, occupancy and cooking odors. Its severity as a nuisance derives from its properties: it is an apparent allergen for some persons; it is an eye, nose, and throat irritant for most persons; it is an odorant; it is a soiling agent; and it is a stimulus for chest discomfort in persons with angina pectoris.[57,71] Its "tar" content causes it to adsorb strongly to surfaces. After adsorption, it desorbs slowly and thereby promotes so-called secondary sources of odor. In general, such sources concentrate previously airborne odorants on their surfaces (e.g., air-conditioning coils) or in their interstices (e.g., fabric). When the adsorbed odorants desorb, they often have an odor character somewhat different from that of the parent contaminant, and this generally seems true of tobacco smoke.[54]

Tobacco-smoke odor increases in intensity and unpleasantness immediately after active smoking has ceased and after the particulate-vapor complex has adsorbed to surfaces.[79] Figure IV-10 demonstrates how odor increased in intensity during a period after cigarettes were extinguished in an unventilated room. During this time, the odor character changed from pungent and burnt to stale and sour. This presumably reflected some chemical instability of the airborne matter. The contaminants of mere occupancy also seem somewhat unstable; but, unlike tobacco odor, occupancy odor diminishes rapidly and dramatically with time (see Figure IV-10).

Cooking. One brand of cigarettes differs from another in type and blend of tobacco, type of additives sprayed on the tobacco or paper, and various other characteristics, such as porosity of paper and temperature of the ember.[82] In spite of these variations, all cigarettes give rise to an odor readily identifiable perceptually as tobacco odor.[19] In similar fashion, cooking gives rise to perceptually characteristic odors. These do not possess quite the simple integrity as a perceptual class as does tobacco-smoke odor. Some cooking odors constitute more serious nuisances than others. Some (e.g., cabbage odor) are generally considered disagreeable, whereas others (e.g., baking odors) are generally considered inoffensive. Some vapors (e.g., those from deep frying) adsorb tenaciously to surfaces and thereby become long-term contaminants. During initial generation, such vapors may evoke pungency, as well as odor; whether the pungency derives from organic gases generated by the reaction of the oil with the food, from inorganic gases generated by combustion, or from particulate matter remains unknown, but deserves attention.[80]

Bathroom and Waste Odors. In many buildings, exhaust hoods remove cooking odors at the site of generation. In some residences, cooking odors may have no such easy route of egress. Ductless range hoods, equipped with aluminum-mesh filters and carbon filters, sometimes serve as substitutes for exhaust ducts and true exhaust fans. Similarly, ductless bathroom "ventilators" have begun to see some use in homes. Nevertheless, most commercial, institutional, and industrial buildings exhaust bathroom air to the outside. In theory, recirculation of properly filtered bathroom air in nonresidential buildings offers an opportunity for great energy savings. In practice, the putative need to eliminate moisture before recirculation and the possible, if only occasional, breakthrough of contaminants through any filtration system limit the prospects for recirculation.[83] People will tolerate inadequate control of cooking odors (e.g., operation of a ductless range hood with a spent carbon filter) much more readily than inadequate control of bathroom odors.

Odor Control. Elimination of the odor source, local exhaust, general ventilation, and filtration (usually adsorption) are the principal ways of controlling indoor malodors physically. In some places, such as bathrooms and smoking areas, generation of malodor may exceed the limits of physical control and persons in charge of

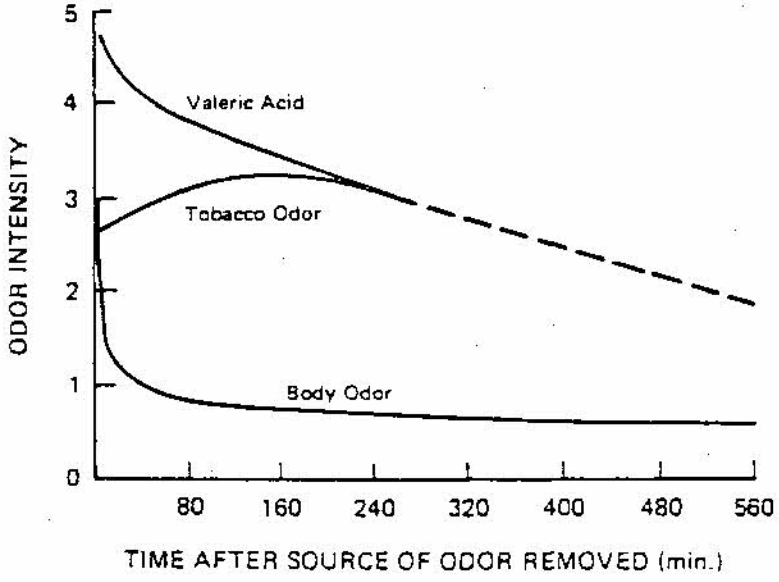


FIGURE IV-10 Decay of odor in still air in an unventilated chamber after an open flask of valeric acid had been removed, five cigarettes had been smoked, and a number of nonsmoking occupants had left the chamber ("body odor"). Odor judgments were made by observers who entered momentarily from time to time. Reprinted with permission from Yaglou and Witheridge.[79]

maintenance resort to commercially marketed odor counteractants.[59] Such products generally comprise a fragrance base made up of many aroma chemicals and possibly a single proprietary "active" ingredient. Typically, a manufacturer claims that the product has eliminated the objectionability or diminished the intensity of some standard malodor in laboratory tests. The claim invariably has some validity, because all odorants, including malodorants, can influence the perception of other odors. This rule forms the foundation of practical perfumery.[13] Basic research in olfaction supports the notion that a gas-phase mixture of components generally has an odor less intense than the total of the separate odors of the unmixed components,[20] perhaps less intense than some of its constituents alone--an extreme case of perceptual hypoadditivity.[10 23] The matter of alterations in odor quality (character) has received little attention in the scientific laboratory. The perfumer knows through experience how to manipulate or blend malodorants to produce acceptability. Hence, some fruity-smelling natural essences (always mixtures of constituents) may contain some "subsurface" putrid-smelling constituents. The perfumer may therefore blend a "deodorizing" fragrance that will assimilate a putrid-smelling contaminant into a fruity complex. When unable to anticipate the particular malodorous quality of interest, the perfumer generally blends a fragrance with a nondescript, unnameable odor character. Such a broad-band masker may assimilate some malodors readily, but at the very least adds perceptual "noise" to the maldodorous environment. Eventually, the usual or frequent occupants of a space may come to smell the malodorant through the olfactory noise. Persons who ride airplanes often, for instance, eventually find that the smoking area smells strongly of both tobacco smoke and masking agent.

It might seem that deodorizing products are used commercially only in special locations (e.g., bathrooms) or only on the occasions of uncontrollable malodorous emission (e.g., in the case of water damage to upholstery). In fact, such products, under the generic name of "reodorants," appear in virtually every cleaning product (e.g., degreasers, detergents, soaps, and fabric shampoos) and in many other materials (e.g., fabrics, plastics, floor finishes, and carpets). Reodorants are sometimes used to cover up undesirable ambient odors (e.g., mildew in damp spaces) and often to cover up the intrinsic odors of manufactured products themselves (e.g., formaldehyde in permanent-press fabrics). No matter what their purpose, reodorants and odor counteractants have become permanent parts of the indoor environment.[59]

Building Materials and Furnishings

Almost any object indoors may serve as a primary or secondary source of odor. Accordingly, the list of indoor odorous contaminants could go on indefinitely. In fact, Jarke[58] identified over 200 organic constituents in residences under conditions designed to minimize active generation of contaminants. Mølhave and Møller[60]

identified a similar number and noted that only six of 46 dwellings seemed odorless.

The identities of the more common indoor contaminants led Jarke to conclude that notable more or less permanent sources include food, plants, bodies, dry-cleaned clothes, cosmetics, household products, attached garages, heating systems, new furniture, carpeting, and redecorated surfaces. Chemical contamination is apprently much higher in new than in old homes.[60] New materials require a considerable amount of time to "cure" before the off-gassing of volatile odorous substances diminishes to an imperceptible point. One of the most notorious of the odorous contaminants in new buildings is formaldehyde.[8] It emanates from chipboard, panel adhesive, carpet backing, vinyl wall-covering, resin-treated fabrics, and urea-formaldehyde foam insulation. In some Danish homes, the concentration of gas-phase formaldehyde has exceeded threshold limit values for occupational exposure.[5]

Although formaldehyde has an odor, it has developed a reputation as an olfactory anesthetic.[52] The reason for its putative anesthetic properties has not been fully studied. Conceivably, its irritant properties play a role in the anesthetic phenomenon. Cain and Murphy[26] have reported that an irritant can immediately diminish and actually block olfaction. This effect seems to occur through interplay between sensory activity in the trigeminal nerve system and sensory activity in the olfactory nerve system. The trigeminal nerve mediates all cutaneous sensations of the face, as well as the pungency, irritation, warmth, cooling, and pain that can arise from chemical stimulation of mucosal tissue in the nose, mouth, and eyes. The evidence suggests that the interaction between odor and irritation takes place in the central nervous system and, hence, that it would hold true for virtually all combinations of odorous and irritating stimuli. It seems relevant that various successful deodorizers have contained irritants or pungent materials. For example, one commonly used deodorizer of the wick type contained formaldehyde, and other types of deodorizers contained other aldehydes. Ozone, a pungent gas, has long had a reputation as a deodorizer, even at concentrations too low to eliminate malodors through oxidation.[73] The "fresh-air" smell that deodorizers sometimes are claimed to produce can stem from the pungency produced by these substances at low concentrations in the product. Fresh Arctic air generally contains noticeable amounts of ozone. Hence, the association of pungency with "fresh air" has some basis in common experience. Nevertheless, the deliberate addition, even at low concentrations, of products that are irritating has questionable justification.

Odors That Enter from Outdoors

In theory, ventilation dilutes and displaces contaminants that are generated indoors. Bringing in odor-contaminated air for use in ventilation to reduce odors obviously can defeat the purpose. Odorous outside air is encountered in areas of great industrial pollution or

where micrometeorologic conditions allow entrainment of emitted substances from local sources into intake vents. In recognition of the possibility that the air used for ventilation sometimes fails to meet normal standards of quality, the American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) specified, in Standards for Natural and Mechanical Ventilation,[6] that intake air should meet both objective and subjective criteria for cleanliness. A draft revision of this standard contains a list of notable outdoor contaminants, some odorous and some nonodorous, and concentrations not to be exceeded (see Table IV-21). Adherence to these criteria will not guarantee the odorlessness of air, but will probably minimize difficulties in polluted regions.

Odors may also be generated in the air delivery system itself. Berglund and Lindvall[11] discovered cases where the air supplied to a room had a higher degree of odorant contamination than the air already in the room. That can occur when air-to-air heat exchangers, installed to conserve energy, allow exchange of organic materials between exhaust and makeup air. Another source may be the accumulation of odorous materials on roughing filters and cooling coils and in humidifiers in the intake system.

MEASUREMENT OF ODOR

Odors have various attributes: intensity, quality (character), affective charge (acceptability-objectionability), and duration. Most environmental odorants are mixtures of substances. Hence, intensity and character generally represent the net action and nonlinear perceptual combination of various constituents. Figure IV-11 depicts a gas chromatogram of a sample of odorous air (perspiration odor). In this case, the odorous sample is split into two streams after it passes through the column of the chromatograph.[12] One stream goes to a flame ionization detector and the other to a sniffing port, where the experimenter can note the odor character associated with the various chromatogram peaks. Such an odor-annotated chromatogram, called an "odorogram," can help in deciding how many peaks (constituents) seem odor-relevant, whether a few constituents seem particularly redolent of the unfractionated sample, and whether there is a relationship between the height of a peak and the magnitude of its odor. Often, a barely detectable peak is related to a strong odor and an enormous peak to a barely detectable odor. This situation reflects the nonuniform sensitivity of the nose; some substances stimulate at much lower concentrations than others. Table IV-22 shows, for instance, that ethyl acrylate stimulates at a concentration six orders of magnitude below that of ethylene and five orders of magnitude below that of acetone.[16] Odor science has long sought, with limited success, to account for such large disparities in stimulating efficiency.[15,20]

TABLE IV-21

Ambient-Air Quality Standards for Notable Contaminants Unregulated by Federal Clean Air Act[a]

Contaminant[b]	Long-Term Standard		Short-Term Standard	
	Concentration	Period	Concentration	Period[c]
Acetone*	7 mg/m^3	24 h	24 mg/m^3	30 min
Acrolein*	--	--	25 µg/m^3	C
Ammonia*	0.5 mg/m^3	1 yr	7 mg/m^3	C
Beryllium	0.01 µg/m^3	30 d	--	--
Cadmium	2.0 µg/m^3	24 h	--	--
Calcium oxide (lime)	--	--	20-30 µg/m^3	C
Carbon disulfide*	0.15 mg/m^3	24 h	0.45 mg/m^3	30 min
Chlorine*	0.1 mg/m^3	24 h	0.3 mg/m^3	30 min
Chromium	1.5 µg/m^3	24 h	--	--
Cresol*	0.1 mg/m^3	24 h	--	--
Dichloroethane*	2.0 mg/m^3	24 h	6.0 mg/m^3	30 min
Ethyl acetate*	14 mg/m^3	24 h	42 mg/m^3	30 min
Formaldehyde*	--	--	150 µg/m^3	C
Hydrochloric acid*	0.4 mg/m^3	--	3 mg/m^3	30 min
Hydrogen sulfide*	40-50 µg/m^3	24 h	42 µg/m^3	1 h
Mercaptans*	--	--	20 µg/m^3	1 h
Mercury	2 µg/m^3	24 h	--	--
Methyl alcohol*	1.5 mg/m^3	24 h	4.5 mg/m^3	30 min
Methylene chloride*	20 mg/m^3	1 yr	150 mg/m^3	30 min
	50 mg/m^3	24 h	--	--
Nickel	2 µg/m^3	24 h	--	--
Nitrogen monoxide	0.5 mg/m^3	24 h	1 mg/m^3	30 min
Phenol*	0.1 mg/m^3	24 h	--	--
Sulfates	4 µg/m^3	1 yr	--	--
	12 µg/m^3	24 h	--	--
Sulfuric acid*	50 µg/m^3	1 yr	200 µg/m^3	30 min
	100 µg/m^3	24 h	--	--
Trichloroethylene*	2 mg/m^3	1 yr	16 mg/m^3	30 min
	5 mg/m^3	24 h	--	--
Vanadium	2 µg/m^3	24 h	--	--
Zinc	50 µg/m^3	1 yr	--	--
	100 µg/m^3	24 h	--	--

[a]Reprinted with permission from ANSI/ASHRAE.[1] Concentrations listed should be corrected to standard conditions--25°C and 760 mm Hg.

[b]Contaminants marked with an asterisk have odors at concentrations sometimes found in outdoor air; concentrations listed do not necessarily result in absence of odor.

[c]C, ceiling (maximal allowable concentration).

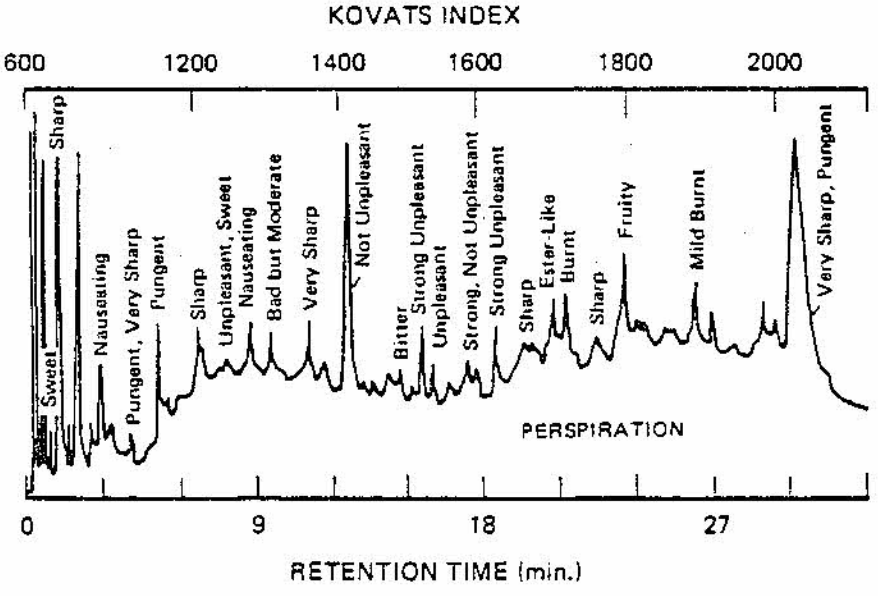


FIGURE IV-11 Odor-annotated gas chromatogram, called an "odorogram." Test vapor was human perspiration. Annotations refer to odor qualities noted by an observer when various constituents (represented by peaks) eluted from the chromatographic column. Reprinted with permission from Dravnieks.[32]

TABLE IV-22

Odor Threshold and Quality of Various Petrochemicals[a]

Compound	Threshold Concentration, ppm			Quality
	Absolute	50% Recognition	100% Recognition	
Acetone	20.0	32.5	140	Sweet-fruity
2,6-Butanol	0.30	1.0	2.0	Rancid-sweet
Di-N-butylamine	0.08	0.27	0.48	Fishy-amine
Diethylamine	0.02	0.06	0.06	Musty-fishy-amine
Ethyl acetate	6.3	13.2	13.2	Sweet-ester
Ethyl acrylate	0.0002	0.00030	0.00036	Sour-pungent
Ethylene	260	400	700	Olefinic
N-Ethyl morpholine	0.08	0.25	0.25	Ammoniacal
Isobutyl acetate	0.35	0.50	0.50	Sweet-ester
Isobutyl acrylate	0.002	0.009	0.012	Sweet-musty
Methanol	4.26	53.3	53.3	Sour-sharp
Methylethylketone	2.0	5.5	6.0	Sweet-sharp
2-Methyl-5-ethyl pyridine	0.006	0.008	0.010	Sour-pungent
2,4-Pentanedione	0.01	0.020	0.024	Sour-rancid
Propanal	0.009	0.040	0.080	Sweet-ester
Propionic acid	0.028	0.034	0.034	Sour
Propylene	22.5	67.6	67.6	Sharp-amine

[a]Data from Hellman and Small.[46]

Sample Collection

Procedures used to collect environmental samples for odor analysis have begun to approach standardization. Figure IV-12 displays a collector that contains the porous polymer material Tenax GC.[35] Use of this adsorbent material avoids the need for solvents in collection and analysis of air samples. A 2-L sample drawn through the collector typically retains organic materials in a quantitatively faithful fashion if their molecules have more than about six carbon atoms. For analysis of its contents, the collector is connected to a gas chromatograph, where flash-heating desorbs the contents into a stream of inert carrier gas. New techniques, such as high-pressure capillary-column chromatography, allow greater resolution and sensitivity than could be achieved with packed columns of the sort used to analyze the sample in Figure IV-11. Unfortunately, there is some incompatibility between capillary injection and the odorogram procedure.

Psychophysical Analysis

The standardization that has evolved in the collection of samples has extended also to psychophysical procedures for evaluation of environmental odors. The choice of procedures obviously depends on the question of interest. A variety of techniques are used in the laboratory, and a considerable number of techniques compete for attention in the field. Nevertheless, techniques that have recently emerged from activities of the ASTM Committee on Sensory Evaluation seem reasonably stable and precise and therefore legitimate for use as standard techniques. This section highlights these methods for the assessment of important attributes of odors.

Odor Character. The odor of any substance can be described precisely by only one term--the name of the substance itself. Stated otherwise, the only precise name for the odor of a lemon is "lemon odor," for the odor of a rose, "rose odor," and for the odor of a goat, "goat odor." Unlike colors, tastes, and sounds, odors have never given rise to their own glossary.[17] Moreover, people often find it difficult to retrieve the names of even familiar odors, not to mention describing unfamiliar ones.[18] This situation motivated the derivation of a list of 146 descriptors to aid in the characterization of odors (Figure IV-13).[33] It seems necessary, if unwieldy, and has led to surprising reliability in a multilaboratory comparison.[36] Earlier systems of odor classification, generally hierarchic, with a small number of major categories and associated subdivisions never proved practical.[15]

Data derived from the list of 146 odor descriptors can have particular use in an effort to track down the source of a malodor, particularly an episodic malodor. The list enables persons influenced by the same malodor to express possible consensus regarding its character. Without such uniform terminology, even articulate persons often give such impoverished qualitative descriptions of malodors as "stinky," "rotten," "yucky," and "foul."

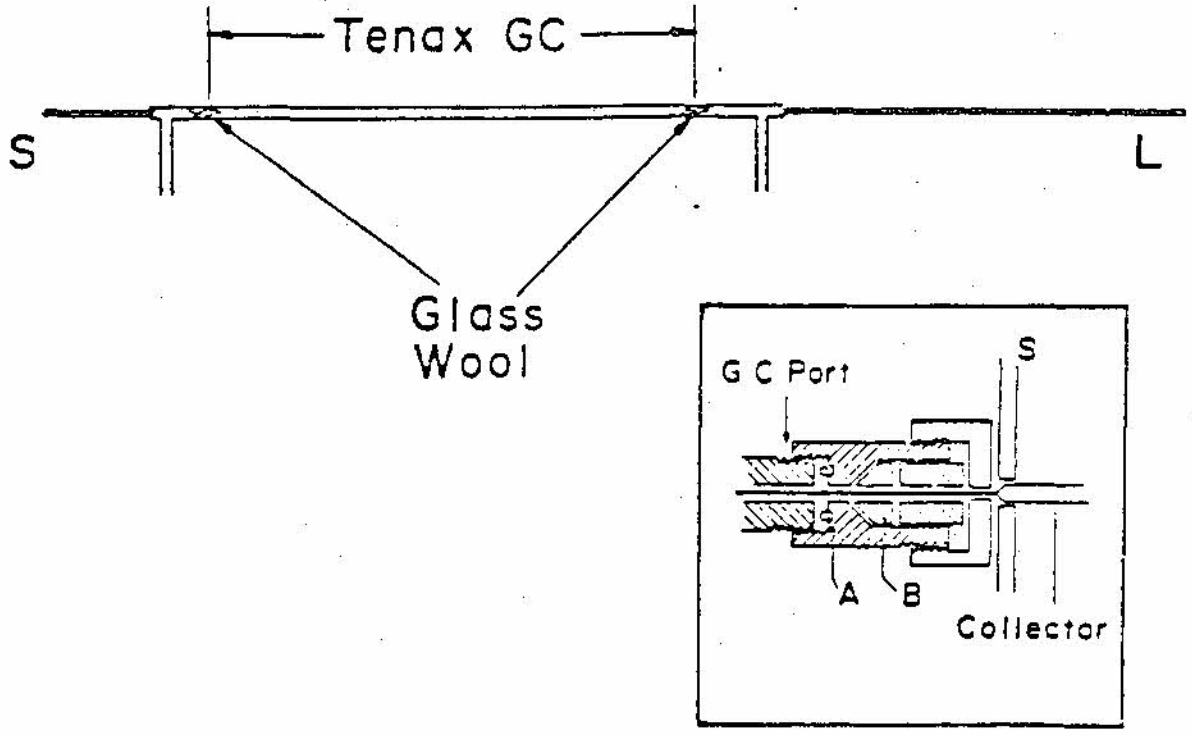
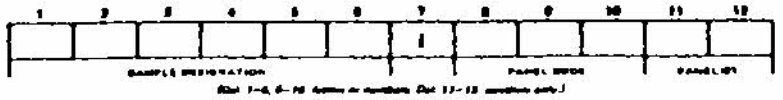


FIGURE IV-12 Tenax-filled collector for organic contaminants. Insert shows details of connecting the end of the collector to the injection port of a gas chromatograph. Length of collector, 200 mm; outside diameter, 3.1 mm. Reprinted with permission from Jarke.[48]



ODOR QUALITY EVALUATION

* SMELL SAMPLE. YOU CAN RESMELL IT AS NEEDED FOR EVALUATION

** GO THROUGH LIST BELOW FOR EACH DESCRIPTOR. *ENCIRCLE* THAT SCORE NUMBER WHICH BEST CHARACTERIZES THE *DEGREE OF PRESENCE* OF THAT ODOR NOTE IN THE SAMPLE ODOR. IF ABSENT, DO NOT *ENCIRCLE ZERO*

*** INITIAL OR SIGN, AND DATE

Initial or Signature: ..... Date: .....

MEANING OF THE ODOR QUALITY SCALE

ABSENT	SLIGHTLY		MODERATELY		EXTREMELY
0	1	2	3	4	5

Analyte	DESCRIPTOR	SCORE	Analyte	DESCRIPTOR	SCORE
001	FRAGRANT	0 1 2 3 4 5	031	OILY, FATTY	0 1 2 3 4 5
002	SWEATY	0 1 2 3 4 5	032	LIKE MOTHBALLS	0 1 2 3 4 5
003	ALMOND LIKE	0 1 2 3 4 5	033	LIKE GASOLINE, SOLVENT	0 1 2 3 4 5
004	BURNT, SMOKY	0 1 2 3 4 5	034	COOKED VEGETABLES	0 1 2 3 4 5
005	HERBAL, GREEN CUT GRASS	0 1 2 3 4 5	035	SWEET	0 1 2 3 4 5
006	ETHEREAL, ANAESTHETIC	0 1 2 3 4 5	036	FISHY	0 1 2 3 4 5
007	SOUR, ACID, VINEGAR	0 1 2 3 4 5	037	SPICY	0 1 2 3 4 5
008	LIKE BLOOD, RAW MEAT	0 1 2 3 4 5	038	PAINT-LIKE	0 1 2 3 4 5
009	DRY, POWDERY	0 1 2 3 4 5	039	RANCID	0 1 2 3 4 5
010	LIKE AMMONIA	0 1 2 3 4 5	040	MINTY, PEPPERMINT	0 1 2 3 4 5
011	DISINFECTANT, CARBOLIC	0 1 2 3 4 5	041	SULPHIDIC	0 1 2 3 4 5
012	AROMATIC	0 1 2 3 4 5	042	FRUITY (CITRUS)	0 1 2 3 4 5
013	MEATY (COOKED, BOOD)	0 1 2 3 4 5	043	FRUITY (OTHER)	0 1 2 3 4 5
014	SICKENING	0 1 2 3 4 5	044	PUTRID, FOUL, DECAYED	0 1 2 3 4 5
015	MUSTY, EARTHY, MOLDY	0 1 2 3 4 5	045	WOODY, RESINOUS	0 1 2 3 4 5
016	SHARP, PUNGENT, ACID	0 1 2 3 4 5	046	MUSK-LIKE	0 1 2 3 4 5
017	CAMPHOR LIKE	0 1 2 3 4 5	047	SOAPY	0 1 2 3 4 5
018	LIGHT	0 1 2 3 4 5	048	GARLIC, ONION	0 1 2 3 4 5
019	HEAVY	0 1 2 3 4 5	049	ANIMAL	0 1 2 3 4 5
020	COOL, COOLING	0 1 2 3 4 5	050	VANILLA-LIKE	0 1 2 3 4 5
021	WARM	0 1 2 3 4 5	051	FECAL (LIKE MANURE)	0 1 2 3 4 5
022	METALLIC	0 1 2 3 4 5	052	FLORAL	0 1 2 3 4 5
023	PERFUMERY	0 1 2 3 4 5	053	YEASTY	0 1 2 3 4 5
024	MALTY	0 1 2 3 4 5	054	CHEESY	0 1 2 3 4 5
025	CINNAMON	0 1 2 3 4 5	055	HONEY-LIKE	0 1 2 3 4 5
026	POPCORN	0 1 2 3 4 5	056	ANISE (LICORICE)	0 1 2 3 4 5
027	INCENSE	0 1 2 3 4 5	057	TURPENTINE (PINE OIL)	0 1 2 3 4 5
028	Cantaloupe, Honey Dew MELON	0 1 2 3 4 5	058	FRESH GREEN VEGETABLES	0 1 2 3 4 5
029	TAR LIKE	0 1 2 3 4 5	059	MEDICINAL	0 1 2 3 4 5
030	EUCALYPTUS	0 1 2 3 4 5	060	ORANGE (FRUIT)	0 1 2 3 4 5

Analyte	DESCRIPTOR	SCORE	Analyte	DESCRIPTOR	SCORE
061	BUTTERY (FRESH)	0 1 2 3 4 5	101	HOUSEHOLD GAS	0 1 2 3 4 5
062	LIKE BURNT PAPER	0 1 2 3 4 5	102	PEANUT BUTTER	0 1 2 3 4 5
063	COLOGNE	0 1 2 3 4 5	103	VIOLETS	0 1 2 3 4 5
064	CARAWAY	0 1 2 3 4 5	107	TEA LEAVES LIKE	0 1 2 3 4 5
065	BARK-LIKE, BIRCH BARK	0 1 2 3 4 5	108	STRAWBERRY-LIKE	0 1 2 3 4 5
066	ROSE-LIKE	0 1 2 3 4 5	109	STALE	0 1 2 3 4 5
067	CELERY	0 1 2 3 4 5	110	CORK-LIKE	0 1 2 3 4 5
068	BURNT CANDLE	0 1 2 3 4 5	111	LAVENDER	0 1 2 3 4 5
069	MUSHROOM LIKE	0 1 2 3 4 5	112	CAT URINE-LIKE	0 1 2 3 4 5
070	WET WOOL, WET DOG	0 1 2 3 4 5	113	PINEAPPLE (FRUIT)	0 1 2 3 4 5
071	CHALKY	0 1 2 3 4 5	114	FRESH TOBACCO SMOKE	0 1 2 3 4 5
072	LEATHER LIKE	0 1 2 3 4 5	115	NUTTY (WALNUT, ETC.)	0 1 2 3 4 5
073	PEAR (FRUIT)	0 1 2 3 4 5	116	FRIED CHICKEN	0 1 2 3 4 5
074	STALE TOBACCO SMOKE	0 1 2 3 4 5	117	WET PAPER LIKE	0 1 2 3 4 5
075	RAW CUCUMBER LIKE	0 1 2 3 4 5	118	COFFEE-LIKE	0 1 2 3 4 5
076	RAW POTATO LIKE	0 1 2 3 4 5	119	PEACH (FRUIT)	0 1 2 3 4 5
077	MOUSE-LIKE	0 1 2 3 4 5	120	LAUREL LEAVES	0 1 2 3 4 5
078	BLACK PEPPER LIKE	0 1 2 3 4 5	121	BURNT MILK	0 1 2 3 4 5
079	BEAN-LIKE	0 1 2 3 4 5	122	SEWER ODOR	0 1 2 3 4 5
080	BANANA-LIKE	0 1 2 3 4 5	123	BOOTY	0 1 2 3 4 5
081	BURNT RUBBER-LIKE	0 1 2 3 4 5	124	CRUSHED WEEDS	0 1 2 3 4 5
082	GERANIUM LEAVES	0 1 2 3 4 5	125	RUBBERY (NEW RUBBER)	0 1 2 3 4 5
083	URINE-LIKE	0 1 2 3 4 5	126	BAKERY (FRESH BREAD)	0 1 2 3 4 5
084	BEERY (BEER-LIKE)	0 1 2 3 4 5	127	OAK WOOD, COGNAC-LIKE	0 1 2 3 4 5
085	CEDARWOOD-LIKE	0 1 2 3 4 5	128	GRAPEFRUIT	0 1 2 3 4 5
086	COCONUT LIKE	0 1 2 3 4 5	129	GRAPE JUICE-LIKE	0 1 2 3 4 5
087	ROPE-LIKE	0 1 2 3 4 5	130	BODY (FRESH BODY)	0 1 2 3 4 5
088	SEMINAL, SPERM LIKE	0 1 2 3 4 5	131	BITTER	0 1 2 3 4 5
089	LIKE CLEANING FLUID (Carbona)	0 1 2 3 4 5	132	CADAVEROUS, Like Dead Animal	0 1 2 3 4 5
090	CARDBOARD LIKE	0 1 2 3 4 5	133	MAPLE (AS IN SYRUP)	0 1 2 3 4 5
091	LEMON (FRUIT)	0 1 2 3 4 5	134	SEASONING (FOR MEAT)	0 1 2 3 4 5
092	DIRTY LINEN LIKE	0 1 2 3 4 5	135	APPLE (FRUIT)	0 1 2 3 4 5
093	SMOKY (SMOKED FISH)	0 1 2 3 4 5	136	SOUPY	0 1 2 3 4 5
094	CARAMEL	0 1 2 3 4 5	137	GRAINY (AS GRAIN)	0 1 2 3 4 5
095	SAUERKRAUT-LIKE	0 1 2 3 4 5	138	CLOVE-LIKE	0 1 2 3 4 5
096	CRUSHED GRASS	0 1 2 3 4 5	139	RAISINS	0 1 2 3 4 5
097	CHOCOLATE	0 1 2 3 4 5	140	HAY	0 1 2 3 4 5
098	MOLASSES	0 1 2 3 4 5	141	KEROSENE	0 1 2 3 4 5
099	ALCOHOL-LIKE	0 1 2 3 4 5	142	NAIL POLISH REMOVER	0 1 2 3 4 5
100	DILL-LIKE	0 1 2 3 4 5	143	FERMENTED (Ferment) FRUIT	0 1 2 3 4 5
101	CHEMICAL	0 1 2 3 4 5	144	CHERRY (BERRY)	0 1 2 3 4 5
102	CREOSOTE	0 1 2 3 4 5	145	VARNISH	0 1 2 3 4 5
103	GREEN PEPPER	0 1 2 3 4 5	146	SOUR MILK	0 1 2 3 4 5

FIGURE IV-13 Data sheet for profiling of odor quality with 146 descriptors. Reprinted from National Research Council.[65]

Odor Intensity. From the standpoint of environmental engineering, intensity is the most important attribute of an odor. It also permits relatively precise measurement by matching. A recently adopted ASTM butanol reference scale has already served well in this capacity.[2] The reference scale entails the use of an olfactometer that sets up eight concentrations of butanol spanning a range of subjective intensity from very weak to very strong. As Figure IV-14 shows, an observer seeks to find a nozzle on a lazy Susan that matches the intensity of a test odor.[11] Figure IV-15 gives an example of some results obtained in this fashion.[37]

Intramodality matching, such as that used with the butanol reference scale, is the most fundamental and secure psychophysical operation. The widespread, successful use of intramodality matching in vision and hearing ensures its basic validity.[38] Nevertheless, its use requires some knowledge of psychophysics in general and olfactory psychophysics in particular. For example, adaptation is important in determining how many judgments a person can make in a given period.[24] Inattention to time-dependent changes in sensitivity can severely distort the outcome of the matching operation.

The NRC report Odors from Stationary and Mobile Sources[65] presented other techniques for assessing odor intensity. Some involve the use of a matching odorant other than butanol.[11 29] Others involve only numerical judgments. The potential value of numerical judgements is derived from the possibility that they reflect the true form of the psychophysical function. Such judgments imply, for instance, that the perceived magnitude of butanol varies with concentration, not proportionally, but with approximately the 2/3 power of concentration. This relation, expressed in the equation $S = 0.261C^{0.66}$ (where S is perceived magnitude and C is concentration), can make it possible to convert a concentration of butanol chosen during matching to a numerical perceived magnitude.[61] The psychophysical technique of magnitude estimation used to derive the equation falls into a class of ratio scaling techniques. These commonly used techniques assume that subjects can judge ratio relations among sensations.[67]

Although the equation above has been proposed as a standard function, its parameters can vary systematically with conditions of stimulation, type of numerical scaling procedure, etc. Hence, the standard function requires specification of standard means of data acquisition. Some category scaling procedures--wherein subjects use a scale of five categories, seven categories, and so on--typically fail to produce a function that conforms to the equation. In principle, however, a category scale with a properly chosen number (and hence range) of categories should produce such a relation and might thereby bring the outcome of category scaling and ratio scaling into register. The issue of why these two classes of procedures generally produce different results has escaped simple resolution throughout sensory research.[58]

Odor Acceptability. The acceptability-objectionability or pleasantness-unpleasantness of an odor can be assessed by essentially

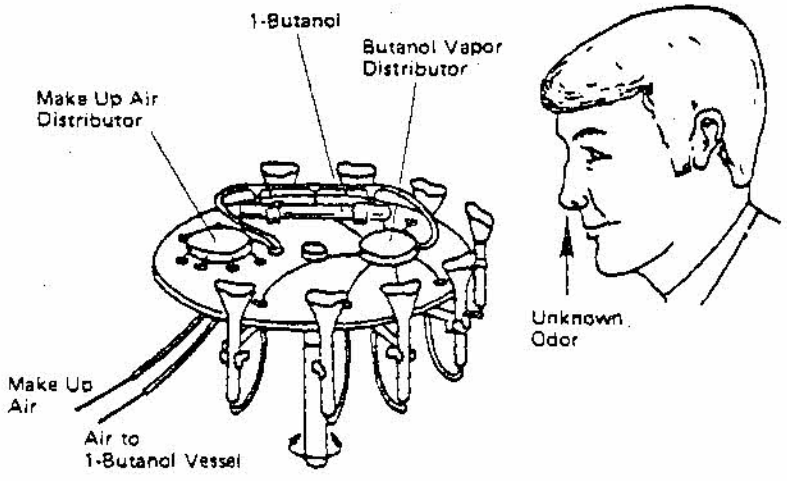


FIGURE IV-14 A subject using the butanol olfactometer (lazy Susan configuration) to find an odor that matches an unknown stimulus. As customarily arranged, the device delivers concentrations that range from 16 to 2,000 ppm. Concentration changes by a factor of 2 from port to port. Reprinted with permission from Flavor Quality: Objective Measurement.[31(p. 13)]

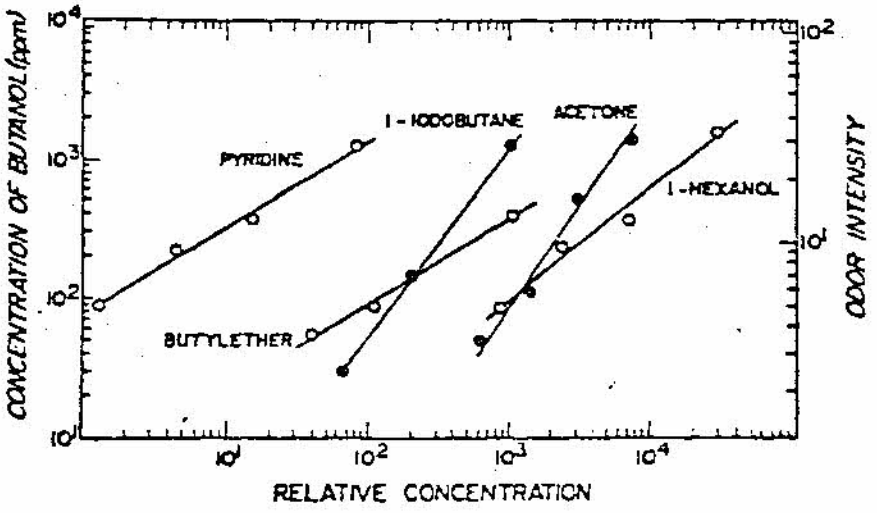


FIGURE IV-15 Psychophysical functions for five odorants. Functions were obtained by matching butanol (note left ordinate) to various concentrations of each odorant. Right ordinate shows odor intensity derived via the psychophysical function for butanol. Data from Dravnieks and Laffort.[37]

the same techniques as its intensity. For instance, Lindvall and Svensson[56] used matching to hydrogen sulfide (rotten-egg odor) to assess the unpleasantness of combustion-toilet emission. Many investigators have used numerical scaling, sometimes ratio scaling,[39] and sometimes category scaling.[38] The category scales have typically been bipolar, ranging, for instance, from -3 (very unpleasant) to +3 (very pleasant).[7] Unfortunately, such a scale seems often to misrepresent the variability of acceptability and objectionability in the extremes of the hedonic continuum. Accordingly, some persons have used a line-marking or line-producing technique.[62] This generally involves a bipolar scale, but without fixed categories, and seems to yield a more realistic picture of response variability than does category scaling.[25] Other techniques of continuous, rather than categoric, judgment may behave just as well as line-marking, with respect to response variability.

Odor Threshold. The measurement of odors has often comprised merely the measurement of the concentration (or dilution) necessary to achieve some criterion of detectability. The literature on olfaction contains well over 1,000 threshold values for odorants. All too commonly, these were obtained by techniques devised specifically for particular investigations;[41 72] for this reason, the threshold value for a particular odorant may vary widely, even by orders of magnitude, from one investigation to another. Specific factors contributing to this variability include the means of stimulus presentation to the odor-panel member, judgmental factors, and the implicit or explicit definition of threshold. Few experimenters have sought to verify the reliability of the vessels or olfactometers used to present odorous stimuli. Furthermore, each device customarily uses arbitrarily chosen conditions (e.g., flowrate, temperature, humidity, and solvent), and this often precludes comparison with results obtained with other devices or, more important, may preclude a comparison with typical environmental conditions.

The psychophysical means of eliciting information on detectability can alter apparent sensitivity by a factor of 100 or more.[55] Presenting a pair of stimulus samples--one containing an odorant at a fixed, weak concentration and the other containing only air--and forcing the subject to choose one of the two in each of scores or hundreds of trials will maximize detection. (This forced-choice procedure could also offer one odorous sample and two blanks, etc.) But presenting various concentrations randomly with no direct comparison stimulus ("blank") invites variability. One subject may set a low criterion and say "yes" to almost anything. Another subject may behave much more conservatively. Such differences between subjects have given incentive to erect a new psychophysics of signal detection.[36] Because of its time-consuming demands in data collection, the theory of signal detection is not popular with researchers. That theory challenges the concept of threshold and instead specifies, in probabilistic and relatively bias-free terms, the detectability of any given signal. In this respect, it highlights the probabilistic nature of all thresholds. Despite the layman's view, "threshold" hardly

refers to a concentration below which a normal person can never detect odor. Even concentrations well below "threshold" may be detectable with sufficient frequency to cause concern about the validity of the threshold concentration.

Complications involved in the interpretation of existing threshold data and pitfalls in the collection of new data raise the question of whether "thresholds," or comparable indexes of detection, offer greater benefits than suprathreshold matching. Nevertheless, some persons will undoubtedly choose threshold measurement over matching. A relatively new ASTM procedure at least offers some standardization.[3] It recommends three-alternative forced-choice presentations wherein one nozzle of an air-dilution olfactometer presents an odorous stimulus at a given dilution and two companion nozzles present odorless air. Testing begins at a very low concentration and progresses to higher concentrations until the subject detects the stimulus reliably. Although the method contains arbitrary ingredients, it combines various important features designed to minimize response bias; and it allows a relatively speedy, if gross, estimate of the degree of dilution that a sample of odorous air can withstand before it ceases to be detected readily.

ODOR CONTROL

The control of odor should follow the same strategy as the control of virtually any other type of indoor contaminant. The first step should involve good housekeeping and prevention of the source. This will obviously fail in the many cases where the mere presence and normal activities of occupants inevitably give rise to odorous organic materials. A second step would eliminate airborne contaminants, such as kitchen odors, by local exhaust.

In principle, the removal of odorous contaminants can follow one or more of the classical strategies: oxidation (e.g., incineration), scrubbing (e.g., spray-washing), chemical conversion (e.g., chlorination), filtration, and dilution.[70] The first three of these are used almost exclusively in the control of industrial odorous emission. For odors in residential, commercial, and institutional spaces, filtration and dilution are the methods of choice. The use of deodorizers is justified only under unusual and temporary conditions.

Ventilation

Before the development of mechanical ventilation, the entrance of outside air through windows was the means of both thermal and contaminant control. The need for thermal control often dictated the demand for outside air.[21] Mechanical ventilation systems, however, allowed separate control of the temperature and contaminants. The amount of ventilation air necessary for control of contaminants historically has been a matter of contention. Some persons argued for ventilation rates that would render the air odorless. This strategy

rested on the premise that odor-laden air necessarily contained harmful organic contaminants. Similarly, some argued for rates that would maintain carbon dioxide at a concentration only twice that of the ambient air.[51] Here again, the strategy rested on the notion that conservative control of a measurable (or, in the case of odors, perceptible) contaminant, even a rather innocuous one, would take care of unknown, but possibly harmful, airborne substances. This strategy led to unreasonably high ventilation rates. To achieve a criterion of approximate odorlessness, the ventilation rate must generally exceed 30 ft^3/min (about 14 L/s) per occupant, even during nonsmoking occupancy.

The New York State Commission on Ventilation found that the concentration of carbon dioxide in a normally ventilated schoolroom correlated only weakly with odor.[55] Hence, the use of one contaminant seemed unable to predict the concentrations of all contaminants. The correlation rule may actually work reasonably well under conditions of active control of the delivery of ventilation air.[53] Nevertheless, the New York State commission found that a rate of 10-15 ft^3/min (about 4.7-7.1 L/s) per student in a classroom with about 250 ft^3 (9 m^3) per student sufficed to control odor and carbon dioxide concentration reasonably well; furthermore, it seemed acceptable on the basis of criteria of comfort, health, and performance. In Winslow's[76] words:

> The chemical vitiation of the air of an occupied room (unless poisons or dusts from industrial processes or defective heating appliances are involved) is of relatively slight importance. The organic substances present, manifest as body odors, may exert a depressing effect upon inclination to work and upon appetite; therefore occupied rooms should be free from odors which are obvious to anyone entering from without. (Such odors are never perceived by those who have been in the room while they have been accumulating.) Objectionable effects of this sort have only been demonstrated, however, with a carbon dioxide content of over .2 per cent, which would correspond to an air change of less than 6 cubic feet per person per minute. (pp. 77-78)

As mechanical ventilation systems became more common, there was more interest in discovering how the odor of a room would vary with changes in the proportion of total supply air that consisted of ventilation (outdoor) air. Figure IV-16 depicts a functional relation that Houghten and colleagues[57] erected from judgments in a junior-high-school classroom. The function, derived from judgments of visitors who entered the occupied classroom briefly, intersects the line equal to a judgment of 2 ("noticeable [odor] but not objectionable") at a ventilation rate of about 11 ft^3/min (about 5.2 L/s) per student. This outcome seemed to confirm the findings of the New York State commission.

Experiments performed at the Harvard School of Public Health shortly after the study of Houghten and colleagues implied that ventilation requirements per occupant would vary with the amount of space (volume) available to each occupant. Yaglou and colleagues[78]

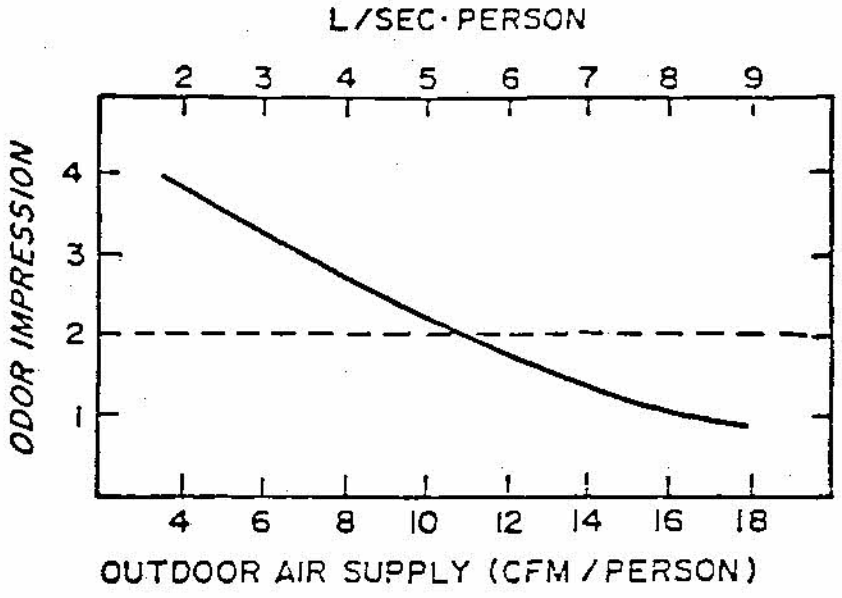


FIGURE IV-16 Relation between odor intensity and ventilation air in junior-high-school classrooms. Observers entered the occupied classroom from a relatively odor-free corridor. Adapted from Houghten et al.[47]

of Harvard, like Houghten et al., derived functions that related odor to ventilation rate and added such variables as relative crowding, hygiene, and age. Each variable had some influence, but crowding (i.e., occupant density in an experimental room) had the most noteworthy influence. Figure IV-17 depicts ventilation requirements decided by various criteria versus air space per occupant. Function C represents requirements according to an odor criterion of "moderate" (a rating of 2 on a scale of 0-5). It reflects the intersection of this perceived extent of odor with the various combinations of ventilation rate and air space per person in the functions for occupancy odor depicted in Figure IV-18.

Although admittedly incomplete, the work of Yaglou et al. stands as the most definitive investigation of ventilation requirements ever performed. It seemed to figure at least implicitly in the recommendations of ASHRAE Standard 62-73, a standard based on professional consensus.[6] As Figure IV-19 shows, the recommended ventilation rates for a diverse group of residential and commercial spaces follows much the same curvature as the function of Yaglou et al.[16] Almost all the recommended rates fall above the function--an unsurprising feature, inasmuch as most spaces have a higher odor load than that imposed merely by sedentary occupancy. Cigarette-smoking, for instance, leads to much higher ventilation requirements. Unfortunately, the study shown in Figure IV-20 proffered the only thorough look at the ventilation requirements necessary to control the odor of fresh tobacco smoke.[77] In many of the spaces represented in Figure IV-17, smoking or some physical activity might occur. Curve D in Figure IV-17 formed one attempt to account for the requirements in spaces with such activities. The function, a 50% upward transposition of curve C, apparently has no experimental justification, but may nevertheless serve well as a rough guide for ventilation requirements.[73]

See Chapter IX for more complete discussion of ventilation standards.

Air-Cleaning

The principles of contaminant dilution and displacement, achieved through ventilation, offer the simplest means of indoor contaminant control. In some instances, however, ventilation alone proves inefficient or ineffective. Other means of control can then assist. Outdoor air sometimes contains unwanted concentrations of contaminants, and recirculation of indoor air becomes desirable. For instance, the outdoor air may contain enough sulfur dioxide to damage sensitive electronic equipment. Filtration of one sort or another can reduce the concentrations of the contaminant in the incoming air or, sometimes more productively, can reduce the concentrations of the various contaminants generated indoors and thereby reduce reliance on the use of contaminated outdoor air.[50] In principle, filtration of recirculated air can achieve indoor air quality that exceeds that of outdoor air. But in practice, filtration leaves some contaminants (e.g., carbon monoxide and carbon dioxide) unattenuated. Only very expensive procedures will eliminate these contaminants. Thus, an

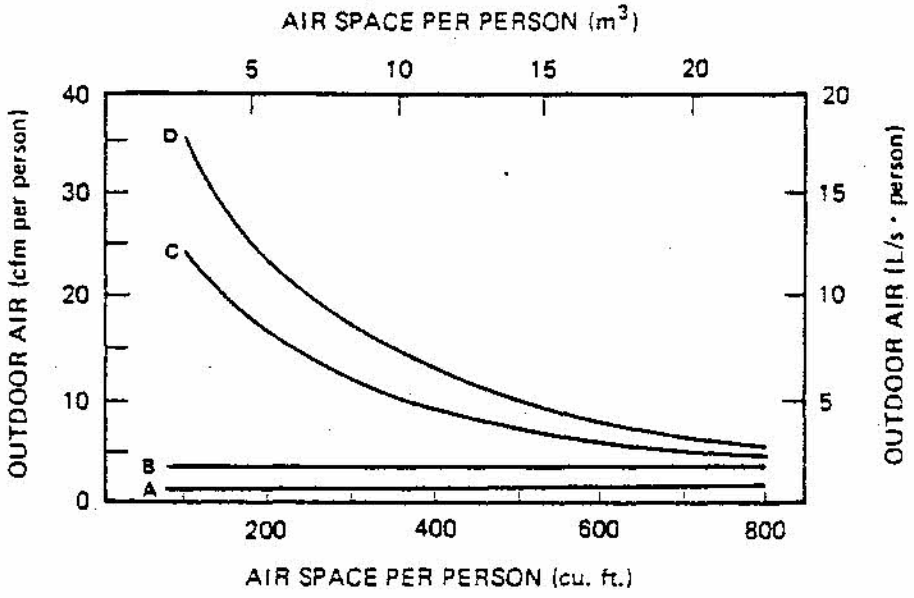


FIGURE IV-17 Relation between ventilation rate and air space per occupant according to four criteria: A, maintenance of oxygen; B, control of carbon dioxide (<0.6%); C, control of body odor under sedentary conditions (no smoking); and D, control of odor when occupants were slightly active and when smoking was permitted. Lower curve derived from data of Yaglou et al. Adapted from Viessman.[73]

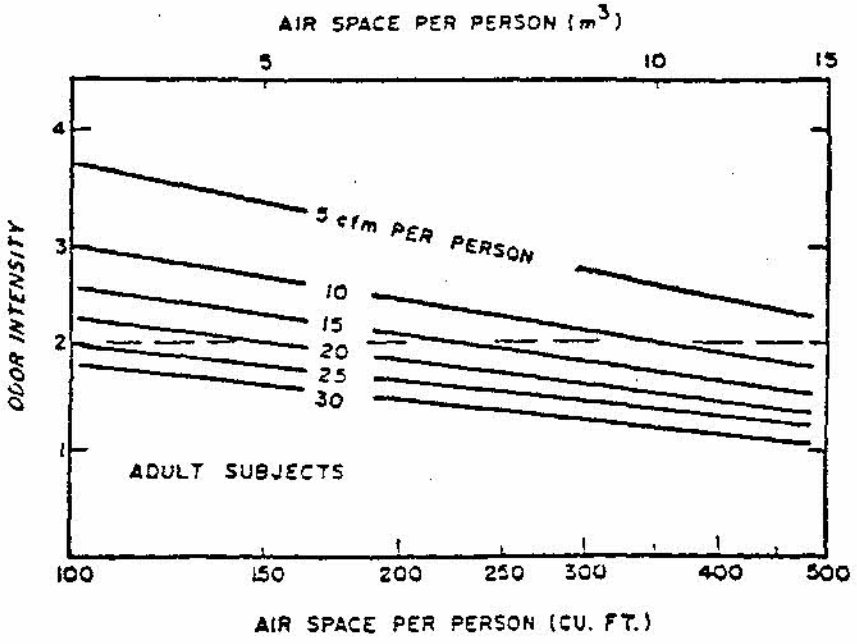


FIGURE IV-18 Odor intensity versus net air space per person for ventilation rates of 5-30 cfm (2.5-15 L/s). Adapted from Yaglou et al.[78]

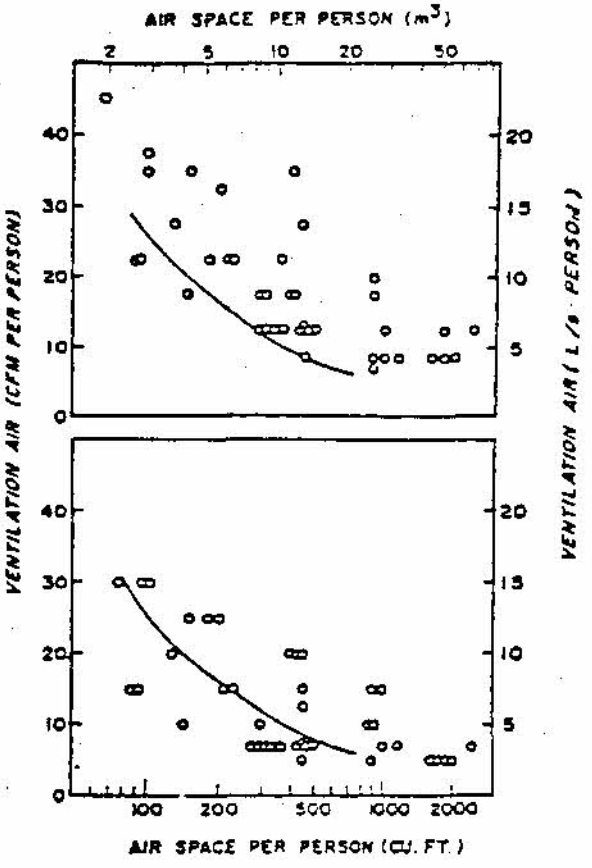


FIGURE IV-19 Top, points depict rates of ventilation recommended for various residential and commercial spaces by ASHRAE Standard 62-73 versus air space per person (logarithmic scale). Air space per person was derived from estimates of occupancy per 1,000 ft^2 (93 m^2) of floor area (incorporated into the standard) and from estimate of ceiling height. Excluded were spaces, such as theaters, where ceiling height might vary considerably from one space to another. Bottom, points depict rates of ventilation recommended for new buildings by ASHRAE Standard 90-75. The points represent the so-called minimal ventilation rates of Standard 62-73. Lines in top and bottom portions are function C from Figure IV-17.

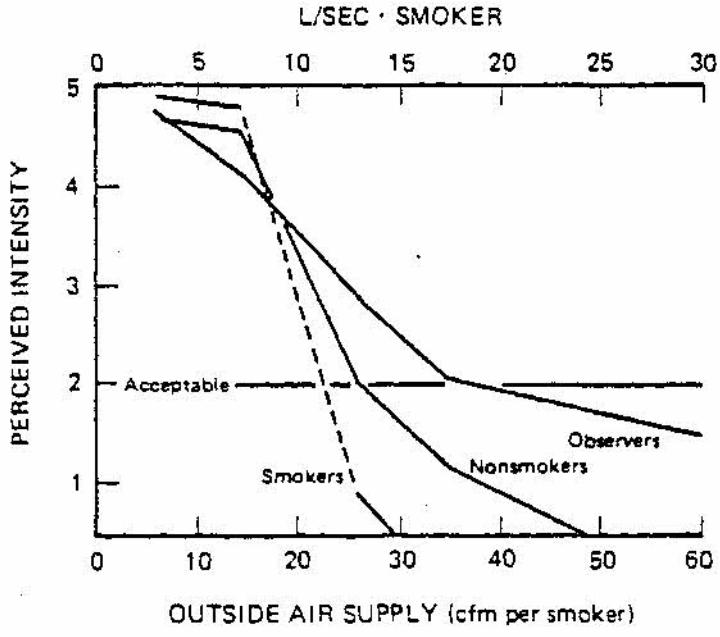


FIGURE IV-20 Variation in odor intensity with fresh air supply when nine persons, including six smokers, occupied Yaglou's chamber and smoked cigarettes at a rate of 24 per hour. Functions labeled "smokers" amd "nonsmokers" depict judgments of occupants. Function labeled "observers" depicts judgments of persons who entered briefly from an odor-free room. Adapted from Yaglou.[77]

engineer or designer cannot rely entirely on recirculation, but must deliver some minimal quantity of outdoor air. According to ASHRAE guidelines,[6] the minimum should equal 5 ft^3/min (about 2.4 L/s) per person when the filtration system has high efficiency. This quantity of air guarantees adequate control of carbon dioxide with a substantial margin of safety. In a space that might normally demand outdoor air at, say, 20 ft^3/min (about 9.4 L/s) per occupant to control the concentrations of organic materials generated during occupancy, use of a high-efficiency filtration system could save considerable amounts of the energy generally used to heat or cool ventilation air.

The characteristic way to clean indoor air involves the use of granular filter media and, if necessary, particle filters.[7] Particle filtration will prove necessary in the presence of cigarette-smoking. Some portion of tobacco-smoke odor is presumably eliminated by such filtration. Particle filters protect the granular filter.

Activated carbon is the most common type of granular medium for control of airborne organic matter.[68] It removes most odorous material and generally renders the air "fresh-smelling." Its adsorbent property leads to actual retention of the contaminant up to a saturation point, when the filter bed must be replaced. Activated carbon is available in many varieties, depending on starting materials and production conditions. Efficiency varies considerably, and life span may be unpredictable. Such technical vagaries detract from the use of activated carbon by nonspecialists, and it currently is little used in ordinary ventilation systems. Other adsorbent materials, such as porous polymers, have been developed for gas chromatography,[68] and perhaps these now-costly materials will become cheap enough for use in ventilation.

Finally, activated carbon or activated alumina can be impregnated with other materials to increase efficacy against particular contaminants. Activated alumina impregnated with postassium permanganate, developed specifically for odor control, offers the most readily available commercial alternative to activated charcoal.[65] It operates by adsorption and oxidation and thereby capitalizes on an empirically observed rule that a malodorant may lose its malodorous properties when oxidized.

RESEARCH NEEDS

Ventilation Codes

A survey of building codes in the 1960s uncovered a tenfold variation in the listed ventilation rates.[10] Such variation reflects in part the change in modes of thought regarding ventilation requirements through the years. Some codes apparently arose from local notions of "good engineering practice" and had seen little or no revision in decades. In the late nineteenth century, the American Society of Heating and Ventilating Engineers recommended 30 ft^3/min per occupant. As mentioned above, this value arose from the notion that ventilation should keep the indoor concentration of carbon dioxide

at less than twice its outdoor concentration. Forty years later, the Society recommended a rate of 10 ft^3/min per occupant. Although the studies of the New York State commission and other researchers had made it clear that a rate as low as 10 ft^3/min per occupant would not lead to harm, the members of the Society's committee apparently felt the need for considerably more data. W. H. Driscoll, a member of the committee, noted:[78]

> When we finally decided that we would take 10 cfm per person as the minimum it was a sheer compromise, merely an attempt to finish the work of the Committee and get the report before the Society. There was a difference of opinion as to whether the 30 cu ft that have been set up as a standard since time immemorial should be adopted, or whether no cubic feet, for which there was very aggressive support, not necessarily within the Committee but from outside of the Committee, on the theory that no scientific studies had ever been made to support the necessity for the introduction of any outdoor air as a ventilation requirement. (p. 160)

The experiments of Yaglou et al.[78] added some critical information, but this did not necessarily become part of the thinking of code-makers. For instance, some codes still specify ventilation requirements in terms of air changes per hour, irrespective of density of occupancy. Yaglou et al. had suggested a need for great sensitivity regarding density of occupancy. Indeed, Yaglou and Witheridge's experiment prompted the comment:[79]

> One result of this investigation, for which I think we can be particularly grateful to the authors, is that it effectively explodes the myth of air change based on room volume as a measure of ventilation. I consider it a real step forward if the idea of air change on a room volume basis can be eliminated from our thinking. (p. 432)

Irrespective of the reasons for variation in ventilation codes, its mere existence implies that a reduction of rates in some of the localities now requiring high rates would probably arouse few complaints. Despite variation from one location to another, both code-makers and ventilating engineers have generally sought to supply ventilation air liberally. Most codes specify rates for conditions of full occupancy; but full occupancy may occur only rarely.

The increasing cost of energy has prompted an unprecedented desire for uniformity in American ventilation codes. The bottom part of Figure IV-19 shows the so-called minimal ventilation rates promulgated in ASHRAE Standard 62-73.[6] These values, like the recommended values shown above them, capture much of the curvature of the recommendations of Yaglou et al.[78] The minimal values actually cluster around the Yaglou et al. function. When energy first became particularly expensive, it seemed desirable to reduce the "recommended" ventilation rates to these minimal values, at least on a trial basis. These were therefore incorporated into the general standard, ASHRAE 90-75, Energy

Conservation in New Building Design.[5] The various model-code groups (Building Officials and Codes Administrators International, International Conference of Building Officials, and Southern Building Code Congress International) have since cooperated with the National Conference of States on Building Codes and Standards (NCSBCS) in drawing up an energy-conservation code, Code for Energy Conservation in New Building Construction.[53] It too incorporated the minimal rates of the ASHRAE ventilation standard.

An NCSBCS survey of building codes[54] conducted in May 1979 revealed widespread adoption of energy conservation in ventilation (Table IV-23). Virtually all large jurisdictions have conformed or will soon conform to conservation standards through adherence to one or another model code, a separate state code rooted in a model code, or ASHRAE Standard 90-75. At the time of the survey, only five jurisdictions had failed to take overt action toward the adoption of an energy-efficient code.

Energy Efficiency, Comfort, and Health

Both the recommended and minimal ventilation rates in ASHRAE Standard 62-73 comprise consensus values. Except, perhaps, for spaces with unexpectedly high rates of cigarette-smoking, the recommended values probably serve well. In some spaces with heavy smoking or cooking, the minimal values will probably serve very poorly. Without a foundation of modern research on a variety of contaminants, the justification of the minimal values will rest heavily on considerations of energy, rather than health. The research must seek to specify rates and strategies that will achieve both energy efficiency and healthful conditions. This will eventually require strict control over the internal atmosphere. Without guidance, some engineers will shut down intake-air dampers to save fuel and will thereafter rely on infiltration alone for fresh air. This strategy actually reduces, rather than increases, the engineer's active control over the building.

The dimensions of ventilation research are roughly the same now as in the time of the work of Yaglou et al. Ventilation dilutes contaminants. Its efficacy depends on the nature of indoor contaminants, the size of a space, environmental variables, furnishings, duration of occupancy, aesthetic standards of occupants of or visitors to the space, and sensitivity of occupants. Modern methods available for experimentation include the newly standarized psychophysical methods discussed here and monitoring equipment not available during earlier research on ventilation (e.g., continuous carbon monoxide analyzer, continuous carbon dioxide analyzer, gas chromatograph, mass spectrometer, particle-mass monitor, condensation nucleus counter, and electric aerosol analyzer). With these various tools, it is now possible to characterize the indoor environment in both a psychophysical and a physicochemical manner. Such characterization has now begun in North America and Europe.[22]

TABLE IV-23

NCSBCS Survey of Energy-Conservation Codes[a]

Jurisdiction	Energy-Conservation Code	Jurisdiction	Energy-Conservation Code
Alabama	SBCCI	Nebraska	(ICBO)
Alaska	(ASHRAE 90-75)	Nevada	MCEC
Arizona	(State code)*	New Hampshire	MCEC
Arkansas	(State code)*	New Jersey	BOCA
California	State code	New Mexico	ICBO
Colorado	MCEC	New York	State code*
Connecticut	State code*	North Carolina	State code
Delaware	(MCEC)	North Dakota	ICBO
District of Columbia	(City code)	Ohio	MCEC
		Oklahoma	None
Florida	State code*	Oregon	ICBO
Georgia	MCEC	Pennsylvania	(ASHRAE 90-75)
Hawaii	ICBO	Rhode Island	ASHRAE 90-75
Idaho	ICBO	South Carolina	SBCCI
Illinois	(State code)*	South Dakota	(MCEC)
Indiana	MCEC	Tennessee	MCEC
Iowa	MCEC	Texas	(MCEC)
Kansas	State code	Utah	MCEC
Kentucky	MCEC	Vermont	(ASHRAE 90-75)
Louisiana	(MCEC)	Virginia	BOCA, ASHRAE 90-75
Maine	(State code)	Washington	State code
Maryland	(ASHRAE 90-75)	West Virginia	None
Massachusetts	MCEC	Wisconsin	State code*
Michigan	ASHRAE 90-75	Wyoming	ICBO
Minnesota	ASHRAE 90-75	American Samoa	MCEC
Mississippi	(MCEC)	Guam	ICBO
Missouri	(MCEC)	Puerto Rico	MCEC
Montana	MCEC		

[a]( ) denotes that legislation is pending.
ASHRAE 90-75 = ASHRAE STANDARD 90-75.
BOCA = Model Code, Building Officials & Code Administrators International, Inc.
ICBO = Model Code, International Conference of Building Officials.
MCEC = Model Code for Energy Conservation in New Building Construction.
SBCCI = Model Code, Southern Building Code Congress International Inc.
Asterisk denotes obvious incorporation of energy-conserving aspects of a model code or ASHRAE 90-75; codes or pending codes for California, Maine, and North Carolina also include some such aspects.

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TEMPERATURE AND HUMIDITY

The atmosphere not only has an important role in respiratory gas exchange (supplying oxygen and accepting carbon dioxide), but also serves as the heat-exchange medium surrounding the human body. Atmospheric pressure, including its constituents and especially water-vapor pressure, environmental temperature, and the rate of air movement all affect rates of heat loss and heat gain. A change in the rate of heat loss or heat gain ultimately has an effect on body heat content and body temperature.

Human body temperatures are maintained within very narrow ranges either by involuntary physiologic responses controlled by the thermoregulatory system or by behavioral adjustments that modify the thermal environment toward thermal equilibrium. The physiologic responses are proportional to deviations from preferred body temperature, especially those associated with the hypothalamic region of the brain. Behavioral adjustments are proportional to deviations in thermal sensation and thermal comfort from thermoneutral and acceptable, respectively. In general, people prefer to use the behavioral adjustments, rather than having to rely on the physiologic responses.

The thermal state of the human body can be described by the heat balance equation:

$$S = M - E - W - R - C, \qquad (1)$$

in which S = rate of storage of body heat,
M = rate of metabolic heat production,
E = rate of evaporative heat loss,
W = rate of external work done,
R = rate of radiant heat loss, and
C = rate of convective heat loss.

All the above terms are usually expressed in watts per square meter of skin surface area. Body surface areas in adults range from 1.6 to 2.2 m^2 and can be estimated by Equation 2 from height and weight:

$$A_D = 0.202\, m^{0.425} H^{0.725}, \qquad (2)$$

in which A_D = body surface area, m^2,
m = body weight, kg, and
H = body height, m.

Except for short periods, Equation 1 should result in values of S very near zero. Any deviation from zero results in lowering or raising of body temperature (hypothermia or hyperthermia, respectively), with adverse consequences for health and well-being. The metabolic heat generated in the body varies from a minimum of 45 W/m^2 to a maximum that varies between 600 and 900 W/m^2 for short periods.

Heat losses in excess of 45 W/m^2 in the resting state tend to produce hypothermia in some people. Depending on the individual, evaporative heat loss must be at least 100-300 W/m^2 to prevent hyperthermia under conditions of sustained hard work. The range of deep-body temperatures that can be encountered is shown in diagrammatic form in Figure IV-21. A range of ambient temperatures with the appropriate responses is shown in Figure IV-22, and a range of metabolic rates associated with some typical activities is presented in Figure IV-23.

As is evident from the heat-balance equation, many factors determine the heat balance, and they must always be evaluated simultaneously. Energy-conservation strategies may involve lower or higher air temperatures, higher or lower vapor pressures, and higher or lower air velocities. Interruptions in energy supplies can result in sharply higher or lower air temperatures, which can have additional adverse health effects.

HEAT EXCHANGE WITH THE INDOOR ATMOSPHERE

A complete assessment of heat exchange between man and his thermal environment can be found in a recent review by Gagge and Nishi.[8] The following is a limited overview.

Whenever the value of S differs from zero in the heat-balance equation, body temperature will change. At an average body weight of 40 kg/m^2 and a weighted average specific heat of 36.5 $Wh \cdot °C \cdot m^{-2}$, it follows that, for example, with S at 36.5 W/m^2 for 1 h, the average body temperature will rise by 1°C.

The rate of metabolic heat production, M, cannot be reduced to below about 40 W/m^2, but can rise to as high as 800 W/m^2 during maximal exercise. Heat exchange by convection (C), by radiation (R), and by evaporation (E) is affected by ambient temperature T_a, by air velocity, and by clothing insulation. For a nude person, convective heat exchange in still air (velocity, V, less than 1 m/s) is approximated by:

$$C = h_c(\bar{T}_s - T_a), \qquad (3)$$

in which C = convective heat exchange, W/m^2,
h_c = convective-heat-transfer coefficient, $W \cdot m^{-2} \cdot °C^{-1}$,
$\bar{T}_s$ = mean weighted skin temperature, °C, and
T_a = ambient air temperature, °C.

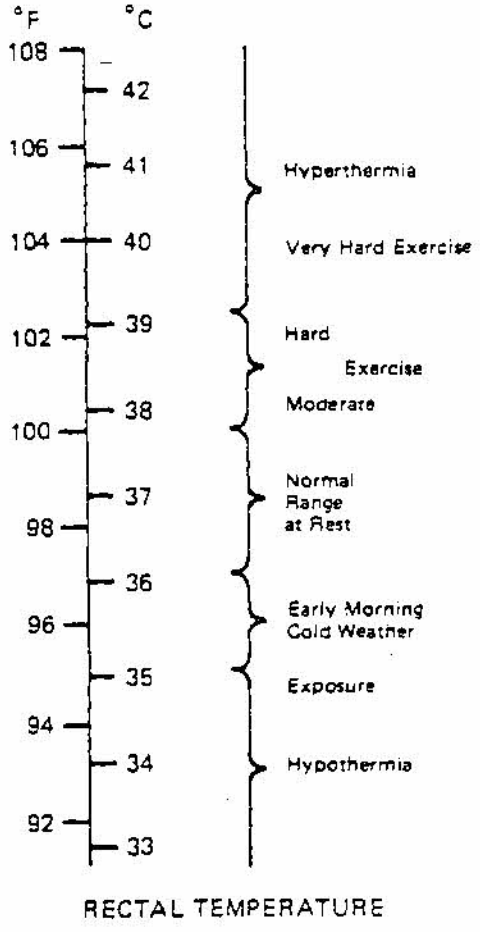


FIGURE IV-21 Range of rectal temperatures encountered in different conditions. Temperatures designated as "hyperthermia" and "hypothermia" are associated with increased risk to health and should be avoided.

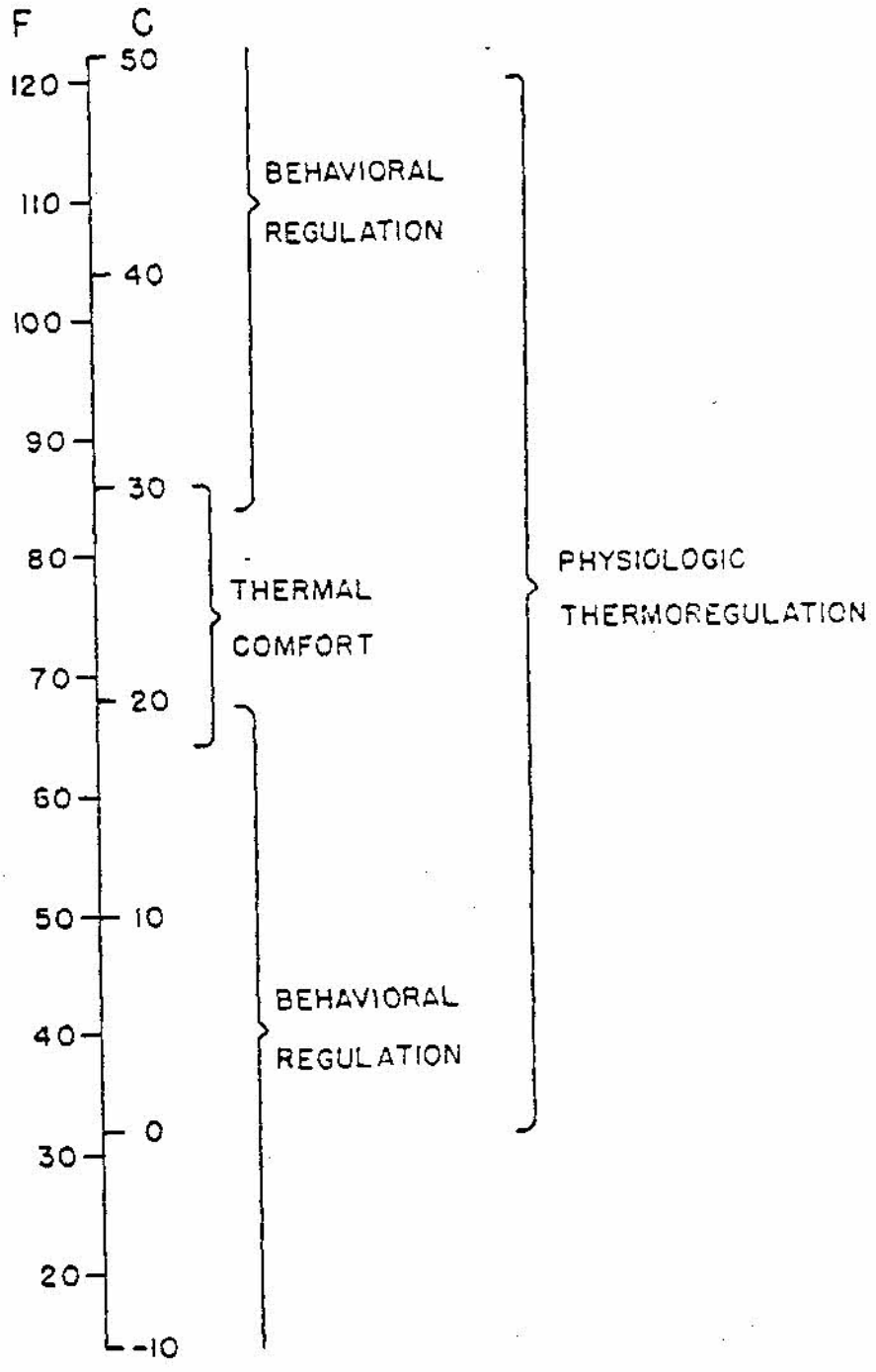


FIGURE IV-22 The range for thermal comfort assumes minimal clothing at the high end and substantial clothing at the low end and thus includes some behavioral regulation. The zones designated "behavioral regulation" require modification by means other than clothing. "Physiologic thermoregulation" indicates the limits of short-term physiologic regulation in a healthy person at rest with minimal clothing.

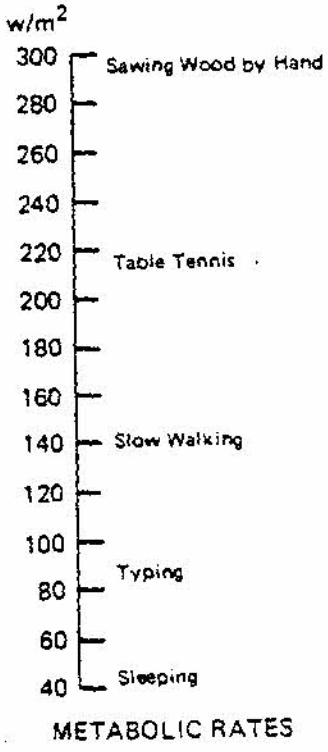


FIGURE IV-23 Metabolic heat production rates asspciated with various activities. Adapted from Berenson and Robertson.[1]

The convective-heat-transfer coefficient depends on position and posture and on air velocity. Reasonable estimates of h_c are about 4 $W \cdot m^{-2} \cdot °C^{-1}$ in still air and 11.6 $v^{0.5}$ in air with a velocity of over 0.2 m/s.[11] The human body also exchanges heat by radiation. The radiant environment is usually characterized by a mean radiant temperature ($\bar{T}_r$) or by an effective radiant field (H_r) in watts per square meter. Equation 4 shows the conversion between these terms:

$$H_r = h_r(\bar{T}_r - T_a), \tag{4}$$

in which h_r = radiant-heat-transfer coefficient.

A reasonable approximation of h_r is 4.5 $W \cdot m^{-2} \cdot °C^{-1}$ at a skin temperature of 34°C and an ambient temperature of 29°C. It is often convenient, especially when $\bar{T}_r$ and T_a are relatively close together, to combine radiation and convection into an overall heat-transfer coefficient, h, and to use the operative temperature, T_o, for the thermal environment. Operative temperature is the temperature of an environment with uniform air and wall temperature that exchanges heat with the body at the same rate as with the complex environment that it describes. The combined heat-transfer coefficient (h) in still air is about 8-10 $W \cdot m^{-2} \cdot °C^{-1}$.

The use of clothing reduces the heat exchange with the environment. This insulation, I_{clo}, is usually expressed in clo units; 1 clo unit corresponds to insulation at 0.155 $m^2 \cdot °C \cdot W^{-1}$. The reciprocal of I_{clo} is h_{cl}, the conductance of clothing. If clothing efficiency is F_{cl}, then

$$F_{cl} = h_{cl}/(h_{cl} + h) = 1/(1 + 0.155hI_{clo}), \tag{5}$$

and the total radiant and convective heat transfer with clothing becomes:

$$R + C = F_{cl}h(\bar{T}_s - T_o). \tag{6}$$

F_{cl} varies from 1 for a nude man down to 0.25 for heavy winter clothing, including an overcoat. The temperature at which man feels thermoneutral at rest varies with clothing. At $F_{cl} = 1$, thermoneutrality occurs at 28°C; at $F_{cl} = 0.25$, it occurs at 15°C. Figure IV-24 further illustrates the relationship between clothing and temperature for thermoneutrality.

At temperatures above thermoneutrality, or when increased metabolic heat production exceeds loss of "dry" heat to the environment, sweat is secreted and the resulting evaporative heat loss (E) restores overall thermal equilibrium. For every gram of sweat that evaporates from the skin, 0.68 Wh of heat is lost from the skin. The maximal evaporative heat loss is limited by the maximal amount of sweat that can be secreted (about 1,500 g/h) and by the evaporative power of the environment. The maximal rate of evaporation, E_{max}, from a completely wetted skin is given in Equation 7:

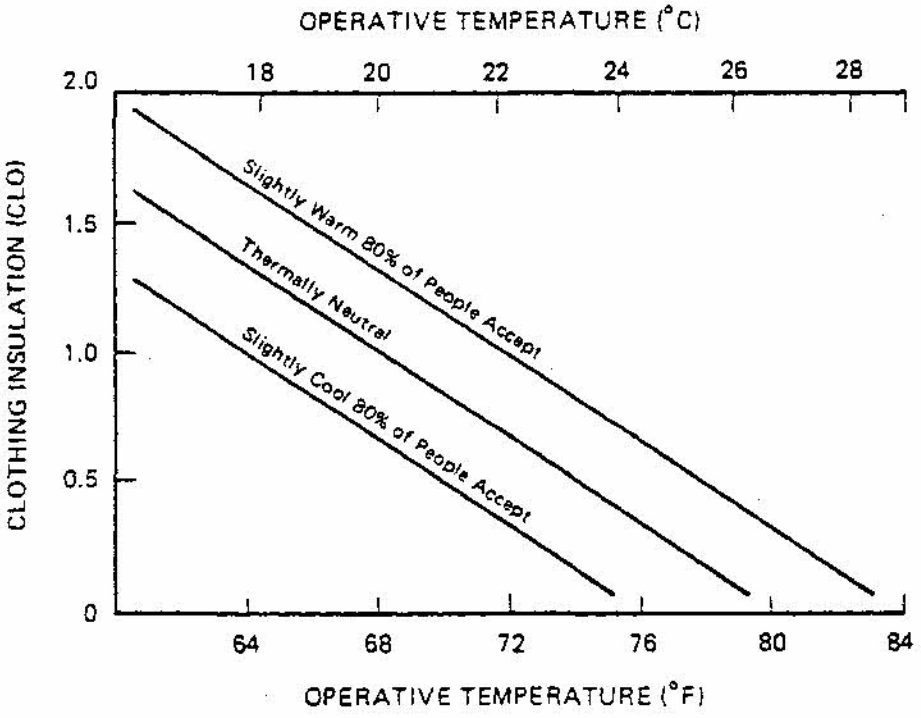


FIGURE IV-24 Effect of interaction of operative temperature and clothing insulation on thermoneutrality, and ambient temperatures accepted by 80% of people when sedentary at minimal air velocity.

$$E_{max} = 2.2h_c(P_{sk} - P_{dp})F_{pcl}, \quad (7)$$

in which E_{max} = maximal evaporative rate, W/m^2,
2.2 = Lewis relation, °C/Torr,
h_c = convective heat transfer coefficient, $W \cdot m^{-2} \cdot °C^{-1}$,
P_{sk} = saturated water-vapor pressure at skin,
P_{dp} = water-vapor pressure in the atmosphere, Torr, and
F_{pcl} = Nishi permeation factor.[12]

For most types of porous clothing, Nishi and Gagge[12] have shown experimentally that

$$F_{pcl} = 1/(1 + 0.143h_cI_{clo}). \quad (8)$$

If the evaporative heat loss required for thermal equilibrium (E_{req}) is less than E_{max}, the skin will be less than 100% wet. If W is the percent of skin surface area wetted by sweat, then

$$W = E_{req}/E_{max}. \quad (9)$$

Increasing values of W produce increasing discomfort. The value of E_{max} is decreased by adding clothing, by increasing water-vapor pressure, and by lowering air velocity. Lowering the humidity, reducing clothing, and increasing air velocity reduce W and physiologic strain and increase thermal comfort.

PHYSIOLOGIC RESPONSES TO THE THERMAL ENVIRONMENT

In environments in which the body tends to lose or gain heat, body temperature changes. Such changes produce physiologic responses aimed at keeping body temperature constant. When body temperature falls, skin temperature tends to fall first, and then (more gradually) deep-body temperature. The first physiologic response is a reduction in blood flow to the extremities and to the skin in general. Peripheral vasoconstriction reduces the convective heat transfer between the skin and the trunk core and causes rapid lowering of the temperature of hands and feet while reducing the loss of heat from the core. Normal skin blood flow is at about 250 ml/min and can easily be reduced to 50 ml/min or less. If further reduction occurs in body temperature, the thermoregulatory system causes an increase in metabolic heat production through involuntary shivering, which can add 100-150 W/m^2 to the basal 40 W/m^2.

If, however, the thermal environment causes body temperature to rise, the initial physiologic response is an increase in blood flow to the extremities and the skin. Vasodilatation increases the convective heat transfer between the skin and the trunk core. The normal skin blood flow of 250 ml/min can be increased to as much as 3,000 ml/min. Effective overall thermal conductance between skin and core is about 18 $W \cdot m^{-2} \cdot °C^{-1}$, with a low of 6 $W \cdot m^{-2} \cdot °C^{-1}$ in the cold and a high

of 100 $W \cdot m^{-2} \cdot °C^{-1}$ in the heat. Increased body temperature also causes the secretion of sweat over most of the skin surface area at rates proportional to the body-temperature increase. Above the sweating threshold, a further 1°C rise in mean body temperature produces sweat secretion at 200-600 $g \cdot m^{-2} \cdot h^{-1}$, corresponding to evaporative heat loss at 150-400 W/m^2. For a more complete and quantitative review of human thermoregulation, the reader is referred to Stolwijk and Hardy.[15]

The involuntary physiologic responses just described are usually associated with feelings of thermal discomfort. Relationships among ambient temperature, thermal pleasantness, comfort, and temperature sensation are illustrated in Figure IV-25. Prediction of thermal sensation, thermal comfort, and thermal acceptability must take into account all the factors involved in heat production, heat loss, and heat transfer; and it must do it simultaneously. In a nearly steady state, this is fairly feasible, but it becomes more complex when body temperature or ambient temperature is changing.

Berglund and Gonzalez[2] evaluated the effect of slowly changing ambient temperature and water-vapor pressure and found that environmental temperature changes of 0.6°C/h from a 25°C starting point were quite acceptable, as were changes in the water-vapor pressure, as long as the instantaneous water-vapor pressure was kept below 16 Torr.

Internal body temperature has a considerable effect on comfort sensation. When the body interior is hyperthermic, as after vigorous exercise, a low air temperature even at high velocity (cold draft) is felt as comfortable and pleasant, although the same cold draft would be extremely uncomfortable to a person who is already slightly hypothermic from a previous exposure to cold. A hypothermic person would prefer very warm air, which in turn would be felt as very uncomfortable by someone in a hyperthermic state.

There is general agreement that age, gender, and physical fitness do not affect preferred ambient temperature, if metabolic heat production and clothing insulation are constant.

When people are exposed to thermal conditions outside the comfort range, the extent of their discomfort is affected by age, gender, and physical fitness, even if activity and clothing are controlled. The extent of discomfort is closely correlated with physiologic thermoregulatory responses. Those who have a vigorous involuntary response, such as sweat secretion on body warming, experience more intensive and earlier discomfort than those whose sweat secretion begins at higher body temperature.

When a space has thermal nonuniformities (such as vertical temperature gradients) or radiant nonuniformities (such as radiant temperature in one direction 10°C higher than in another direction), such nonuniformities can be perceived as uncomfortable, even if the average temperature is in the comfortable zone.

In a cool or thermoneutral environment, increased air movement is felt as uncomfortable; but at temperatures above thermoneutrality, increased air movement is desirable and extends the comfort zone to higher ambient temperatures.

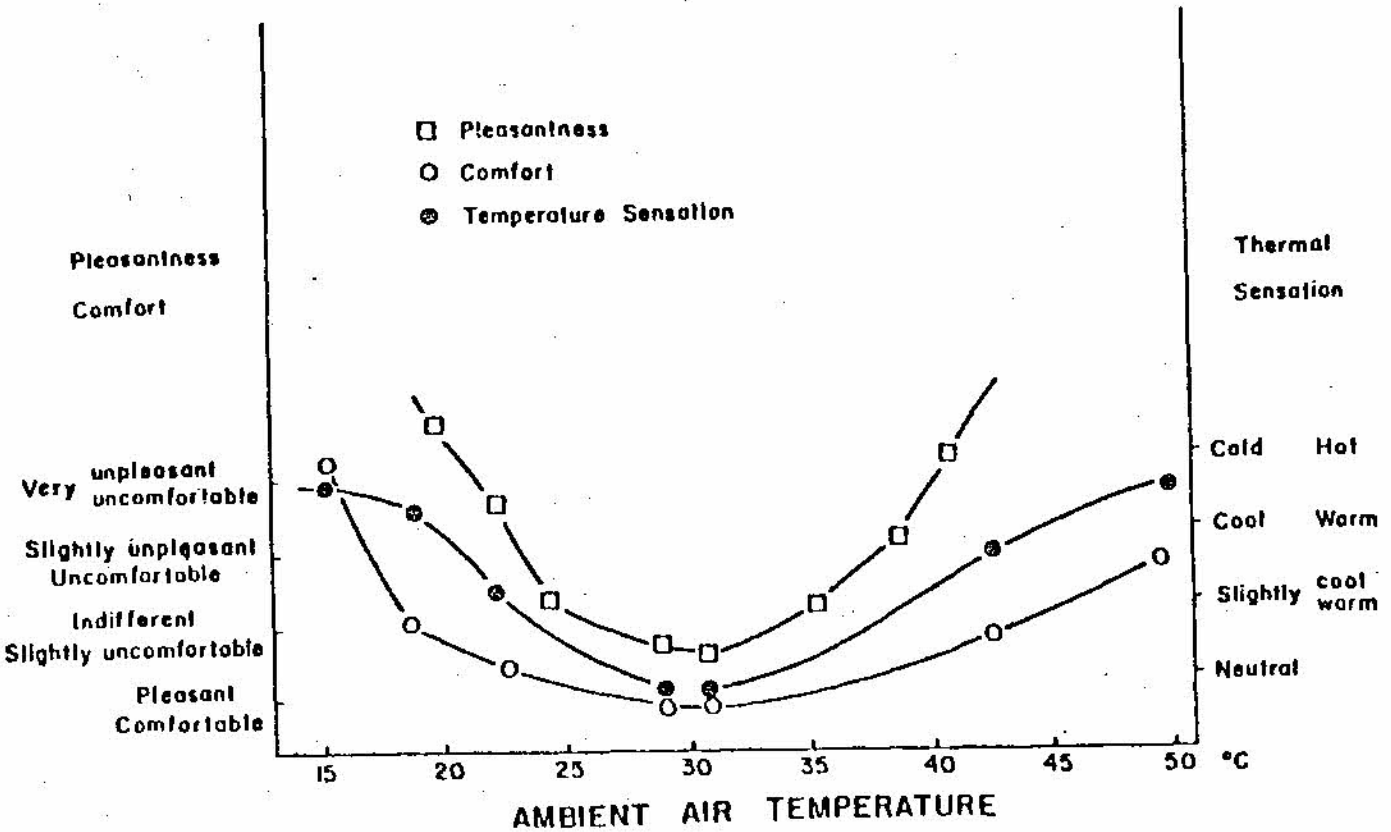


FIGURE IV-25 Reports of pleasantness, comfort sensation, and thermal sensation from sedentary subjects in minimal clothing in environments with low air velocities and uniform air and wall temperatures. Adapted from Hardy et al.[9]

HEALTH CONSEQUENCES OF EXTREMES OF TEMPERATURE AND HUMIDITY

Adverse health effects of extremes of temperature and humidity are in several categories. As extremes of temperature and humidity, we designate operative temperatures below 18°C and above 30°C and humidities below 4 Torr and above 16 Torr water-vapor pressure, for people in conventional clothing and at rest. Any temperature or water-vapor pressure that requires physiologic responses makes demands on the reserves of the systems that regulate cardiovascular function or fluid balance. In healthy people, these responses reduce such reserves, thus perhaps interfering temporarily with work capacity or limiting athletic achievement. In diseased persons, such as those with cardiovascular disease, such reserves are very small or nonexistent; such persons can be seriously threatened by even small excursions of temperature or water-vapor pressure.

Assessments of health consequences of extremes of temperature and humidity tend to be based on two kinds of observations: observations of acutely ill people whose status can be correlated with temperature measurements in their hospital rooms, and observations in epidemiologic studies of whole populations, with daily outdoor temperatures correlated with daily mortality or with the number of emergency-room visits. In the first case, the number of patients observed is relatively small, and each patient has a different disease state; it is difficult to draw very detailed conclusions from such observations. In the second case, very large populations can be observed, but it is difficult to evaluate actual exposure and to assess the pre-existing disease of those who have died.

Clinical reports of particular sensitivity of patients with congestive heart failure to hot and humid environments have been presented by Burch and co-workers.[3-6] Ellis,[7] Schuman,[14] and Oechsli and Buechley[13] have reported on excess mortality in populations exposed to unusually hot weather. This excess mortality occurred particularly among the aged, tne hypertensives, diabetics, and patients with arteriosclerotic and other cardiovascular disease and chronic respiratory disease.

Exposure to low temperatures can produce accidental hypothermia, especially in the aged (Watts;[16] McNicol and Smith[10]). Low winter temperatures are associated with increased mortality,[7] although few studies have specifically addressed this association.

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CHARACTERIZATION OF ADDITIONAL PHYSICAL INDOOR POLLUTANTS

The indoor pollutants thus far described have been defined primarily as airborne contaminants. Other forms of pollution do not depend on mass concentration, such as sound and electromagnetic radiation. Electromagnetic radiation occurs in the radiofrequency,

infrared, visible light, ultraviolet, and x-ray portions of the electromagnetic spectrum. The frequencies of the electromagnetic radiation discussed here range from 10^4 Hz (radiofrequency) to 10^{15} Hz (ultraviolet). Table IV-24 summarizes the frequency distribution for each of these portions of the spectrum and of audible sound, which is transmitted via the vibration of air molecules.

SOUND AND NOISE

Physical Characteristics

The audibility of sound depends on intensity and frequency, with a maximal human response in the region near 3×10^3 Hz. A sound with predominant frequencies below 100 Hz or above 10^4 Hz may require a million times more energy to have the same audibility as a sound with a predominant frequency of 3×10^3 Hz. A method of weighting the pressure exerted by the sound waves at different frequencies has been developed to compensate for these variations. The decibel values (which constitute a logarithmic intensity scale) cited herein are measurements with level A weighting, the scale that most closely matches the response of the human ear. The differences in the treatment of the intensity content of a sound are slight and do not change substantially from one source to another.[14]

Sounds in the indoor environment are generated both outside and inside the occupied space. Table IV-25 gives examples of sound intensities in the outdoor environment. Table IV-26 lists sound intensities produced by typical household appliances in the indoor environment.

Sound intensities are usually measured by a meter satisfying the requirements of American National Standards Institute Specification SI.4-1971 (for sound meters).

Psychophysiologic Effects

The possible effects of sound include permanent and temporary loss of hearing, cardiovascular disease, sleep disruption, and psychologic effects.[14] The physiologic and psychologic responses to sound may be transitory;[14] however, there is insufficient information on the effects of sound by itself or in combination with other stressors. Sound at intensities that are found to be objectionable will affect productivity and decrease enjoyment of the environment.[14]

The EPA has identified sound intensities that, if not exceeded, should protect against some of the adverse effects of sound.[14] These values are expressed in terms of maximal 8-h (75 dB) and 24-h (70 dB) averages required to protect against hearing loss. There are also yearly average long-range environmental goals of 55 dB outdoors and 45 dB indoors, which are recommended to avoid activity interference or annoyance.[12] There is still debate in professional circles about the maximal intensities of short-duration environmental sound that can be

TABLE IV-24

Radiation Wavelengths and Frequencies

Type of Radiation	Wavelength	Frequency, Hz
Ultraviolet		
Ultraviolet C	0.19-0.28 μm	1.07×10^{15}-1.58×10^{15}
Ultraviolet B	0.28-0.315 μm	9.5×10^{14}-1.07×10^{15}
Ultraviolet A	0.315-0.4 μm	7.5×10^{14}-9.5×10^{14}
Visible light	0.4-0.7 μm	4.29×10^{14}-7.5×10^{14}
Infrared		
Near infrared	0.7-1.4 μm	2.14×10^{14}-4.29×10^{14}
Infrared	1.4-3 μm	1.00×10^{14}-2.14×10^{14}
Far infrared	3-1,000 μm	3×10^{11}-1.00×10^{14}
Radiofrequency		
Microwave	1-1,000 mm	3×10^{8}-3×10^{11}
Very high frequency	1-10 m	3×10^{7}-3×10^{8}
High frequency	10-100 m	3×10^{6}-3×10^{7}
Medium frequency	100-1,000 m	3×10^{5}-3×10^{6}
Low frequency	1,000-10,000 m	3×10^{4}-3×10^{5}
Very low frequency	10,000-30,000 m	1×10^{4}-3×10^{4}
Sound, audible	0.016-20.0 m	15-2×10^{4}

TABLE IV-25

Examples of Outdoor Day-Night Average Sound Intensities at Various Locations[a]

Location	Average Sound Intensity, dB(A)
Apartment next to freeway	88
Downtown with some construction	79
Urban high-density apartment	78
Urban row housing on major avenue	68
Old urban residential area	59
Wooded residential area	51
Agricultural cropland	44
Rural residential area	39
Wilderness ambient	35

[a]Data from Council on Environmental Quality.[4]

TABLE IV-26

Examples of Sound Intensities Generated Indoors by Household Appliances[a]

Appliance	Average Sound Intensity, dB(A)
Blender	80-90
Garbage disposer	80
Window air conditioner	60
Refrigerator	45
Vacuum cleaner	70-75
Hair dryer	78
Mixer	82

[a]Data from Jones.[8]

considered safe. However, above 110 dB, sound is so intense that most people experience pain or a tickle in their ears.[11]

Although it is difficult to determine the exact day and night indoor sound intensities, studies have indicated that an intensity of 60.4 dB with a standard deviation of 5.9 dB can be expected in a typical urban residential area, with instantaneous intensities exceeding 80 dB.[12] An expected intensity of 60.4 dB is below the 70 dB recommended by the EPA to prevent hearing loss, but it is well above the intensity recommended to avoid interference and annoyance. Therefore, day and night sound intensities in the 100-site EPA survey may contribute to speech interference, reduced worker productivity, and annoyance.[10(pp. 66-69)]

A high intensity of background noise in urban areas stemming primarily from transportation appears to affect the developing fetus. Women exposed to aircraft noise have a higher proportion of low-birthweight children, who are at higher risk of mortality and both physical and mental effects.[10(pp. 110-111)] This association cannot be separated from the social status of the women (a codetermining variable), inasmuch as many members of the lower social classes live in "noisy" areas.

Exposure to high intensities of sound affects communication and learning, including the acquisition of language.[10(p. 115)] Adaptation or resignation to annoyance may occur, and there do not appear to be groups of people that are particularly sensitive. After-effects of noise have been noted at home and at work, and noise appears to influence aggressiveness and minimize voluntary helping behavior.[10(pp. 120-121)]

RADIOFREQUENCY AND MICROWAVE RADIATION (10^4 to 3×10^{11} Hz)

Physical Characteristics

Although the physical characteristics of all electromagnetic radiation are similar, the frequency is inversely proportional to wavelength, and the effects of the longer wavelengths, such as radiofrequency radiation, are radically different from those of the shorter-wavelength ionizing radiation, such as x rays and gamma rays. The photon energy in radio waves is so small that there is no ionization when it is absorbed in an organism.[15]

Table IV-27 summarizes the radiation properties of some common nonionizing-radiation systems and their expected far-field power densities. Energy radiated by these systems can be additive, provided that the frequencies are within the same octave band.

For the purposes of this report, the densities of all radio-frequency energies generated outdoors are defined as "background power densities," and those of radiofrequencies generated indoors as "generated power densities."

Some radiofrequency energy is generated in the indoor environment. In general, all electric equipment produces some radiofrequency radiation. However, all but a few electric devices radiate energy at

TABLE IV-27

Comparison of Radiation Properties of Some Common Radiofrequency-Generating Systems

Radiating System	Frequency	Average Transmitter Power	Far-Field Power Density at Selected Distance
AM radio transmitter	500 kHz	50 kW (maximum allowed)	0.016 mW/cm^2 at 400 m
FM radio transmitter	88-108 MHz	100 kW	0.03 mW/cm^2 at 500 m
VHF TV	174-216 MHz	300 kW (maximum allowed)	0.04 mW/cm^2 at 500 m
UHF TV	470-890 MHz	5,000 kW (maximum allowed)	0.5 mW/cm^2 at 500 m
Acquisition radar[a] (ARSR)	1 GHz	20 kW	0.05 mW/cm^2 at 3,000 m (near field, 100 mW/cm^2 at 30 m)
Tracking radar:[a]			
Type A	10 GHz	5 kW	0.01 mW/cm^2 at 3,000 m (near field, 800 mW/cm^2 at 9 m)
Type B	1.3 GHz	150 kW	1 mW/cm^2 at 5,000 m (near field, 56 mW/cm^2 at 523 m)

[a]Radar power densities given along line of sight of transmitted power; off axis, power densities are generally 0.01 less than those along line of sight.

well below the American National Standards Institute recommended exposure limits, even in combination with one another. One major exception is the microwave oven. Under normal operating conditions, a residential microwave oven radiates approximately 1 mW/cm^2 at the seal on the door. However, if the door is defective, values in excess of 1 W/cm^2 can be achieved.

Psychophysiologic Effects

Effects of radiofrequency radiation can be divided into two major categories:[15] thermal effects (when the radio-wave energy is converted into heat) and nonthermal effects (which cannot be directly explained by thermal equivalents).

Biologic effects depend on the frequency and the intensity of the radiation; the duration of exposure; the dielectric constant, temperature, and thermal conductivity of the irradiated tissue; the ability of the tissue to dissipate heat; and the dimensions of the body. Absorption of microwave radiation by body tissues results in an increase in temperature, often producing internal burns due to local hot spots caused by nonuniformity in the field. The eyes and testes were found to be the most sensitive.[17]

Specific effects at the cellular or molecular level were postulated more than a decade ago without resolution of the importance of these effects with respect to biologic damage.[3] The possibilities of nonthermal effects, such as rearrangements within macromolecules and subcellular structures, have been under investigation for many years, but further studies will be necessary to clarify the issues. It is relatively clear that metabolic and functional disturbances at the cellular level can be caused by microwave radiation, but the mechanisms of these effects are not yet well understood.

Table IV-28 characterizes the relative rates of absorption by the human body; however, it is difficult to determine the exact effect of each frequency. Because the radiofrequency energy generated indoors is low, the major emphasis should be on outdoor sources. Indoor radiofrequency fields are generally lower than outdoor. Osepchuck has discussed sources of microwave and other forms of radiofrequency energy.[11]

FAR-INFRARED AND INFRARED RADIATION (3×10^{11} Hz to 4.3×10^{14} Hz)

Physical Characteristics

The infrared energy spectrum ranges from far-infrared (3×10^{11} Hz to 10^{14} Hz), through infrared ($1.0\text{-}2.14 \times 10^{14}$ Hz), to near-infrared ($2.14\text{-}4.29 \times 10^{14}$ Hz). Infrared radiation is produced naturally by the sun and by all common heating and artificial-light sources. The incandescent lamp is one of the major sources of infrared radiation and the most common artificial-light source in the indoor environment. Of the total input wattage of an incandescent lamp,

TABLE IV-28

Relative Absorption of Radiofrequencies by Human Body[a]

Frequency, 10^6 Hz	Maximal Absorption by Human Body, %
<400	<50
400-1,000	50-100
1,000-3,000	20-100
3,000-10,000	>50

[a]Data from U.S. Department of Health, Education, and Welfare.[13]

75-80% is converted to near-infrared and infrared radiation.[9] The ACGIH has adopted a TLV of 10 mW/cm^2 for infrared radiation in the workplace.[1] The power density 2 m from a 100-W lamp is approximately 0.6 mW/cm^2 for the total infrared spectrum. Sunlight on the earth's surface produces a flux of about 70 mW/cm^2, of which about half is infrared.

Psychophysiologic Effects

Depending on its wavelength, infrared is absorbed in the surface of the skin (wavelengths larger than 2 μm) or can penetrate several millimeters (wavelengths between 0.7 and 1.5 μm). Safety standards in industrial environments are based on the risk that infrared radiation may induce cataracts in the eyes of persons exposed to excessive infrared radiation, such as glassblowers or open-hearth steelworkers.[16]

Excessive infrared radiation is most easily controlled by shielding the source with reflecting metallic foils.

VISIBLE RADIATION

Physical Characteristics

Radiation in the near-infrared and visible spectrum is produced by many sources, both natural and artificial. Our sense of sight, feeling of well-being, and comfort are all, to a great extent, influenced by visible and near-infrared radiation.

Psychophysiologic Effects

Retinal burns from observation of the sun have been described throughout history. Chorioretinal burns rarely occur from exposure to artificial light, because the normal aversion to high-brightness light sources (the blink reflex) provides adequate protection, unless the exposure is hazardous within the duration of the blink reflex.

Many factors affect the usefulness of visible light. Among the most important are discomfort glare and disability glare. Light sources can cause a reduction in contrast of an image, owing to scattered visible radiation, by adding a uniform veil of luminance to the object. This effect, commonly called "veiling luminance," may cause a reduction in visual performance without physical damage.

Discomfort glare is a sensation of annoyance or pain caused by brightness in the field of view that is greater than that to which the eyes are adapted. It has been shown that the threshold of discomfort glare changes as a function of age.[2] Although discomfort glare does not necessarily interfere with visual performance, it can cause eye strain and contribute to fatigue. Disability glare and ocular stray light influence one's ability to perform a task by artificially veiling

the contrast of the visual target. It is therefore a great contributor to eye fatigue.

ULTRAVIOLET RADIATION ($0.75\text{-}1.58 \times 10^{15}$ Hz; wavelength, 0.19-0.400 μm)

Physical Characterstics

Ultraviolet radiation is divided into three wavelength categories: ultraviolet-A (UV-A), 0.315-0.400 μm; ultraviolet-B (UV-B), 0.28-0.315 μm; and ultraviolet-C (UV-C), 0.19-0.28 μm. All fluorescent lamps emit UV-A, but not UV-B or UV-C. High-intensity discharge lamps produce UV-A, UV-B, and some UV-C. Incandescent lamps produce small amounts of UV-A, and essentially no UV-B and UV-C.

Ultraviolet radiation is measured with specialized radiometric photometers.

Psychophysiologic Effects

UV-B and UV-C are known photocarcinogens.[5] Doses of UV-B and UV-C 10 times the human minimal erythema dose (MED) have initiated squamous cell carcinomas, and chronic continuous exposure to UV-A can also have a carcinogenic effect.[5]

The ACGIH recommends limits on workplace ultraviolet exposure that depend on wavelength and on the duration of exposure.[1] For UV-A, the intensity should not exceed 1 mW/cm^2 for more than 1,000 s, nor should the dose exceed 1 J/cm^2 if given in less than 1,000 s. For UV-B and UV-C, the dose should not exceed about 3-10 mJ/cm^2 in any 8-h period. The degree of hazard seems to be associated with the erythemal efficiency of each frequency.[1,5]

SUMMARY

Ionizing and nonionizing electromagnetic radiation occurs in the indoor environment. This radiation can be harmful, and one cannot always sense its presence.

Sound can generally be heard and in some cases felt. Excessive sound can cause deterioration of hearing acuity and, if extremely intense or prolonged, cause deafness. Background sound in the urban residential environment can exceed the recommended intensities and result in interference and annoyance. Sound of 70-80 dB, commonly found in indoor environments, can inhibit task performance and possibly contribute to aggressive human behavior.[6]

Infrared, far-infrared, and radiofrequency radiation produce no visible or audible evidence of their presence. However, infrared radiation does provide sensory indication of its presence by heating of human tissue. Far-infrared and radiofrequency radiation, however, provide no indication of their presence, unless their power levels are

so high as to increase skin temperature. Heating of human tissue occurs because of the infrared output of incandescent lamps. However, the detrimental effects of this heating have not been fully investigated. Surveys have shown that in several cities 98% of the people are exposed to less than 1 $\mu W/cm^2$ from broadcasting transmitters.[7] However, ultrahigh-frequency television transmitters can radiate radiofrequency pollution to adjacent buildings at 5-200 $\mu W/cm^2$.

Ultraviolet-A, visible light, and near-infrared radiation can produce surface heating of human and animal tissue. These frequencies are of concern because of their ability to affect human performance. The veiling reflections caused by most artificial lighting systems can have substantial influence on human visual performance. Reduction of veiling reflections can increase visual performance and decrease the energy consumed by lighting systems. Transient adaptation (dilation during or immediately after eye movements) is caused by sudden changes in the visual spectrum power. Transient adaptation contributes to eye fatigue and decreased visual performance.

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V

FACTORS THAT INFLUENCE EXPOSURE TO INDOOR AIR POLLUTANTS

The types and quantities of pollutants found indoors vary temporally and spatially. Depending on the type of pollutant and its sources, sinks, and mixing conditions, its concentration can vary by a factor of 10 or more, even within a small area.

Human mobility constitutes an important kind of complexity in the determination of exposure to air pollutants. Human activity patterns differ between midweek and weekend, between one season and another, and between one part of one's lifetime and another. Activity patterns determine when and how long one is exposed to both indoor and outdoor pollutants. Therefore, in reviewing the factors that influence air-pollution exposures, we have specifically separated them into two major components: time (activity) and concentration (location).

Information on the time spent in various activities is summarized first, and then the variations in concentration encountered in different locations. Unfortunately, most of the studies discussed were not longitudinal and thus do not offer information on seasonal differences in time spent indoors and outdoors or on regional differences in activity patterns.

Outdoor concentrations of pollutants and rates of infiltration affect the concentrations to which people are exposed indoors. The emphasis of the second section of this chapter is on geographic variations in outdoor pollution and their impact on indoor pollution. Building construction techniques, as they vary geographically, and their effect on pollution infiltration rates are particularly important. But the measurement techniques available are limited; the need for additional studies is discussed. The rates of infiltration on a neighborhood scale have been studied by only a few researchers. Although their work has focused on energy conservation, their findings can easily be applied to the study of the impact on indoor pollution.

As shown in Chapter IV, there can be large indoor-outdoor differences in pollutant concentrations. Concentrations also vary among indoor locations and from one time to another. In determining total exposure to pollutants, therefore, both indoor and outdoor concentrations must be well characterized. The ways in which building characteristics affect indoor pollution vary with type of pollutant,

type of building, building location and orientation, and even room use within a given building. Building characteristics are the subject of the final section of this chapter.

HUMAN ACTIVITIES

Patterns of human behavior and activity determine the time spent in any specific location, and thus knowlege of them is essential in estimating exposures of populations to pollutants. As indicated by Ott,[51] a large number and variety of studies in which data on human activities were collected from population samples have been completed over the last 50 yr.

When one examines the literature on human activities, the term "time budget" ("zeitbudget," "budget de temps") is encountered often. A time budget produces a systematic record of how time is spent by a person in some specified period, usually 24 h. It contains considerable detail on a person's activities, including the locations in which the activities take place.[66]

One way of obtaining time budget information from the populations surveyed is to ask each respondent to maintain a diary of his or her activities over a 24-h period or longer. In another approach, the so-called "yesterday" survey approach, the interviewer asks each respondent about his or her activities on the preceding day.

Several summaries of the historical development of time-budget research have been published.[11 16 52 70] Ott[51] discussed the literature on activity patterns in the context of estimation of exposure to air pollution.

The Multinational Comparative Time Budget Research Project, launched in September 1964 by a small group of social scientists from eastern and western countries, used common principles for sampling, interviewing, and data coding and tabulation on an international basis. The population sample consisted of nearly 30,000 persons in 12 countries (Belgium, Bulgaria, Czechoslovakia, France, East Germany, West Germany, Hungary, Peru, Poland, United States, Soviet Union, and Yugoslavia). A standardized coding sytem was developed for comparing activities in different countries. The multinational study developed a coding system with 100 categories of activities represented by a two-digit code (from 00 to 99). The activities represented by these codes can be grouped into 10 classes: working time and activities related to work, domestic house work, care of children, purchasing of goods and services, private needs (such as meals and sleep), adult education and professional training, civic and collective participation, sports and active leisure, passive leisure, and spectacles, entertainment, and social life.[69]

The Project yielded a rich data base that has been summarized in a number of tables, figures, and articles.[69] For example, the average time spent by employed men, employed women, and married housewives in various locations in 12 countries is shown in Table V-1. The data show that employed men in the 12 countries spend between 12 h (in Hungary) and 15.2 h (in Belgium) in their homes, whereas housewives spend

TABLE V-1

Time Spent in Various Locations in 12 Countries
(Average Hours per Day)[a]

	Belgium	Kazanlik, Bulgaria	Olomouc, Czechoslovakia	Six cities, France	100 electoral districts, Fed. Rep. Germany	Osnabruck, Fed. Rep. Germany	Hoyerswerda, German Dem. Rep.	Gyor, Hungary	Lima/Callao, Peru	Torun, Poland	Forty-four cities, USA	Jackson, USA	Pskov, USSR	Kragujevac, Yugoslavia	Maribor, Yugoslavia
7.1.1 Employed men, all days															
inside one's home	15.2	13.5	14.3	13.6	13.6	14.2	13.8	12.0	12.9	14.0	13.4	13.6	13.4	12.9	13.0
just outside one's home	0.5	0.7	0.3	0.3	1.0	0.5	0.4	1.0	0.1	0.2	0.2	0.3	0.5	0.5	1.4
at one's workplace	5.0	7.7	5.9	7.2	5.4	5.1	6.8	3.5	6.4	7.0	6.7	6.5	6.8	7.1	6.1
in transit	1.5	2.1	1.6	1.5	1.3	2.2	1.7	2.0	2.5	1.7	1.6	1.5	2.0	1.8	2.2
in other people's home	0.5	0.2	0.3	0.5	0.5	0.6	0.3	0.5	0.5	0.5	0.5	0.6	0.2	0.3	0.5
in places of business	0.7	0.6	0.6	0.5	0.4	0.4	0.6	0.4	0.7	0.4	0.7	0.7	0.4	0.5	0.5
in restaurants and bars	0.2	0.0	0.1	0.2	0.5	0.4	0.1	0.2	0.3	0.0	0.4	0.4	0.2	0.2	0.0
in all other locations	0.4	0.2	0.9	0.2	0.9	0.6	0.3	0.6	0.6	0.7	0.5	0.4	0.7	0.3	0.3
total	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0
7.1.2 Employed women, all days															
inside one's home	17.1	14.6	16.0	15.3	17.0	16.7	16.7	14.5	16.1	15.0	15.4	15.3	14.0	15.0	15.0
just outside one's home	0.1	0.3	0.2	0.0	0.7	0.2	0.2	0.3	0.4	0.1	0.0	0.1	0.1	0.1	0.4
at one's workplace	3.6	6.5	5.1	6.3	3.6	3.6	4.9	6.8	4.4	5.8	5.2	5.0	6.7	6.1	6.4
in transit	1.2	1.6	1.3	1.1	1.1	1.3	1.1	1.4	1.8	1.5	1.3	1.3	1.7	1.4	1.5
in other people's home	0.4	0.3	0.2	0.5	0.4	0.9	0.2	0.3	0.3	0.6	0.7	0.6	0.2	0.6	0.2
in places of business	1.0	0.6	0.8	0.6	0.6	0.8	0.7	0.5	0.7	0.8	0.9	1.1	0.6	0.4	0.4
in restaurants and bars	0.2	0.0	0.0	0.1	0.2	0.3	0.1	0.0	0.1	0.0	0.2	0.2	0.2	0.0	0.0
in all other locations	0.4	0.2	0.4	0.1	0.4	0.2	0.1	0.2	0.2	0.2	0.3	0.4	0.5	0.2	0.1
total	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0
7.1.3 Housewives, all days (married only)															
inside one's home	21.6	20.4	20.9	21.7	20.4	20.5	21.3	19.7	21.0	20.9	20.5	20.9	19.6	20.5	19.7
just outside one's home	0.3	1.4	0.3	0.1	0.8	0.6	0.3	2.1	0.5	0.1	0.1	0.1	0.4	0.8	2.3
in transit	1.0	0.9	1.2	1.0	1.0	1.0	1.0	0.9	1.2	1.2	1.0	0.9	1.9	1.5	1.1
in other people's home	0.4	0.4	0.3	0.5	0.6	0.6	0.3	0.2	0.4	0.5	0.8	0.7	0.7	0.7	0.3
in places of business	0.5	0.7	1.1	0.6	0.7	1.1	0.9	0.9	0.7	1.2	1.2	1.1	1.1	0.4	0.5
in restaurants and bars	0.1	0.1	0.0	0.0	0.1	0.1	0.0	0.0	0.0	0.0	0.1	0.1	0.0	0.0	0.0
in all other locations	0.2	0.1	0.2	0.1	0.4	0.3	0.2	0.2	0.2	0.1	0.3	0.2	0.3	0.1	0.1
total	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0	24.0

[a]Reprinted with permission from Szalai.[69] Data are weighted to ensure equality of days of the week and number of eligible respondents per household.

between 19.7 h (in Hungary) and 21.6 h (in Belgium) in their homes. In the 12 countries, therefore, employed men spend, on the average, 50-63% of the day in their homes, and housewives spend 82-90% of the day in their homes. It is difficult to determine the overall amount of time spent indoors from these data, because categories like "at one's workplace" do not distinguish between indoor and outdoor workplaces. Similarly, the categories "in places of business" and "in all other locations" do not specify whether they are indoors or outdoors. However, if one assumes that all "workplaces," "places of business," and "restaurants and bars" are indoors, along with the category "in other person's homes," and that the category "in all other locations" is assumed to be entirely outdoors, then it is possible to estimate the amount of time spent by respondents in three general categories: indoors, outdoors, and in transit (see Table V-2).[51]

With these assumptions and the restructured data shown in Table V-2, it is estimated that employed men in the 12 countries spend between 84% (in Maribor, Yugoslavia) and 92% (in France) indoors. It should be emphasized, however, that many of the entries in Table V-2 cannot be compared with each other on a statistical basis, because the numbers of respondents in the samples vary. Also, the representativeness varies, because some countries, such as the Soviet Union, are represented by a single city and its suburbs (Pskov, population 115,000), whereas others, such as the United States (44 cities), are represented by a national sample of metropolitan areas. Finally, some assumptions as to whether a location was indoors or outdoors need to be examined, because they may introduce error. However, the estimates in Table V-2 appear useful as rough approximations of the times spent by residents of 12 countries indoors, outdoors, and in transit.[51]

If only the data for the United States (44 cities) are considered, it appears that, on the average, employed men spend 90% of the day (21.7 h) indoors, whereas married housewives spend 95% of the day (22.8 h) indoors. Employed men in the United States are estimated to spend 2.9% of the day (0.7 h) outdoors, and housewives 1.7% (0.4 h).

Although the estimates in Tables V-1 and V-2 are useful for determining the total amount of time spent in various locations, they give little information about the time of day when persons are present in each location. Data from the multinational study can be displayed in a composite profile that shows the proportion of the population that are engaged during the day in selected activities, such as sleeping, eating, working, travel, and watching television (Figure V-1).

In addition to the studies of activities in the United States by Robinson,[57-59] activity-pattern studies have been carried out in Durham, N.C., by Chapin and Hightower,[13] on a sample of 43 Standard Metropolitan Statistical Areas (SMSAs) by Chapin and Brail,[12] on a followup U.S. national sample by Brail and Chapin,[8] and on the Washington, D.C., metropolitan area by Hammer and Chapin.[32] Information supplied in this section is limited to urban areas; this reflects the available published information. No comments are made on variations in numbers, because it is beyond the scope of this document to assess their reliability.

TABLE V-2

Estimated Time Spent in Three Environmental Categories[a]

Country	Average Time Spent, h/d					
	Employed Men			Housewives[b]		
	Indoors	Outdoors	In transit	Indoors	Outdoors	In transit
Belgium	21.6	0.9	1.5	23.2	0.4	0.4
Bulgaria (Kazanlik)	21.0	0.9	2.1	22.1	1.5	0.4
Czechoslovakia (Olomouc)	21.3	1.1	1.6	23.2	0.5	0.4
France (six cities)	22.0	0.5	1.5	23.3	0.2	0.5
West Germany (100 districts)	20.4	1.9	1.7	22.2	1.2	0.6
West Germany (Osnabruck)	20.7	1.1	2.2	22.7	0.7	0.6
East Germany (Hoyerswerda)	21.6	0.7	1.7	23.2	0.5	0.3
Hungary (Gyor)	20.4	1.6	2.0	21.5	2.3	0.2
Peru (Lima-Callao)	20.8	0.7	2.5	22.9	0.7	0.4
Poland (Torun)	21.9	0.4	1.7	23.3	0.2	0.5
United States (44 cities)	21.7	0.7	1.6	22.8	0.4	0.8
United States (Jackson, Mich.)	21.8	0.7	1.5	23.0	0.3	0.7
U.S.S.R. (Pskov)	21.0	1.0	2.0	22.6	0.7	0.7
Yugoslavia (Kragujevac)	21.4	0.8	1.8	22.4	0.9	0.7
Yugoslavia (Maribor)	20.1	1.7	2.2	21.3	2.4	0.3

[a]Reprinted from Ott.[51] Derived from data originally published in Szalai;[69(p. 795)] data are weighted to ensure equality of days of the week and number of eligible respondents per household.

[b]Married persons only.

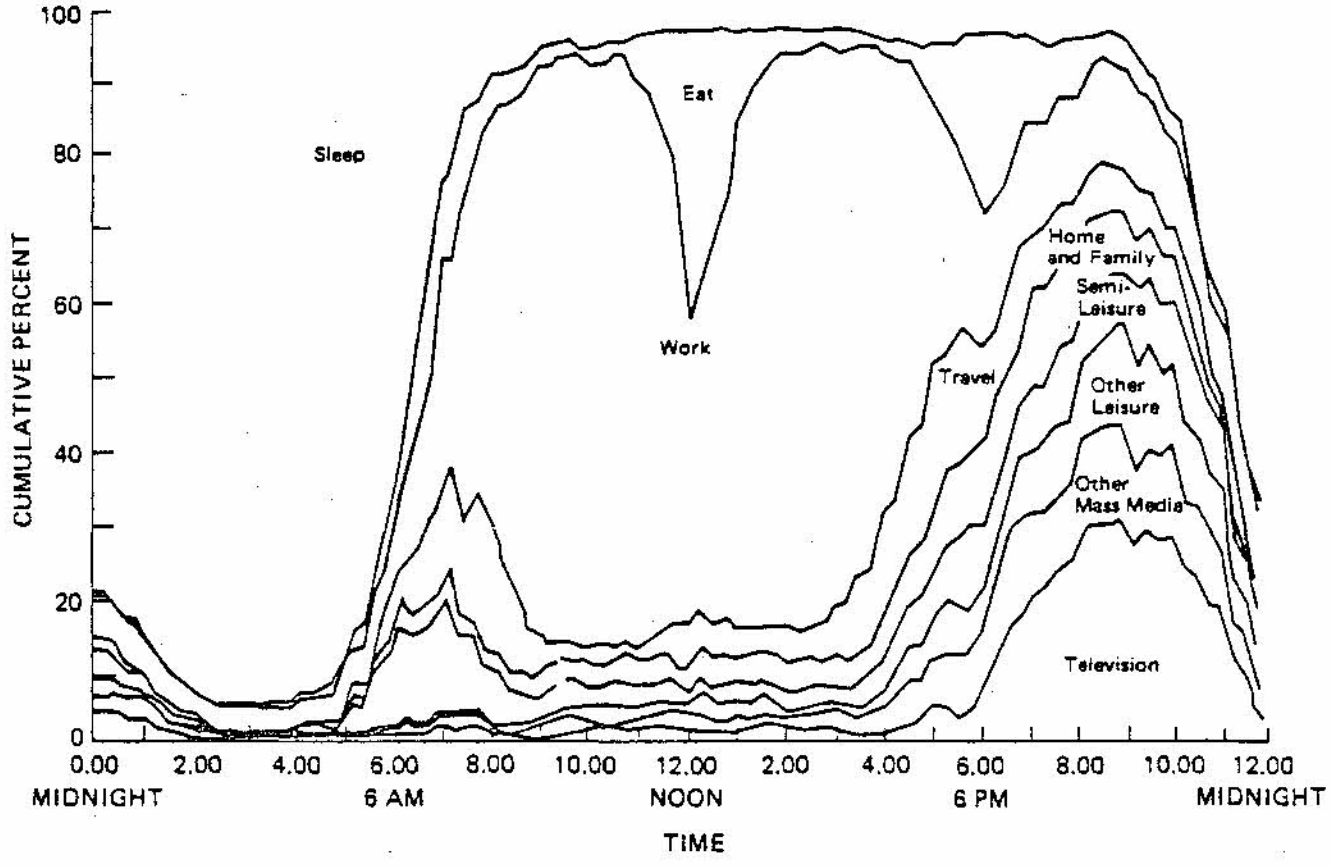


FIGURE V-1 Diurnal profiles showing percentage of employed men in United States (44 cities) engaged in nine types of activities as a function of time of day (weekdays only). Data weighted to ensure equality of days of the week and number of eligible respondents. Reprinted with permission from Szalai.[69]

In the United States, legislation passed in 1952 required urban areas to conduct metropolitan-area transportation studies as a prerequisite for receiving federal funds for highway construction.[64] As a result, transportation studies have been undertaken in 200 areas of the United States,[76] and these studies have usually involved collection of considerable detail about the transportation activities of the urban population, particularly in cities with populations in excess of 50,000. As reported by Robinson, Converse, and Szalai,[60] the multinational research project also collected information on the average time spent in commuting to and from work in various countries (see Table V-3). Most of the summaries of findings from time-budget studies have presented only average values and seldom given histograms or information on the variance of the time spent in various locations or activities.

In 1969-1970, the U.S. Department of Transportation made arrangements with the Bureau of the Census to carry out a nationwide study of the transportation-related activities of the U.S population. This study, called the Nationwide Personal Transportation Study, was based on home interviews and covered individual activities in considerable detail.[3,4,6,26,27,33,56,65-68] Figure V-2 shows a frequency distribution of the amount of time spent in commuting based on these data. Assuming two trips per day, the overall average of 22 min/trip compares reasonably well with the average of 46 min/d reported by Robinson, Converse, and Szalai.[60]

There is a need for a special-purpose activity-pattern study specifically tailored to the problem of estimating air-pollution exposures. Previous activity-pattern studies have not considered questions that apply to exposures to air pollutants. Such a survey should begin with a pilot study on a single city, to perfect the experimental design and data-collection methods, and should use personal monitoring instruments to measure exposures. Once the pilot study is completed and the results are evaluated, a large-scale research investigation could be carried out on a number of cities or on a national probability sample. The large-scale survey would use diaries and personal monitoring instruments to characterize the frequency distribution of air-pollution exposures of the population as a whole and in selected cities. Information from the diaries could be compared with the measurements of exposure to determine how different activities affect population exposure rates.

GEOGRAPHIC AND LOCAL VARIATIONS

The air quality of an indoor environment is often described on the basis of one 24-h average obtained from one indoor sampling location. The spatial distribution of indoor air pollutants within a structure is a little-studied subject. Therefore, recent or current unpublished works and technical papers related to environmental concerns, but not necessarily to indoor air quality, are incorporated in this review. Some of the associations made and conclusions reached are clearly based on explicitly stated assumptions, rather than on scientific documentation.

TABLE V-3

Average Time Spent Traveling To and From Work, by Mode of Transportation[a]

Country	Average Time Spent, min/d			
	Public Transport	Automobile	Walking	All Travel
Belgium	98	55	52	66
Bulgaria (Kazanlik)	93	73	47	57
Czechoslovakia (Olomouc)	73	62	46	59
France (six cities)	82	46	44	50
West Germany (100 districts)	--	--	--	40
West Germany (Osnabruck)	71	41	46	47
East Germany (Hoyerswerda	82	66	30	62
Hungary (Gyor)	104	48	40	64
Peru (Lima-Callao)	103	93	48	89
Poland (Torun)	71	50	41	60
United States (44 cities)	81	46	30	50
United States (Jackson, Mich.)	--	39	34	38
U.S.S.R. (Pskov)	67	--	32	--
Yugoslavia (Kragujevac)	70	53	47	51
Yugoslavia (Maribor)	71	44	40	51
Average	82.0	55.1	41.2	56.0
Standard deviation	13.3	15.1	7.2	12.7

[a]Reprinted with permission from Robinson et al.[60]

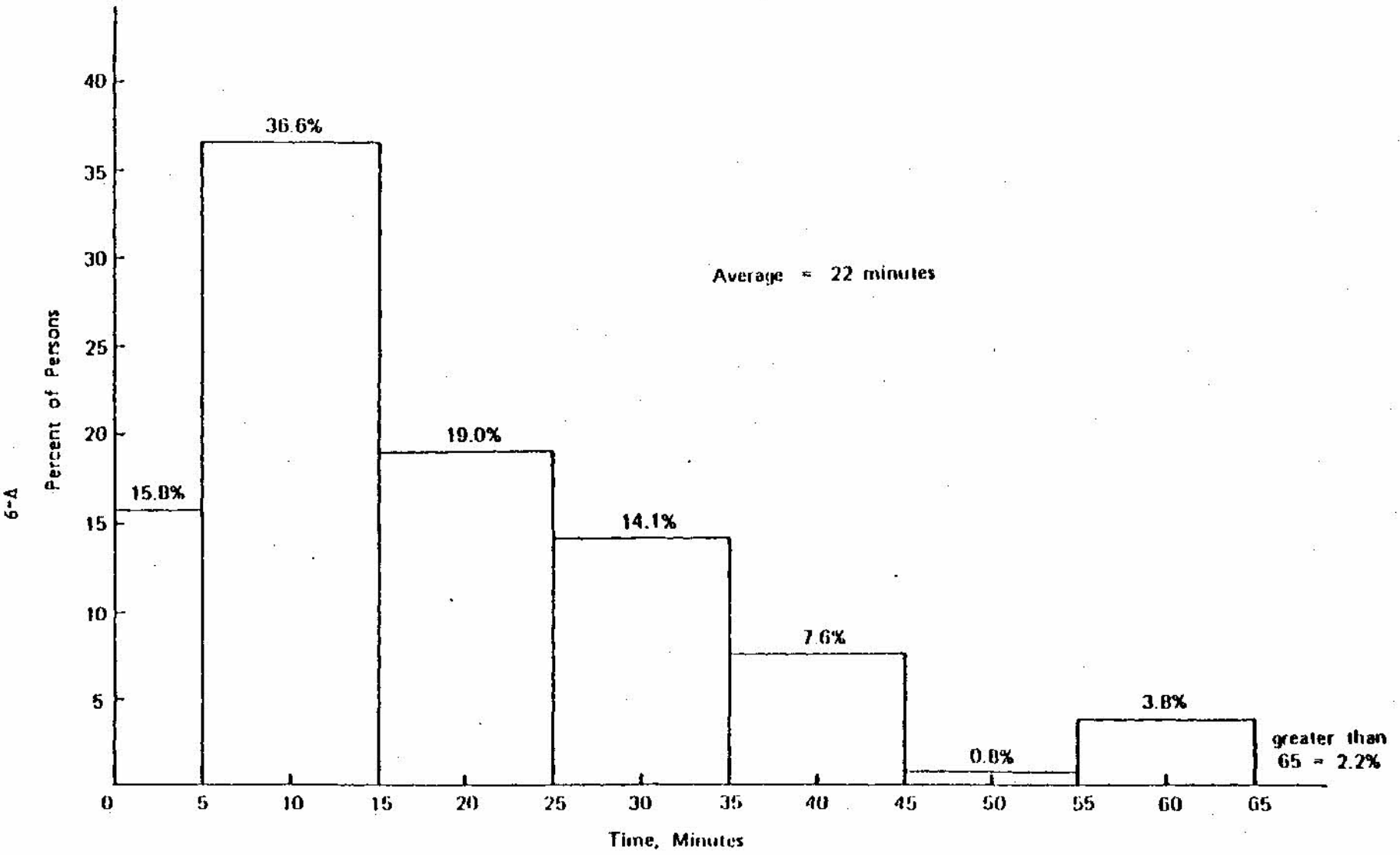


FIGURE V-2 Frequency distribution of home-to-work commuting times for employed persons in the United States (excludes persons who work at home or at no fixed address). Reprinted from Svercl and Asin.[68]

The types of pollutants and the concentrations of each type vary between locations within a structure, between structures within a geographic area, and between geographic areas. This section discusses some of these interrelationships.

GEOGRAPHIC VARIATIONS IN INDOOR AIR QUALITY

Owing to the small number of field monitoring studies, the geographic distribution of indoor air pollutants has not been determined. However, it is instructive to review the geographic distribution of the major factors that affect variations in the concentrations of pollutants and their impact on the quality of the indoor environment. Outdoor air quality, air-infiltration rates, and sources of emission of indoor pollutants are the major factors. Outdoor air quality has been studied with respect to some pollutants, and the geographic distribution of these few pollutants is well understood. Descriptive statistics published annually by EPA and state and local air-quality agencies furnish much scientific information useful in discerning regional and local differences in concentrations of carbon monoxide, total suspended particles, ozone, NO_x, sulfur dioxide, sulfates, and others. Clearly, it is beyond the scope of this document to summarize the existing information on geographic variations in the types and concentrations of all ambient pollutants. It should be noted that the geographic distribution of some criteria pollutants has been studied and is easily accessible from the literature; information on noncriteria pollutants is sparse and often collected and analyzed by questionable methods.

Concentrations of chemically nonreactive pollutants in residences generally correlate with those outdoors. Results from the six-city study,[61] which monitored indoor and outdoor environments for an extended period, clearly showed the influence of outdoor concentrations on the indoor environments (see Figure V-3). Another study,[67,68] sponsored by EPA, supported the conjecture that indoor concentrations of inert gaseous contaminants correlate with outdoor concentrations. The available data base is not large enough to support statistical conclusions, but there is little doubt that the variations in indoor pollutant concentrations correlate with variations in outdoor concentrations. Thus, it is expected that a city with high outdoor pollutant concentrations will have high indoor concentrations, unless control strategies are used. Although this is a broad, general conclusion, it must be emphasized that the indoor concentration of a given pollutant is expected to vary widely among residences within one city. This variation may be sufficient to mask the impact of varying outdoor concentration.

Distribution of indoor air quality is extremely difficult to describe on a geographic scale, because indoor air quality is determined by complex dynamic relationships that depend heavily on occupant activity and highly variable structural characteristics. Weather, which has a regional character, influences indoor air concentrations of some chemicals, such as formaldehyde, and biologic

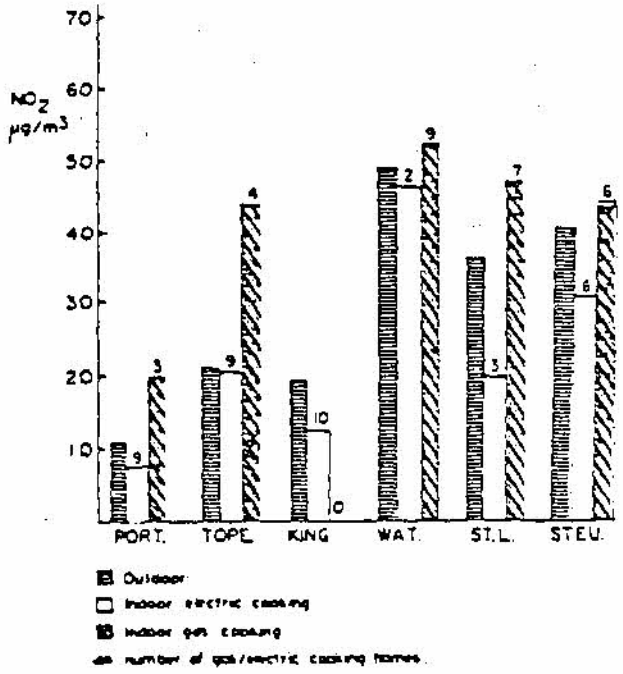


FIGURE V-3 Annual nitrogen dioxide concentration outside and inside electric- and gas-cooking homes, averaged across each community's indoor and outdoor network (May 1977-April 1978). Reprinted with permission from Spengler et al.[63]

contaminants, such as bacteria and molds. Therefore, the influence of relative humidity and other weather-related conditions affecting indoor environmental quality needs to be studied geographically. Research specifically addressed to geographic distribution of indoor air quality is needed.

Other sections of this document address ventilation rates of large buildings, and the discussion of the geographic distribution of air-infiltration rates in this section focuses on residences. Typically, the air-infiltration rate for American residences is assumed to be 0.5-1.5 ach. This assumption is supported by the results of several energy and air-quality studies that experimentally determined the range of ventilation rates for typical residences to be between 0.7 and 1.1 ach (Moschandreas and Morse;[28] C. D. Hollowell, personal communication). Recently built or retrofitted residences had lower infiltration rates, between 0.5 and 0.8 ach (J. Woods, personal communication). However, the sample that yielded the data is small, and statistical documentation for such statements is not strong.

Only one experimental study appears to have been broad enough to allow generalizations on the spatial distribution of air-infiltration rates. A 1979 report[29] presented air-leakage characteristics of low-income residences 10-90 yr old in 14 cities in all climatic zones of the United States. Two measurement techniques were used: a tracer-gas decay technique with air bags to measure natural air infiltration,[28] and a fan-depressurization test that measures induced air-exchange rates.[33] Of the 266 low-income residences tested with these two techniques, 68% were frame buildings, 16% masonry, and 11% masonry-veneer. These proportions do not necessarily reflect those of the universe of low-income dwellings. Figure V-4 illustrates the findings of the study on air-infiltration rates from three cities. With the tracer-gas technique, it was found that 19% of the rates were below 0.5 ach; 40% were moderate, between 0.5 and 1.0 ach; 20% were high, between 1.0 and 1.5 ach; and 20% were very high, greater than 1.5 ach. This characterization of the rates as moderate, high, and very high was given by Grot and Clark and does not reflect a universally accepted nomenclature. Although their paper did not discuss the general geographic distribution of air-infiltration rates, investigations of the data base are continuing. They observed that, the higher the number of degree days, the lower the infiltration rate of the residences. This observation is preliminary (R. A. Grot, personal communication), and further work is needed to verify it. Furthermore, this was a study of low-income residences, not typical residences.

There have been studies that indicate the geographic distribution of residential indoor sources. The residential energy consumption in various geographic regions (Table V-4) shows the use of fuel types that are potential sources of high concentrations of nitric oxide, nitrogen dioxide, and carbon monoxide in residences with gas cooking and heating. A second example shows the number of mobile homes in each state (Table V-5)--an indoor environment with a reported potential for high formaldehyde concentrations. Finally, residences in Polk County, Florida, Grand Junction, Colorado, and Butte, Montana, are built with

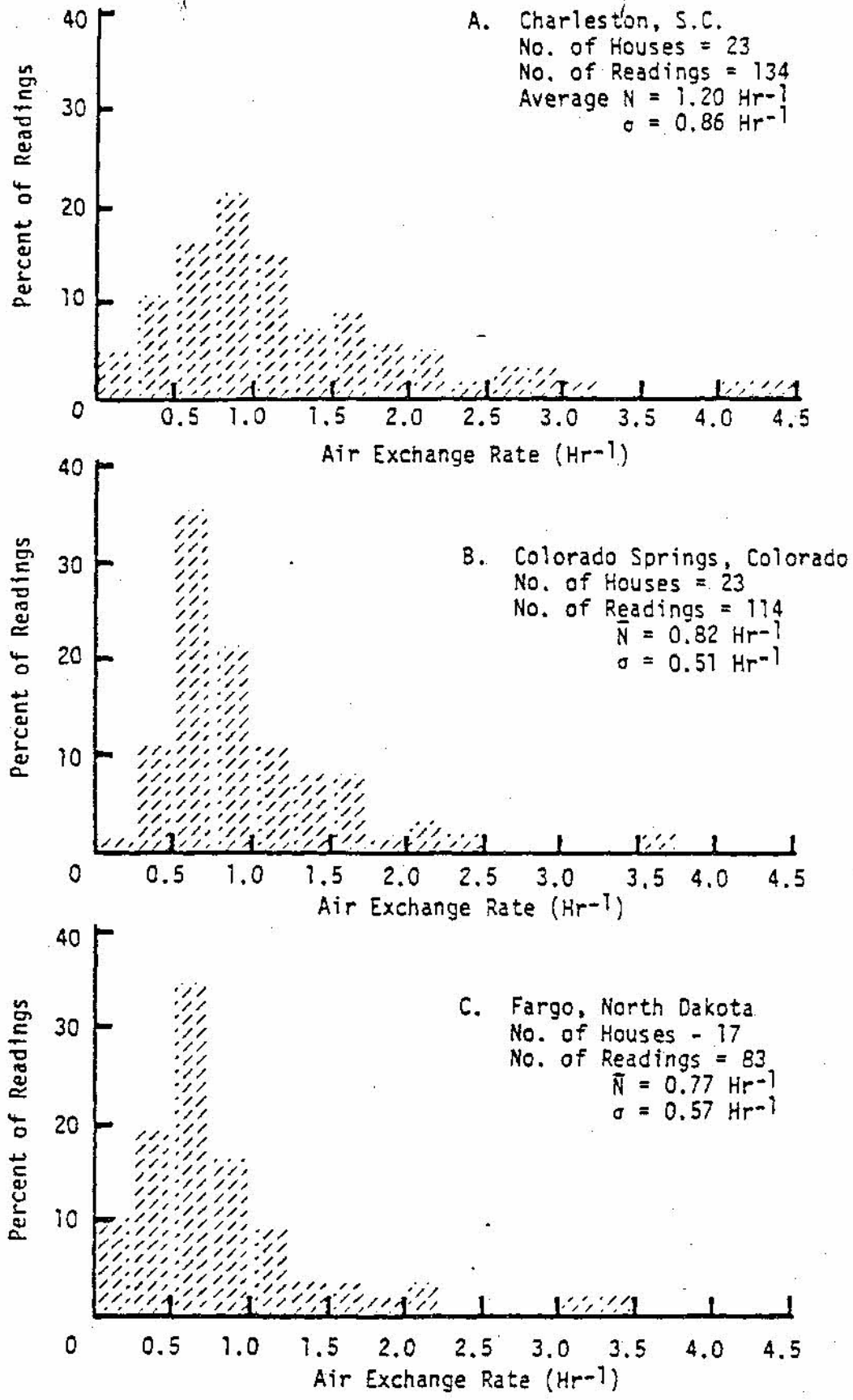


FIGURE V-4 Histograms of measured natural air-infiltration rates for three cities. Reprinted with permission from Grot and Clark.[29]

TABLE V-4

Distribution of Residential Energy Consumption by Fuel Type and Region, 1970 (Single-Family Detached Homes)[a]

Region	Distribution of Fuel Use, %			
	Gas	Oil	Electricity	Coal and Wood
Northeast				
New England	20	76	3	1
Middle Atlantic	46	45	3	6
North central				
East north central	71	23	2	4
West north central	76	20	2	2
South				
South Atlantic	41	39	13	7
East south central	60	4	20	16
West south central	93	—	4	3
West				
Rocky Mountain	81	8	5	6
Pacific	76	12	10	2

[a]Reprinted from Keyes.[37]

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CONSUMER PRODUCT
SAFETY COMMISSION

UNITED STATES GOVERNMENT

Memorandum

U.S. CONSUMER PRODUCT SAFETY COMMISSION

09 SEP 1985

TO: The Commission

THROUGH: Sadye Dunn, Secretary

THROUGH: Daniel R. Levinson, General Counsel

THROUGH: Leonard DeFiore, Executive Director

THROUGH: Andrew G. Ulsamer, Acting Associate Executive Director for Health Sciences

FROM: Sandra Eberle, Program Manager
Chemical Hazards Program

SUBJECT: Indoor Air Quality Staff Documents for Peer Review by the Clean Air Scientific Advisory Committee

Attached for your information are staff documents (Tab A) which are being peer reviewed by the Science Advisory Board, Clean Air Scientific Advisory Committee, (CASAC), of the U.S. Environmental Protection Agency (EPA). The CASAC, at the request of Chairman Scanlon and with the concurrence of EPA Administrator Lee Thomas, (Tab B) will be assisting in the review of the potential for adverse health effects associated with exposure 0.1 to 1.0 plus ppm nitrogen dioxide in indoor air generated by the unvented combustion sources used in the home. The scientific review by the CASAC is intended to facilitate further progress in the establishment of voluntary standards limiting nitrogen dioxide emissions from these appliances.

The CASAC is tentatively scheduled to have an open public meeting to review these documents on September 26 and 27 in room 456, 5401 Westbard Avenue. Please call me if you have any questions on this matter.

Attachment

NOTE: This document has not been reviewed or accepted by the Commission.
Initial _____ Date 9/9/85

SA 6 15(1) Cleared
9/6/85
Mfrs./Privlbls of Products Identified
Accepted by
Firms Notified
Comments Processed.

Report to the
Clean Air Scientific Advisory Committee of the
U.S. Environmental Protection Agency

REVIEW OF NITROGEN DIOXIDE: HEALTH EFFECTS AND EXPOSURE FROM CONSUMER PRODUCTS

SEPTEMBER 1985

U.S. Consumer Product Safety Commission
Directorate for Health Sciences
Washington, D.C. 20207

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Products Identified
Exempted by
Firms Notified
Comments Processed

Consumer Product Safety Commission

Index of CASAC Submission

	Title	Date
1.	Update on Health Effects Associated with NO_2	July 30, 1985
2.	Health Effects of Nitrogen Dioxide	1983
3.	Status Report on Kerosene Heaters (includes update: Health Effects of NO_2; Nitrogen Dioxide Health Assessment 1984)	November 27, 1984
4.	Status Report: Pollutants Generated by Unvented Gas Space Heaters	June 29, 1983
5.	Kerosene Heater Emissions: Estimation of Exposure.	May 19, 1983
6.	Indoor Air Quality Kerosene Heater Testing Documentation	May 18, 1983

4313

UNITED STATES GOVERNMENT

Memorandum

U.S. CONSUMER PRODUCT SAFETY COMMISSION

TO : Andrew G. Ulsamer, Acting AED, HS
THROUGH: Warren K. Porter, Acting Div. Dir., HSPS
Susan Feinman, Branch Chief, HSPS

FROM : Lori E. Saltzman, HSPS

DATE : July 30, 1985

SUBJECT: Update on Health Effects Associated with NO_2 - 1985

Health effects associated with exposure to NO_2 were previously reviewed by the Health Sciences Staff of CPSC in 1983 and 1984. The purpose of this document is to update earlier staff reviews by focusing primarily on recent studies associated with exposure to low level NO_2. This document summarizes the animal toxicology studies, controlled human exposure studies, and epidemiological studies reviewed previously and then addresses more recent studies and interprets them in light of earlier CPSC reports.

Enclosure

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d. Reproductive Effects

e. Carcinogenic Effects

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UPDATE ON HEALTH EFFECTS ASSOCIATED WITH NITROGEN DIOXIDE, 1985

by Lori E. Saltzman

I. INTRODUCTION AND PURPOSE

Data available on the health effects associated with exposure to nitrogen dioxide were reviewed by the Health Sciences staff of CPSC in 1983 and 1984. The purpose of this document is to update earlier staff reviews by focusing primarily on recent studies associated with chronic exposure to less than 1 ppm NO_2.

Nitrogen dioxide (NO_2) is an oxidizing agent commonly found in ambient air. NO_2 is generated along with other nitrogen oxides, particulates, and other gases inside the home by fuel-burning cooking and heating appliances. When NO_2 is inhaled, most of it is absorbed in the respiratory tract where it can cause various biochemical, morphological, immunological, and physiological changes. Existing animal toxicology studies, controlled human exposure studies with NO_2, and epidemiological studies support the conclusion that there are concentrations and exposure patterns of NO_2 which pose risks to human health. Available data also suggest that certain segments of the population (children and individuals with pulmonary disorders, for example) may be more susceptible than the general population to adverse health effects of this pollutant. Although the demonstration of short and long-term effects at low concentrations of NO_2 is not presently

considered definitive, the body of evidence precludes the assumption that there is no significant evidence of harm.

Studies previously reviewed by CPSC which provide evidence of effects at concentrations of NO_2 at 1 ppm or lower are summarized below. (Details of these studies can be found in the earlier CPSC documents.)

II. SUMMARY OF PREVIOUS CPSC REPORTS

While animal toxicology studies cannot be extrapolated to establish levels at which effects would occur in humans, they do indicate a variety of effects from acute, chronic, and intermittent exposures to NO_2 and provide valuable information concerning the mechanism of action of NO_2.

Findings from animal studies indicate the following:

- Biochemical changes in protective lung tissue enzyme levels following exposure to less than 1 ppm NO_2.

- Morphological changes in arithmetic mean thickness of the air-blood barrier, volume density of the alveolar wall tissue, and surface area in one and three-month-old rats exposed to less than 1 ppm NO_2 continuously for one (1) month.

- Morphological changes (Type II cell hyperplasia) in mice exposed to 0.34 ppm NO_2 intermittently for 6 hours/day, 5 days/week, for six (6) weeks.

- Morphological and biochemical changes in animal lung; i.e., the development of emphysema-like lesions and increased urinary hydroxyproline excretion* following chronic exposure to 1.1 ppm NO_2 for six (6) months; the latter finding suggestive of increased lung collagen metabolism.

- Immunological alterations, specifically increased susceptibility to infection following continuous and intermittent exposure to concentrations of NO_2 less than 1 ppm.

- Increased melanoma metastases in mice following inhalation of 0.4 ppm NO_2 for ten (10) weeks (8 hours/day, 5 days/week) (p less than 0.005, 21 days post infusion).

There are several controlled studies which suggest that humans, particularly individuals with pre-existing pulmonary disorders, exposed to low concentrations of NO_2 experience adverse health effects such as measurable changes in pulmonary function and metabolism, and increased

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* Hydroxyproline is a unique amino acid found in collagen, the major connective tissue protein in lung.

gas appliances alone caused increased respiratory illness. Furthermore, since other pollutants (CO, SO_2, CO_2, or formaldehyde, for example) may also be released from gas ranges, any reported health effects cannot be attributed solely to a single pollutant.

Because the effects of NO_2 on the respiratory system have been most extensively studied and are the main concern in terms of both acute and chronic health effects, this update will, for the most part, review recent key studies pertaining to respiratory system effects of NO_2.

III. EVALUATION OF EFFECTS ATTRIBUTABLE TO NO_2 EXPOSURE IN RECENT STUDIES

A. ANIMAL STUDIES

Because controlled human experimentation is limited to the investigation of relatively mild, readily reversible short-term health effects, animal studies have been employed to study wide ranges of concentrations, exposure regimens, and biological parameters.

1. Biochemical Changes

Since NO_2 is an oxidant, biochemical studies have focused on responses to oxidant stress. These studies have examined changes in glucose metabolism, lipid peroxidation and antioxidant activity; as well as connective tissue changes in the lung that correlate with morphological changes (see section 2 Morphological Changes). It is likely that the connective tissue changes are mediated by the products

of recruited and activated inflammatory cells in response to the oxidant (NO_2) injury (Gadek et al, 1984; Kleinerman and Ip, 1979).

Peroxidation of biological membrane lipids is considered to be an integral part of cell damage. Evidence that NO_2 causes lipid peroxidation has been demonstrated and reviewed previously (Sagai, et al, 1981 and Ichinose and Sagai, 1982; reviewed in CPSC, 1983a). In these same studies, the investigators also reported that elevation of antioxidative protective enzymes involved in the reduction of lipid peroxides is in response to lipid peroxidation occurring (in rats) following acute and subacute exposure to low concentrations of NO_2 (0, 0.4, 1.2 or 4 ppm NO_2 for 1-16 weeks). These investigators recently examined the relationship between changes in lipid peroxidation and the antioxidative protective system in lungs of rats exposed to NO_2 for their life span (Sagai et al, 1984). Lung tissue from JCL: male Wistar rats (12 per group) continuously exposed to 0, 0.04, 0.4, or 4 ppm NO_2 for 9 or 18 months was assayed for lipid peroxidation (by measuring thiobarbituric acid, TBA) and antioxidative enzymes (glutathione peroxidase, glutathione reductase, glucose-6-phosphate dehydrogenase, 6-phosphogluconate dehydrogenase, glutathione S-transferases, superoxide dismutase, and disulfide reductase).

Lipid peroxide increased significantly in rats exposed to 4 ppm NO_2 for 9 months and in rats exposed to 0.4 and 4 ppm NO_2 for 18 months. The activities of the main antioxidative protective enzymes in the lungs of rats exposed to 0.4 or 4 ppm NO_2 showed a tendency to decrease with exposure from 9 and 18 months. Glutathione peroxidase and aryl- and

aralkyl-S-transferase activities decreased significantly below control levels after 18 months of exposure to 0.4 or 4 ppm NO_2. The percentage ratios of glutathione reductase, glucose-6-phosphate dehydrogenase, or 6-phosphogluconate dehydrogenase activities in the NO_2 exposure groups to their corresponding control group after the 18 month exposure were also generally lower than percentage ratios after the 9 month exposure. This was especially apparent in the higher exposure groups. These results confirmed earlier observations (Ichinose and Sagai, 1982) that the formation of lipid peroxides and the activities of the antioxidative protective enzymes varied inversely. More importantly, these results demonstrated a decline in the ability of the antioxidative system to protect the lung following chronic (18 month) exposure at concentrations as low as 0.4 ppm NO_2.

2. Morphological Changes

The morphological effects of NO_2 have been the subject of numerous investigations (reviewed in previous CPSC reports). Some of these studies are important in that their results have formed the basis for investigations involving humans. These studies have investigated the role of NO_2 exposure in producing alterations in the lung connective tissue matrix, particularly alterations in the tissue proteins collagen and elastin. Alterations in these moieties can be correlated with anatomical changes in the lung and pathological conditions such as emphysema. One animal study for example, reported emphysematous lesions and increased urinary hydroxyproline (a major amino acid in collagen) excretion following exposure to 1.1 ppm NO_2 (8 hours/day) for six (6)

months (Drozdz et al 1977 reviewed in CPSC, 1983). The authors concluded from this study that chronic low level exposure to NO_2 results in emphysema and that increased levels of urinary hydroxyproline are consistent with degradation of lung collagen. Despite the fact that excretion of hydroxyproline is not specific for lung collagen, one may speculate that under these experimental conditions, it derives from this source.

Although changes in collagen are difficult to characterize (since degradation and synthesis of this protein may occur simultaneously) in vitro studies have shown that a significant fraction of newly synthesized collagen is subject to intracellular degradation immediately after or during the synthesis of collagen (Berg et al, 1980). Animal studies, such as the one mentioned above, correlating morphological changes with a biochemical indicator such as urinary hydroxyproline excretion are important, in that they may support the use of this measurement to easily assess effects of NO_2 exposure in humans (see section C. Epidemiological Studies).

In an abstract presented at the 1985 Annual Meeting of the American Thoracic Society, Gordan et al (1985) reported that taurine (2-amino ethane sulfonic acid), found naturally in most tissues, protects against some forms of NO_2 lung injury. Light and transmission electron microscopy (TEM) analyses of the bronchioles of lungs from rats and hamsters pre-fed taurine and exposed to either 7 or 30 ppm NO_2 for 24 hours, showed that taurine prevented the appearance of peribrochiolar inflammatory infiltrates and the reduction, disorganization, and

fragmentation of tight junction fibrils normally observed following NO_2 exposure. The data suggest that dietary supplementation of taurine may be adequate to eliminate the alterations induced by NO_2 or possibly other pollutants causing oxidant injury. It is reasonable to assume that since taurine protects against oxidant injury following exposure to high concentrations of NO_2 (7-30 ppm), it may do the same at lower NO_2 concentrations.

3. Functional Changes

In recent years, toxicological studies have begun to focus on how various noxious environmental substances impair an organism's ability to function properly. Recent studies have examined the effects of NO_2 exposure which relate to functional capacity and will be reviewed here. These effects include physiological, immunological, pharmacological, reproductive, carcinogenic, and metabolic. Although our interest is primarily in the effects of low levels of NO_2 (below 1 ppm), studies in which NO_2 concentrations greater than 1 ppm were used will be included in this update because they contribute to a more complete understanding of the mechanisms of NO_2 toxicity.

a. Physiological Effects

In a study designed to investigate the long-term consequences of acute lung injury during critical growth periods of the lung, three-day-old (newborn) (n=6) and 21-day-old (young) (n=8) male hamsters were exposed to 30 ppm NO_2 for seven (7) days (Lam et al, 1983).

bronchial responsiveness and symptomology. Controlled human exposure studies, while providing accurate measurements of exposure levels, are limited to examining short-term exposures to a single pollutant or simple combinations of pollutants and, therefore, may over or under-estimate the effect that might be observed with complex mixtures of pollutants in real-life situations.

The major health concern regarding chronic low level exposure to NO_2 is the possible occurrence of irreversible lung damage. Indeed, there could be injury to the lung without initially eliciting detectable changes in the pulmonary function. Therefore, it is important to confirm the use of markers to detect or prevent permanent damage from NO_2 inhalation. In an epidemiological study of 70 workers exposed to 0.4-2.7 ppm NO_2 for 6-8 hours/day for 4-6 years, although there were no reported clinical symptoms or changes in the pulmonary function, there was a significant increase in urinary hydroxyproline (potential marker), suggesting increased lung collagen metabolism. This observation is of particular interest because it can be related to a study in which guinea pigs exposed to 1.1 ppm NO_2 for 8 hours/day for six (6) months had increased urinary hydroxyproline excretion and developed emphysema-like lesions. The available epidemiological studies on exposure to low levels of NO_2 from the use of gas ranges (and unvented kerosene heaters in one study) suggest an association in children between increased respiratory illness and symptoms (and subtle but detectable effects on lung function in a few studies) and the use of gas appliances. This association is, however, not definitely proven since there are no consistent findings within or between studies indicating that the use of

Age-matched control animals (n=7, newborn and n=7, young) were exposed to room air which was monitored for NO_2. Room air concentrations were less than 1 ppm NO_2 (monitoring method not specified), however, at less than 1 ppm the concentration precision is open to question. Physiologic and morphometric parameters (lung volumes, static deflationary pressure-volume curves, mean linear intercept, and internal surface area) measured when the animals were one year of age differed in newborn hamsters exposed to 30 ppm NO_2 compared to age-matched controls. These differences approached statistical significance (p less than 0.10). In the young exposed group there were no differences for any physiologic and morphometric parameters measured when compared to the age-matched controls. Exposed animals killed at three and seven days of exposure showed a mild bronchiolitis with epithelial hyperplasia and minimal peribronchial inflammation. These histologic changes appeared to be reversible since exposed animals killed at one year of age appeared microscopically similar to age-matched controls; that is, no emphysema, pneumonia, or bronchiolitis was observed.

The authors suggest from their data that the infant lung may be more susceptible to injury from NO_2 that is manifest at a later age since mild injury imposed during the newborn period results in physiologic and morphometric (but not histological) changes in the mature animals similar to those seen in the early stages of emphysema. This study in hamsters is not incompatible with the findings of human clinical and epidemiological studies which suggest that respiratory problems in infancy can have permanent sequelae (Burrows et al, 1977, Kattan et al, 1977).

In an experiment designed to determine the effects of chronic exposure of NO_2 on arterial blood parameters [pHa, $PaCO_2$ (arterial carbon dioxide tension), and PaO_2 (arterial blood oxygen tension)], rats exposed to 0.4 ppm NO_2 for 9 or 18 months had significantly decreased PaO_2; thus, demonstrating that hypoxic conditions can result from long-term low level exposure in NO_2 (Suzuki et al, 1980). One possible explanation for this hypoxia is that the decrease in PaO_2 was caused by thickening of the alveolar wall which hindered oxygen exchange across the wall. Also, Yoshikawa et al (1982a,b) reported an increase in TBA (thiobarbituric acids, a measurement of lipid peroxidation) reactants in rats with a decrease in PaO_2. These studies demonstrate that rats exposed to 0.4 ppm NO_2 for 9 or 18 months are chronically hypoxic and suggest that increased lipid peroxidation may be stimulated by chronic hypoxia.

b. Immunological Effects

A large number of investigations have been undertaken in an attempt to ascertain if the immunological defense system of the body is compromised by exposure to NO_2 (reviewed in CPSC, 1983 and 1984). These studies have suggested a variety of immunological effects, the most notable of which, are that: 1) various concentrations and exposure patterns may influence the immunological response and 2) animals exposed to chronic and intermittent low levels (less than 1 ppm) of NO_2 have reduced resistance (increased mortality) to respiratory infection (Gardner, 1980, reviewed in CPSC, 1983). An overview of animal infectivity studies suggest a relationship between length of exposure,

(continuous or intermittant), and increased mortality from bacterial infection perhaps resulting from altered host defense mechanisms.

Since the 1984 CPSC report, there have been no new low level immunological studies, however, a study indicating an enhanced phagocytic response which returned to control levels upon repeated exposures to NO_2 will be reviewed here as an adjunct to the immunological studies previously reviewed by CPSC (1983a and 1984).

Sone et al (1983) examined the effects of in vivo and in vitro NO_2 exposures on the phagocytic and tumoricidal capacities of alveolar macrophages. Macrophages obtained by bronchoalveolar lavage from 8-10 week old male Fischer rats one day after being exposed to 40 ppm NO_2, 4 hours/day for one day or for seven days demonstrated an increased ability to phagocytize opsonized sheep red blood cells in vitro, as compared with macrophages from unexposed rats, with one day of exposure resulting in a greater increase in phagocytic activity (p less than 0.01) than seven days (p less than 0.05). There was no increase in the phagocytic response of macrophages after 14 days of exposure. Repeated 4 hour/day 40 ppm NO_2 exposures (at days 1, 7, or 14 prior to lavage) resulted in phagocytic activity similar to control levels, with some instances of increased phagocytic activity, but not to the extent of that observed for a single four hour exposure. Alveolar macrophages lavaged from rats 24 hours after a single four-hour exposure to 40 ppm NO_2 were significantly (p less than 0.01) more cytotoxic toward syngeneic mammary adenocarcinoma cells in vitro than macrophages lavaged from unexposed rats or from rats exposed to 40 ppm NO_2, 4 hours/day for

7 or 14 consecutive days. Rats exposed repeatedly to 40 ppm NO_2, 4 hours/day at 1, 7 and 14 days prior to lavage showed no increase in tumoricidal activity (cytotoxicity).

A dose-related and time dependent enhanced cytotoxic response was observed in alveolar macrophages lavaged from normal rats following *in vitro* exposure to NO_2. Increased tumoricidal activity occurred after a two-hour exposure to 10 or 20 ppm NO_2; however, 40 ppm NO_2 did not result in increased activation. A two- or three-hour exposure to 10 ppm NO_2 resulted in a significant increase ($p < 0.01$) in tumoricidal activity as compared with unexposed (control) or one-hour exposed macrophages.

These results indicate that acute *in vivo* NO_2 exposures enhance immunological reactivity, while multiple NO_2 exposures after the initial exposure and chronic exposure does not. Thus, while NO_2 may act to initially stimulate macrophage-mediated immune defenses, these defenses subsequently return to control levels upon repeated or continuous exposure.

c. Pharmacological Effects

The nonrespiratory functions of the lung impinge on the regulation of blood pressure, both within the lung and peripherally. The ability of the lung to regulate local blood flow by regulation of vessel diameter is essential to maintaining proper pulmonary function as well. The removal or metabolism of circulating prostaglandins from the blood

by the lung (a nonrespiratory function of the lung) serves to lower blood pressure. Therefore, one could speculate that if toxic substances such as NO_2 interfere with this system, the resulting consequences might not be readily detected by conventional pulmonary function tests (Menzel et al, 1980).

In a study by Menzel et al (1980), rats were exposed either to clean air or to 0.2, 2.0, or 19 ppm NO_2 for a single 3 hour exposure and conversion of radiolabeled prostaglandin E_2 (PGE_2) to its metabolite, 15-keto PGE_2 was studied in a perfused lung preparation (method described in Charles et al, 1978) at various times postexposure. Inhibition of conversion of PGE_2 to its metabolite was produced by exposure to 0.2, 2, or 19 ppm NO_2, with maximal inhibition (40-60%) occurring at 18 hours postexposure. This result suggests a delay between exposure and effect, perhaps explaining the possible lack of measurable effects in acute short-term controlled human clinical studies (see section B Controlled Human Exposure Studies). Recovery from inhibition (restoration of conversion of PGE_2 to its metabolite) required 70 hours following a single exposure to 0.20 ppm and 90 hours following exposure to 2 ppm. Recovery, however, was not achieved even 160 hours after a single exposure to 19 ppm. The increased time required for recovery to normal function suggested to the author that repeated exposures to NO_2 over a 24 hour period may be cumulative with respect to magnitude of effect and that if these alterations in PGE_2 metabolism persist on chronic exposure, significant pathophysiological consequences (ventilation: perfusion mismatch) following exposure to low levels could exacerbate or even initiate chronic obstructive pulmonary disease.

d. Reproductive Effects

An investigation was undertaken to study the testicular changes in rats after chronic exposure to 1 ppm NO_2 based on previous findings (Kripke et al, 1976, 1977 cited in Kripke and Sherwin, 1984) that prolonged exposure to subanesthetic concentrations of nitrous oxide (N_2O) has a depressive effect on the reproductive and hematopoietic systems of small animals and humans (Kripke and Sherwin, 1984).* In the 1984 study, eleven male LEW/f mai rats were exposed to filtered air with or without 1 ppm NO_2 for 7 hours/day, 5 days/week for 21 days. The authors (without showing data) reported no difference between the filtered air (n=5) and NO_2 (n=6) exposed rats with regard to spermatogenesis, germinal cell atrophy, or abnormality of the interstitial cells. These results suggested to the authors that the impaired spermatogenesis demonstrated in the earlier studies resulted from N_2O alone. Other impurities (NO), however, were not ruled out.

e. Carcinogenic Effects

Results from previous studies have indicated that intermittent NO_2 exposure at concentrations of 0.4-0.8 ppm for a period of 400 hours or more will facilitate metastases of cancer in exposed animals at a significantly higher incidence than controls (Richters, 1981 and

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*USP purity standards state that N_2O may contain impurities of up to 1 ppm each of other oxides such as NO^2 or NO_2.

Richters and Kuraitis, 1981, reviewed in CPSC, 1984). In the present study (Richters and Richters, 1983), extended clean air periods were introduced between NO_2 exposure episodes to determine if this would alter the previously reported incidence of lung metastases. Five-week-old C57B1/6J mice (n=31) exposed intermittently to 0.4 ± 0.05 ppm NO_2 7 hours/day, 5 days/week* and then infused with B16 mouse melanoma had more nodules than control animals (n=29) exposed to filtered clean air, but the difference was not significant ($p = 0.747$). Mice (n=30) exposed intermittently to ambient room air [containing NO_2 (concentrations between 0-0.12 ppm), O_3 (concentrations between 0-0.07 ppm), as well as other pollutants] had a statistically significant ($p = 0.008$) increase in the number of nodules compared to the filtered clean air group. The number of nodules in different lobes of the lung were also compared, in the NO_2 exposed group there were significantly more melanoma nodules in the right upper and lower lobe compared to controls, while the ambient room air group showed significantly more nodules in all right lobes (upper, middle, lower). There were no statistical differences between the left lobes and the cardiac lobes among the three groups.

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*NO_2 exposure regimen: 1 week of filtered clean air (NO_x and O_3 free) followed the initial 6 week NO_2 exposure and each of two subsequent 3 week NO_2 exposures for a total NO_2 exposure of 420 hours. After the third clean air exposure the mice were infused with B16F10R1 melanoma cells. Animals were killed 3 weeks later at which time their lungs were removed and the number of melanoma nodules counted.

Analysis of the upper quartile data* showed that both the NO_2 and the ambient air groups have significantly higher numbers of nodules than the controls which the authors interpreted as indicating that animals showed different susceptibilities to NO_2 and other pollutants. Using upper quartile data and comparing left lung vs. right lung, the NO_2 exposed and the ambient room air group mice showed significantly greater number of nodules than clean air control mice. Upper quartile analysis also indicated significant differences in the right lower lobe when analyzed separately. The authors, therefore, suggested that the left lung and the right lower lobe may be the most vulnerable portions of the lung and that the more sensitive animals are detected by upper quartile analysis. Furthermore, the authors postulated that since highly sensitive populations of both the NO_2 exposed and ambient room air mice developed a greater number of nodules in their lungs than controls (but only when analyzed by upper quartile analysis), extended clean air periods between ambient NO_2 exposure may permit partial recovery or reduce the severity of the NO_2 effects in the less sensitive animals. This theory, however, remains to be confirmed.

f. Metabolic Effects

A recent study by Mochitate et al (1984) reported on the effects of NO_2 on the components of the microsomal monooxygenase system

*Statistical method not routinely employed in analyzing data. Analysis is carried out on data derived from the area of the distribution curve associated with the top quartile (25%).

(specifically NADPH -cytochrome c reductase activity and the cytochrome P-450 content) in liver and lung microsomes of 23 to 26-week-old male JCl: Wistar rats continuously exposed to 4.0 ± 0.08, 1.2 ± 0.06, and 0.40 ± 0.04 ppm NO_2 for seven (7) days.* NADPH-cytochrome c reductase activity and the cytochrome P-450 content was reduced in liver microsomes at all exposure concentrations, but not in a dose-dependent fashion; for example, at 4.0 ppm, the NADPH-cyctochrome c reductase activity and the cytochrome P-450 content were 82% (p less than 0.05) and 72% (p less than 0.05) of the control levels, respectively, while at the concentration of 1.2 ppm the levels were 89% (not statistically significant) and 78% (not statistically significant) and at the concentration of 0.4 ppm, the measured levels were 84% (not statistically significant) and 75% (p less than 0.05) of the control levels, respectively. Alterations in the lung were different from those of the liver; at the concentrations of 4.0 ppm and 1.2 ppm the cytochrome P-450 contents were 60% (p less than 0.01) and 64% (p less than 0.01) of the control, respectively. Exposure to 0.4 ppm NO_2, however, did not cause a significant decrease in cytochrome P-450 content in the lung. For all exposure concentrations, the activity of NADPH-cytochrome c reductase in the lung was slightly higher (not statistically significant) than in control rats. These findings suggested to the authors that both measured microsomal monooxygenase system components of rat liver are lowered upon exposure to low concentrations of NO_2 and that NO_2 inhalation is specifically

*This system is complex and consists of many components which are involved in the activation as well as the inactivation of various xenobiotic compounds.

deteriorative to cytochrome P-450 content in the lung but only at higher concentrations (4 ppm and 1.2 ppm). This hypothesis, however, was not always statistically confirmed. Since alterations in the microsomal monooxygenase system can stimulate both the formation of toxic, reactive intermediates from xenobiotic compounds as well as their (xenobiotic) detoxification (depending upon the compound), exposure to NO_2 may effect an organism's ability to process foreign materials. Therefore, it can be postulated that in some instances exposure to NO_2 may decrease an organism's ability to detoxify potentially harmful compounds, and the effect of NO_2 exposure would then be interpreted as adverse.

B. CONTROLLED HUMAN EXPOSURE STUDIES (CHAMBER STUDIES)

Several controlled human studies suggest that short-term peak exposure to low concentrations of NO_2 may cause adverse health effects, particularly in sensitive populations such as asthmatics. Although the demonstration of effects at low concentrations is not presently considered definitive and the available data are not adequate to allow for the determination of a threshold of effects, the body of evidence precludes the assumption that there is no significant evidence of harm. These clinical studies have used various respiratory measurements to assess health effects associated with NO_2 exposure: routine pulmonary function tests; tests of pulmonary mechanics (compliance and airway resistance); increased responsiveness of the airways to a variety of stimuli (occurring even in the absence of altered pulmonary mechanics); and the elicitation of symptoms. These tests are designed to assess the various respiratory functions of the lungs (i.e., volumes, mechanics, distribution of ventilation, and gas exchange).

The interpretation and use of these measurements is confounded by a number of issues: 1) the sensitivity of any one or a combination of tests in detecting specific changes varies with specific pulmonary disorders; 2) inter- and intra-subject variability of routine pulmonary function tests necessitating a large range of clinically normal values; 3) the normal healthy respiratory system has a large reserve capacity and fairly large changes in pulmonary function can occur without substantially impacting on the day-to-day functioning of these individuals; while individuals with respiratory problems do not have these large reserves and even small changes in pulmonary function may adversely effect them; and 4) the studies have been performed on mild asthmatics and bronchitics and there is a significant likelihood that more severe asthmatics and bronchitics would suffer a greater magnitude of effects.

There are studies reported in the scientific literature on exposure of normal subjects to low concentrations of NO_2, but none of which disclose any clear effects on pulmonary function. Conclusions from some of these studies are reported below:

- Beil and Ulmer (1976) reported no increase in airway resistance in 8 healthy adults exposed to 1 ppm NO_2 for 2 hours.

- Hackney et al (1978) reported either no statistically significant or relatively small changes in 16 healthy adults exposed to 1 ppm NO_2 for 2 hours for 2 consecutive days; with

the exception of measurement of forced vital capacity which showed a small decrease (1.5%, p less than 0.05) after the second day of exposure. The authors suggested that this change might have been a chance observation in view of the large number of different pulmonary function parameters measured.

- Folinsbee et al (1978) concluded that there were no consistent changes in pulmonary or cardiovascular function in 15 healthy male nonsmokers exposed to 0.62 ppm NO_2 for 2 hours.

On the whole, the available studies which will be reviewed subsequently, suggest that people with compromised respiratory systems, such as asthmatics and chronic bronchitics, may be sensitive to NO_2 inhalation at low concentrations. The interpretation of these studies has been conflicting, however, and can be accounted for, in part, by the different types of pulmonary tests and methods of statistical analyses used. Furthermore, the design of the majority of these studies has not been adequate to compare the sensitivity of compromised individuals and normal individuals.

Following exposure of asthmatic subjects for 1 hour to 0.1 ppm NO_2 in an exposure chamber, Orehek et al (1976) reported statistically significant increases for 13/20 subjects (student's paired t-test) in specific airway resistance and in bronchial response to inhalation of a carbachol aerosol (reviewed in CPSC, 1983). The authors suggested that asthmatic subjects might be "at risk" for adverse effects at low NO_2

concentrations. Dawson and Schenker (1979) reanalyzed Orehek's data (based on logarithm of dose) and concluded that a statistically significant bronchoconstrictive effect was evident for the complete group of 20 subjects (p less than 0.002).

Hazucha et al (1983) conducted a controlled clinical exposure of an asthmatic population to 0.1 ppm NO_2 (in an exposure chamber) using a bronchial provocation technique and routine pulmonary function tests in order to assess the findings of Orehek et al. Fifteen normal and 15 asthmatics with mild disease (all nonsmokers) were exposed to air and to NO_2 in a randomized double-blind crossover design. Exposure to either air or NO_2 was bracketed by bronchial inhalation challenge with aerosolized metacholine chloride. Specific airway resistance (SRaw) and forced random noise impedence spectrum were obtained after each metacholine dose. These tests are thought to be sensitive in detecting early airway disease in individuals with normal spirometry. Data from the four initial data sets (air -NO_2 and NO_2-air sequences for both asthmatics and normal groups) were pooled to create only two data bases: normals and asthmatics. A total of seven variables per group were evaluated; for each variable, five hypotheses were tested using multivariate analysis of variance for a repeated measures type of experiment. The authors, however, declared a priori only two hypothesis for the two variables, SRaw and PD_{100} (dose of drug that caused a doubling of the baseline value of the SRaw) as of primary importance. The first hypothesis tested the equality of the preexposure values and the second tested whether the change in the means from preexposure to postexposure was the same for both the air and NO_2 exposures. Thus,

they applied a Bonferroni correction factor of 4 (2 hypotheses x 2 variables) to a P value of 0.05 to obtain a P level of 0.0125 (0.05 divided by 4), which was used to reject the null hypotheses.

Following exposure to NO_2, there was a slight but not significant increase in SRaw in both normals (5% increase, p=0.225) and asthmatics (8% increase, p=0.423). The overall resistive properties of the respiratory system as measured by forced random noise impedence was not affected by NO_2 inhalation either. These results suggested to the authors that there was no selective increase in airway resistance following NO_2 exposure. Evaluation of bronchial responsiveness to metacholine showed that the asthmatic group was, as expected more sensitive (i.e., a lower cumulative dose was required to cause doubling of the baseline value of the SRaw) and somewhat more reactive (slope of the dose-response curve). NO_2, however, did not enhance bronchial responsiveness in either group.

With regard to Orehek's data, Hazucha et al suggested that if Orehek had used statistical tests such as analysis of variance and the error rate had been adjusted by the Bonferroni method, null hypotheses would not have been rejected and there would have been no significant difference in sensitivity and reactivity of responders and nonrespondeners to NO_2 in the Orehek et al study.

Ahmed et al (1982) have reported in an abstract still another similar study. In twenty normal and twenty asthmatic subjects specific airway conductance (SGaw) (reciprocal of airway resistance, expressed

per liter of lung volume), forced expiratory volume in 1 sec (FEV_1), and the density dependence of the maximum expiratory flow volume curves (volume of isoflow, VisoV)* were measured before and after a 1 hour exposure to air or 0.1 ppm NO_2, in a single blind cross-over design. The latter test (VisoV) is a sensitive test which provides inferential information about the mechanical properties of the airways; the volume of isoflow is increased in patients with respiratory disorders with an obstructive component. Bronchial responsiveness was determined by obtaining a dose response curve to inhaled carbachol and determining the PD_{35} (cumulative provocation dose of carbachol in "breath units" causing a 35% decrease in SGaw). NO_2 was reported to have no significant effect on baseline SGaw, FEV_1, or VisoV in either subject group. Subjects were classified as responders if their PD_{35} decreased after NO_2 exposure. In 10/20 normals (responders), mean PD_{35} decreased from 26 breath units after exposure to air to 11.6 breath units after exposure to NO_2 (p less than 0.005); while in the remaining 10 normals (nonresponders i.e., individuals without a decrease in PD_{35} after NO_2 exposure), mean PD_{35} increased from 15.4 breath units after air to 27.6 breath units after NO_2 (p less than 0.005). In 13/20 asthmatics (responders), PD_{35} decreased from 8 breath units after air to 2.3 breath units after NO_2 (p = 0.05); while in the remaining 7 asthmatics (nonresponders), PD_{35} changed from 2.4 breath units after air to 3.4 breath units after NO_2 (p = not significant). The authors concluded that 1 hour exposure to 0.1 ppm NO_2 has a variable effect on bronchial reactivity in both normals and asthmatics.

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*VisoV is the lung volume at which maximum expiratory flow becomes independent of gas density.

Following exposure of 20 subjects with asthma and bronchitis and 10 normal healthy subjects to 0.5 ppm NO_2 for 2 hours in a chamber (with light to moderate exercise for 15 minutes during the first hour), Kerr et al (1979) reported a statistically significant decrease in quasistatic compliance for the 10 normal subjects (p less than 0.025) and the 20 asthmatic and chronic bronchitics (analyzed together, p less than 0.05), and a statistically significant increase in the static lung volumes, functional residual capacity, residual volume and total lung capacity for the 20 test subjects when analyzed together.* No significant changes were reported when the 2 test subject groups were analyzed separately. On the whole, the combined results from the test subjects suggest some degree of change in elastic properties, as would be anticipated in persons with chronic obstructive lung disorders. However, the changes did not correlate with symptoms (7/13 asthmatics experienced chest tightness, dyspnea, burning of the eyes, and headache with exercise and exposure to NO_2, 1/7 chronic bronchitics and 1/10 normals experienced slight rhinorrhea). The authors concluded that exposure to 0.5 ppm NO_2 for 2 hours does not produce a significant decrement in pulmonary function in healthy, asthmatic, or bronchitic individuals. Based on results from 6 hour ozone studies by these investigators (Kerr et al, 1975) in which statistically significant pulmonary decrements were noted after 4 or 6 hours of exposure, the authors propose that exposure to 0.5 ppm NO_2 for 6 hours may also result in significant pulmonary decrements.

Kulle (1982) reanalyzed the data of Kerr et al (1979). The previous study, in which Kulle was a contributor, considered the

statistical significance of the day 2 - day 1 difference for the pre-exposure (0 hour) and the 2 hour exposure pulmonary function tests separately (See footnote). Whereas, Kulle analyzed the day 2 - day 1 difference based on each day's changes between the pre-exposure (0 hour) and the 2 hour levels. This analysis revealed a significant decrease in quasistatic compliance for the combined test subjects and the normal healthy subjects, as well as increased phase IV, "closing volume"; a test believed to be sensitive in screening for small airway disease. In practice it is usual to measure the closing capacity (sum of the closing volume and the residual volume); no significant differences were found in either the controls or the test subjects (subjects analyzed as a single group) when this was done. The author believed that the limited pulmonary function changes were relatively insignificant, but again speculated that slightly longer exposure periods, 4-6 hours, could result in significant changes in pulmonary function with possible correlation of symptoms.

To study the respiratory effects of NO_2, 31 asthmatic volunteers were exposed to clean air (control) or to 0.2 ppm NO_2 for 2 hours in a chamber, with light intermittent exercise (Kleinman et al, 1983). Bronchial responses to methachloline chloride challenge and direct exposure-related changes in pulmonary mechanics and symptoms were also

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*A two day study procedure was employed: on the first day (control day) subjects breathed filtered clean air for 2 hours, on the second day they breathed 0.5 ppm NO_2 for 2 hours. Testing was done at the 0 hour and 2 hour points each day. Data were compared between control (day 1) 0 hour and exposure (day 2 0 hour and control (day 1) 2 hours and exposure (day 2) 2 hours and the levels of significance determined by t test of 25 paired observations.

determined. No significant direct effect of NO_2 exposure on forced expiratory volume or total respiratory resistance was observed. Significantly fewer symptoms were reported during NO_2 exposure than during clean air exposure (p less than 0.05); and were explained by the author as a chance occurrence. Data on responses to methacholine suggest a slight overall excess of reactivity after NO_2 exposure as compared to clean air exposure, but this was not firmly supported by statistics.

The authors speculated that statistical analysis of the results of pulmonary tests are complicated by the fact that responses vary greatly among individuals and within individuals over time, thus producing data distributions that conform poorly to assumptions inherent in the analyses. Futhermore, they believe that the inherent biological variability of asthmatics will severely limit the degree of reproducibility and that caution must be exercised in generalizing results from small groups of experimental subjects to the large and diverse group of asthmatics. Therefore, although Kleinman et al, could not conclude unequivocally that NO_2 exposure increased bronchial reactivity in asthmatics, they believed that there was some tendency in that direction.

A study by Bauer et al (1984), presented at the 1984 Annual Meeting of the American Thoracic Society, indicated that low level NO_2 inhalation (0.3 ppm NO_2, 30 minutes) potentiated exercise-induced bronchospasm and airway hyperreactivity after cold air provocation in 10 adult asthmatic subjects which were significantly greater than those

resulting from exposure to clean air with cold air provocation. Interpretation of this study is confounded by the fact that cold dry air is known to cause irritation in some people and the clean air and NO_2 exposure were administered via a mouthpiece, which does not duplicate normal breathing conditions since more of the gas is likely to reach the lungs.

To determine if in fact the route of inhalation influences airway responses to NO_2, 6 of the same 10 adult asthmatics were exposed by double-blind randomization to 0.3 ppm NO_2 while in an environmental chamber, for a 4 hour period on two (2) different days separated by at least 48 hours (Bauer et al, 1985, abstract). All subjects exercised for 10 minutes, after one-half hour, 2 hours, and 3½ hours of exposure. After 30 minutes of exposure, NO_2 inhalation plus exercise compared to control (air) inhalation plus exercise did not produce a significant reduction in forced expiratory volume at 1 second (FEV_1) or specific airway conductance (SGaw). (These results contrast with the earlier study (Bauer et al, 1984) with the same subjects where exercise and 0.3 ppm NO_2 inhalation via a mouthpiece for only 30 minutes resulted in significant reductions in FEV_1 (p less than 0.01). However, after 2 hours of NO_2 exposure and repeat exercise, there were significantly greater reductions in FEV_1 (p less than 0.05) and SGaw (p less than 0.05) when compared to control exposure. These studies suggest that both oral mouthpiece and oral-nasal (chamber) inhalation of 0.3 ppm NO_2 may potentiate bronchospasm in asthmatic subjects, but that oral-nasal inhalation may delay the onset of bronchospasm, possibly due to scrubbing of NO_2 within the upper airways during unencumbered breathing.

Researchers at EPA's Clinical Studies Branch have replicated the Bauer et al (1984) study with natural breathing in a chamber and without cold air using 12 mild asthmatics exposed for 110 minutes to 0.3 ppm NO_2 and to clean air on separate days (Roger et al, 1985, abstract). Forced vital capacity (FVC) and FEV_1 were measured before exposure and after each moderate exercise period (3 cycles of 20 minutes rest followed by 10 minute exercise). After the first exercise period, NO_2 compared to air produced significantly greater reductions in FEV_1 (p less than 0.02) and possibly FVC (p less than 0.1). The reductions in FEV_1 were similar to those reported by Bauer et al (1984). FEV_1 returned to baseline values upon repeated exercise. The authors conclude that inhalation of 0.3 ppm NO_2 causes decrements in pulmonary function in exercising naturally breathing asthmatics. They noted also, a wide range of subjects' responses to NO_2 exposure compared to changes seen following clean air exposure.

To determine if low level NO_2 exposure induced alterations in lung function in normal subjects, 20 nonsmoking volunteers were exposed by double blind randomization to either air or 0.30 ppm NO_2 for a 4 hour period in a chamber on 2 different days separated by at least 48 hours (Smeglin et al, 1985, abstract). Subjects exercised for 10 minutes after 1 hour, 2 hours, and 3 hours of exposure. No significant changes in flow rate or airway resistance occurred during or after NO_2 exposure; similarly no significant changes in diffusing capacity occurred after NO_2 inhalation. Similarly, no significant potentiation of the carbachol bronchoconstrictor effect was observed either immediately after NO_2 inhalation or following a latent period. These results suggested to the

authors that exercising normal subjects are less responsive to NO_2 than exercising asthmatics and that normals do not demonstrate enhanced airway hyperreactivity as reported for asthmatics (Bauer et al, 1984). It should be noted however, the design of these studies is dissimilar and, therefore, the studies are not strictly comparable.

C. EPIDEMIOLOGICAL STUDIES

Epidemiological studies (reviewed in CPSC, 1983 and 1984) have attempted to establish a cause and effect relationship between chronic low level exposure to NO_2 and changes in lung function or respiratory illness. Interpretation of the effects of individual pollutants (such as NO_2) in these studies has been complicated by the complex mixtures of pollutants that were always present in the air. They have also been hampered by methodological problems including inconsistencies in measurement methodology and in the relationship between measured pollutant levels and the levels actually experienced by the cohort. Therefore, although these studies are inadequate to prove a cause and effect relationship between levels of exposure to a single pollutant, such as NO_2, and health effects they do, in a qualitative sense indicate a trend in increased morbidity, particularly in children, from low level exposure to a mixture of pollutants. The fact that NO_2 is a component of the released gases, combined with animal and controlled human toxicology data, raises the possibility that NO_2 may be a cause of or a contributor to the effects seen in children.

Because there could be injury to the lung without initially eliciting measurable changes in pulmonary function (as suggested by the findings

of Menzel et al, 1980 and reviewed previously in this report), several Japanese investigators have measured urinary hydroxyproline excretion (expressed as a ratio of hydroxyproline to creatinine) as an indicator of the effects of NO_2 which might occur prior to the manifestation of symptoms (Matsuki et al, 1981, 1983?, and Yanagisawa et al, 1983). This indicator was selected based on the following studies:

- In an epidemiological study of 70 workers exposed to 0.4 - 2.7 ppm NO_2 for 6-8 hours/day for 4-6 years, there were no reported clinical symptoms or changes in pulmonary function, but, compared to control workers, a significant increase in urinary hydroxyproline was reported, suggesting increased lung metabolism (including increased degradation) (Kosmider et al, 1972 reviewed in CPSC, 1983).

- Guinea pigs exposed to 1.1 ppm NO_2 for 6 months developed emphysema like lesions and had increased urinary excretion of hydroxyproline compared to control animals (Drozdz et al, 1977, reviewed in CPSC, 1983).

In a study which did not measure personal NO_2 exposure, increased hydroxyproline:creatinine (OHPRO:CR) ratios directly increased in healthy school children (total cohort = 4,964; aged 6-11; 1977-1980) with increasing number of cigarettes smoked in the household (passive smoking) and with shortening of the distance between their homes and the main traffic roads. The ratios were also increased in children living in homes with unvented stoves in their living rooms. (Matsuki et al, 1981; English abstract). Healthy adults also had increased OHPRO:CR

ratios which correlated directly with the number of cigarettes smoked (active smoking). The authors supposed that oxidants such as NO_2, which are contained in cigarette smoke and air pollution may contribute to these effects. Other components of cigarette smoke and air pollution also likely contribute to the reported health effects. In a subsequent study of primary school children (n=820 with complete data set) and their mothers (n=546 with complete data set) monitored by personal 24 hour NO_2 filter badges* during April, 1980 - March, 1981, a correlation between OHPRO:CR ratio and passive smoking and OHPRO:CR ratio and personal 24 hour NO_2 exposure (average 35.7 ppb NO_2±21.6 ppb,S.D.) was reported (Matsuki et al, 1983?, and Yanagisawa et al, 1983). These studies suggest that urinary hydroxyproline may be a sensitive biochemical indicator of NO_2 exposure, in the absence of detectable pulmonary function changes, but this remains to be fully confirmed.

III. SUMMARY AND DISCUSSION

The results of recent animal studies provide further substantiation of the pulmonary and nonpulmonary effects of low level NO_2 exposure. Results from a biochemical study (Sagai et al, 1984) confirm earlier observations that the formation of lipid peroxides (an integral part of cell damage) in rats following acute and subacute exposure to the oxidant, NO_2, and the activities of the antioxidative protective enzymes, vary inversely. More importantly, results from this study demonstrate a decline in the ability of the antioxidative enzyme system

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*The filter badges, developed by Yanagisawa et al, 1982, can determine NO_2 concentrations in the air from the amount of NO_2 absorbed to the cellulose fiber filter containing a triethanolamine solution. The sensitivity of the filter is 66 ppb/hr and the accuracy is ±20% under a wide range of conditions.

to protect the lung from injury following chronic exposure at concentrations as low as 0.4 ppm NO_2. Additional studies by Yoshikawa et al (1982b), suggest that rats exposed to 0.4 ppm for 9 or 18 months are chronically hypoxic, which may, in turn, stimulate increased lipid peroxidation.

Futhermore, data from a morphological study (Gordon et al, 1985) suggest that some morphological alterations induced by NO_2, or other pollutants causing oxidant injury, can be prevented by dietary supplements of taurine, a non alpha amino acid.

A variety of functional changes have been reported in response to NO_2 exposure. Lam et al (1983) reported that mild injury in newborn hamsters induced by 30 ppm NO_2 for 7 days, results in physiologic and morphometric changes in the mature animal similar to those seen in the early stages of emphysema. This finding however has not been confirmed at low concentrations of NO_2.

Although no new chronic low level immunological studies have been published, a recent study indicates that while exposure to 40 ppm NO_2 may act to initially stimulate macrophage-mediated immune defenses, multiple NO_2 exposures (to 40 ppm NO_2) may induce an effect whereby the immune defenses are no longer enhanced above control (Sone et al, 1983). This, however, was not tested following low level NO_2 exposure.

On the whole, there is evidence from animal studies that continuous and intermittent exposure to low concentrations of NO_2 alters the host

respiratory immune defense mechanism, potentially making the animal more susceptible to respiratory infections. Thus, foreign organisms that might normally be kept under control by bactericidal, viral, and clearance mechanisms may, under the condition of an impaired immune defense system, cause pulmonary disorders.

Pharmacological studies by Menzel et al (1980) report an effect on prostaglandin metabolism at concentrations of NO_2 less than 1 ppm; alterations which could potentially lead to a mismatch between gas ventilation and local blood perfusion within the lung. In the clinical setting, this mismatch is recognized as an early indicator of pulmonary disease. Furthermore, this study indicates a delay (up to 18 hours) between exposure and effect, in rats exposed to 0.2, 2.0 and 19 ppm NO_2 for a single 3 hour period; results which may explain the lack of measurable pulmonary function in the acute short-term controlled human studies (section 8).

Results from studies previously reviewed by CPSC indicated that intermittent NO_2 exposure at 0.4-0.8 ppm facilitated metastases of cancer in exposed animals probably due to a change in some immunological capability (Richter, 1981 and Richters and Kuratis, 1981). Results from a recently published study (Richters and Richters, 1983) suggest that extended periods of clean air between ambient NO_2 exposure may reduce the severity of the NO_2 effects in the less sensitive animals. Lastly, a study by Mochitate et al (1984) reports that the microsomal components of rats liver are decreased following 7 days exposure to concentrations of NO_2 less than 1 ppm suggesting an alteration of the host's ability to metabolize and/or detoxify xenobiotic compounds.

Several controlled human studies suggest that short term peak exposures to low concentration of NO_2 may cause adverse respiratory effects, particularly in sensitive populations such as asthmatics. Although study design, measurement methodology statistical analysis, demonstration of effects, and interpretation of which test parameter demonstrates adversity are not presently considered definitive; available evidence precludes the assumption that there is no evidence of harm. Results of most human exposure studies suggest that NO_2 at concentrations common in polluted ambient and indoor air has little or no respiratory effect on normal healthy adults. The literature relevant to sensitive populations is however, inconsistent and inconclusive. These studies have focused largely on symptomatology, measurement of lung function and mechanics, or increased airway responsiveness to inhaled agents; with exercising subjects being used in some of the studies . One study (Kerr et al, 1979 and Kulle, 1982) reported chest discomfort, dyspnea, headache, and nasal discharge in asthmatics and bronchitics, following a 2 hour exposure to 0.5 ppm NO_2 with a 15 minute period of light to medium exercise; but the study did not provide clear evidence of pulmonary function change at that NO_2 concentration. Other studies have reported that exposure to low level NO_2 (0.1 ppm for 1 hour) increased responsiveness of the airways in asthmatics following various inhalation of agents (Orehek et al, 1976), sometimes occurring even in the absence of statistically significant changes in pulmonary mechanics (Ahmed et al, 1982). On the other hand, Hazucha et al (1983) performed exposure studies also at 0.1 ppm NO_2, and observed no statistically significant differences in response by asthmatics or normals to NO_2 vs. clean air. Additional studies, however, are not

inconsistent with a trend in increased bronchial responsiveness in a susceptible population (asthmatics) at slightly higher concentrations of NO_2 (Kleinman et al, 1983; Bauer et al, 1984), but not in normals (Smeglin et al, 1985). An abstract presented at the 1985 annual meeting of the American Thoracic Society indicates time differences in response to NO_2 exposure in asthmatics depending upon the mode of inhalation, oral (via mouthpiece) or oral-nasal (via chamber); with a delay in onset of bronchospasm following oral-nasal inhalation.

Although analysis of the available data base are not inconsistent with a trend in increased susceptibility in sensitive populations following low level exposure to NO_2, the data have been inadequate. Further investigation is needed to determine which respiratory measurement represents the most sensitive means of detecting adverse health effects, what constitutes "adversity", whether the effects reported in mild asthmatics and bronchitics can be used to draw conclusions about the larger population with chronic lung disorders, whether the reported effects are reversible over time, and whether the reported effects are similar during acute, chronic, or intermittent exposures.

Epidemiological studies previously reviewed by CPSC (1983) have, in a qualitative sense, suggested an association in children between increased respiratory illness and symptoms and low level exposure to a mixture of pollutants (which includes NO_2). Although no conclusive determination could be made, the reviewed studies do not rule out that NO_2 might be a cause of or a contributor to respiratory illness. Several recent studies measuring urinary hydroxproline excretion as an

indicator of the effects of NO_2 that might occur prior to the manifestation of symptoms, report correlations between increased urinary hydroxyproline excretion and passive smoking and also to personal exposure to NO_2 at levels less than 1 ppm (Matsuki et al, 1983? and Yanagisawa et al, 1983). These studies suggest that measurement of urinary hydroxyproline excretion may be useful for assessing effects of NO_2 exposure.

Because of the uncertainty in the available data for NO_2 and other pollutants, the Health Effects Institute (HEI) a nonprofit organization, was created in 1980 to conduct or support the conduct of research relating to the health effects of emission products. Results of these ongoing studies sponsored by HEI, as well as of other independent studies will hopefully better define the relationship between NO_2 exposure and health effects, and may lead to the development of a threshold level for effects from low level NO_2 exposure.

III. Conclusions

Although there has been human data (pulmonary function and response and nonpulmonary metabolic processes) which qualitatively demonstrates health effects in some sensitive populations following exposure to NO_2 at concentrations ranging from 0.1 ppm to 1 ppm, no unequivocal epidemiological data exist to substantiate such effects at these concentrations. There is, however, an increasing animal data base which demonstrates a variety of potentially serious effects resulting from chronic exposure to such concentrations of NO_2. Because similarities

exist between the basic mechanisms of action of the human and the animal pulmonary defense systems, the responses reported in animals support the need for further investigation to determine the relationship between NO_2 and susceptibility to infection, to define the effects of NO_2 in the developing lung, to study the relationship between NO_2 exposure and the development of chronic lung disease, to examine the effects of NO_2 exposure in potentially susceptible populations, and to define if there are levels of exposure to NO_2 which are not associated with adverse health effects such as impaired lung function and metabolic processes.

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HEALTH EFFECTS OF NITROGEN DIOXIDE (1983

Kerosene Heater Briefing

INTRODUCTION

In August 1982, the Directorate for Health Sciences prepared a preliminary assessment, based on four reviews (EPA, 1980; NAS, 1977; NIOSH, 1976; and WHO, 1977), on the health effects of nitrogen dioxide. Based on available data, exposure levels were recommended in three of the reviews:

> NIOSH 1 ppm (ceiling for 10 hour work day, 40 hour work week). Rationale- below lowest effect level "to prevent acute irritant effect in lungs of workers; prevent repeated episodes of irritancy to lessen risk of chronic lung disease;"
>
> WHO 0.10-0.17 ppm (continuous exposure). Rationale- lowest level of adverse effects in animals and additional safety factor; and
>
> EPA 0.05 ppm (annual average). Rationale- provides a reasonable level of protection against short term peaks which minimizes the potential health effects of NO_2 exposure.

The purpose of the current health assessment of nitrogen dioxide is to provide an in-depth analysis of:

- analytical methodologies and their relationship to the interpretation of reported data;
- data on how NO_2 adversely affects health;
- data published since 1980 and the reviews by EPA (1980), NAS (1977), NIOSH (1976) and WHO (1977); and
- data which associate low level (1 ppm or less) exposure with adverse health effects.

SOURCES

The natural background values for nitrogen dioxide (NO_2) over land range from 0.0002-0.005 ppm. Urban areas have typical annual mean concentrations of 0.01-0.05 ppm (WHO, 1977). EPA monitoring stations in Los Angeles and Chicago during the early 1970's reported average annual outdoor values of 0.26 ppm and 0.22 ppm, respectively.

Oxides of nitrogen may be generated inside the home by fuel-burning cooking and heating appliances. Gas stoves, kerosene heaters, and unvented gas space heaters may emit NO_2 and nitric oxide (NO), along with particulates and/or other gases. Investigators in the United States and England have measured both peak and average values of NO_2 in homes with gas stoves during the 1970's.. Two-week average levels in these homes ranged from 0.06-0.08 ppm and two-hour average levels measured in the kitchen were 0.15 ppm. During the preparation of meals, mean levels of 0.25-0.6 ppm have been measured in the vicinity of the stove, with peaks of 1.0 ppm (EPA, 1980).

CHEMICAL AND PHYSICAL PROPERTIES*

Nitrogen dioxide is a reddish-orange-brown gas with a characteristic pungent odor. It is corrosive and highly oxidizing. Nitrogen dioxide is formed from the oxidation of nitrogen oxide (NO, nitric oxide), a principal by-product of combustion processes, arising from the oxidation of molecular nitrogen in the combustion air and of organically bound nitrogen present in certain fuels such as coal and heavy oil. The oxidation of nitrogen in combustion air occurs primarily through a set of reactions:

$$\text{Nitrogen } (N_2) + \text{molecular oxygen } (O) \longrightarrow NO + N^{\cdot}$$

$$N^{\cdot} + \text{oxygen } (O_2) \longrightarrow NO + O^{\cdot}$$

$$2\,NO + O_2 \longrightarrow 2\,NO_2$$

The above reactions are mainly responsible for the NO_2 (5-10% of the total oxides of nitrogen) present in combustion exhaust gases. Most outdoor NO_2 in populated areas arises from the conversion of NO to NO_2 by a variety of chemical processes in the atmosphere.

Nitric oxide (NO) is an odorless and colorless gas. The amount of NO formed from the oxidation of molecular nitrogen is dependent upon such parameters as peak flame temperature, quantity of combustion air, and gas residence time in the combustion chamber. The amount of NO arising from oxidation of fuel-bound nitrogen does not seem to depend significantly on either the type of nitrogen compound involved or the flame temperature, but instead upon the specific air-to-fuel ratio at various stages in combustion.

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* The discussion of the chemical and physical properties is taken from the EPA Air Quality Criteria for Oxides of Nitrogen (draft 1980).

ANALYTICAL METHODS

There are a number of different methods which are used to determine the concentration of NO_2 in ambient air. An analysis of the methodologies was undertaken in order to determine if the stated concentrations of NO_2 in various studies were directly comparable. A review of the literature shows that several methods offer reliable results. Each method has its limitations. If these limitations are recognized by the investigators the values obtained are comparable. A detailed analysis of the methods used to determine NO_2 is given in Appendix A.

HEALTH EFFECTS

Overview

The purpose of this section is to review the studies which are most relevant to the evaluation of the health hazards resulting from exposure to nitrogen dioxide. The primary target organ for nitrogen dioxide exposure is the lung. Data from animal studies indicate that a single short-term exposure to nitrogen dioxide results in a temporal sequence of events which includes an increased susceptibility to inhaled microorganisms (pneumonia), which peaks at about 10 hours post exposure; death of lung cells, which peaks at 24 hours post exposure; and replacement of dead and injured lung cells, which peaks at 48 hours post exposure (Menzel, 1980). The temporal sequence of events following continuous exposure to nitrogen dioxide indicates that cell replacement

peaks at 48 hours (as in the single short-term exposure); the susceptibility to infection continues to increase nearly linearly over time; and evidence of changes in pulmonary function and the incidence of emphysema-like pathology begin to appear after one to two months exposure (Menzel, 1980).

Toxicological data relating exposure to effects on lung tissue can be placed in three categories: morphological, biochemical, and functional. Biochemical effects are most easily demonstrated because of the sensitivity of the methods of detection, while functional changes are usually not observed until there has been significant alteration in lung tissue.

ANIMAL STUDIES

Morphological

The distribution of cellular injury from NO_2 extends throughout the conducting airways and the proximal part of the alveoli, generally not affecting the alveolar walls in the distal alveoli (Mustafa and Tierney, 1978). In addition, Mustafa and Tierney (1978) have suggested that because NO_2 is soluble in tissue fluids it may also pass into the interstitium and endothelium to produce deeper lung tissue injury. Pathologic changes have been observed in the small airways and alveolar regions of the lung following exposure to NO_2.

The small airways or bronchiolar region of the normal lung are lined with a single layer of ciliated cells plus some protruding nonciliated cells (Clara cells). In the alveolar region 97% of the

surface area is covered by large flat squamous cells (Type I cells) and the remaining 3% of the surface is covered by granular pneumocytes (Type II cells),* see Figure I.

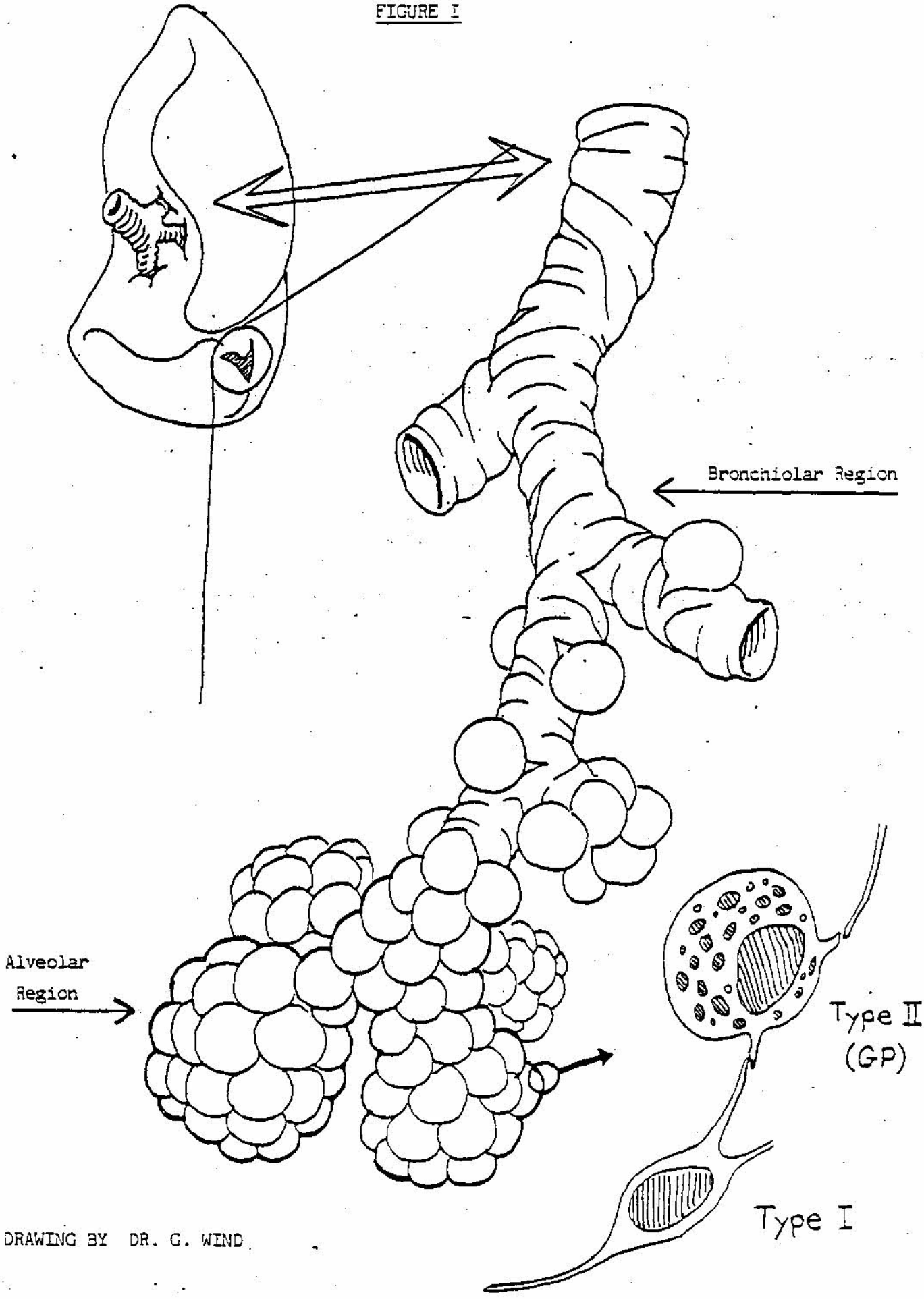
Evans and Freeman (1980) have recently summarized their studies on the morphological effects of NO_2 on the rat lung. When rats were exposed continuously to NO_2 the cells lining the airways and alveoli were damaged. After four hours a loss of cilia began in the bronchiolar regions and by eight hours the sloughing of dying and necrotic cells was observed. In the alveolar region, morphological changes were also observed by the fourth hour of exposure. Type I cells near the opening of the terminal bronchioles exhibited swelling, and occasionally there were areas of lost Type I cells leaving the basement membrane** exposed. These changes increase in intensity after a day of continuous exposure with hyperplasia and loss of cilia seen in the bronchiolar regions, and large areas in the alveolar region devoid of Type I cells. Type II cells, alveolar macrophages, and endothelial cells appeared to be relatively resistant to NO_2.

Evans et al (1971;1972;1975;1978) have exposed young rats to 2 or 17 ppm NO_2 for various periods of time. At the end of the exposure period the rats were injected with radioactive thymidine and then sacrificed one hour later. Replacement of bronchiolar and alveolar epithelium cells damaged by NO_2 was determined by measuring the

* The Type II cells contain granules of phospholipids (lamellar bodies) which are secreted on to the alveolar surface. The phospholipid is very surface active (reduces the surface tension of the alveoli to almost zero), and is essential for the stability of the lung.

** Basement membrane is the membrane to which cells are attached.

FIGURE I



DRAWING BY DR. G. WIND

incorporation of radioactive thymidine* into the new cells. The results of these experiments are shown in Figure II. Radioactive thymidine incorporation is first detected in the nonciliated cells of the terminal bronchiole region of the lung during continuous exposure to NO_2 (Evans et al, 1972). Peak activity was observed at 24 hours (Figure IIA) in nonciliated cells and then declined. Twenty-four hours after exposure to 2 or 17 ppm NO_2 the Type II cells of the rat lung incorporate a detectable amount (dose related) of thymidine (Fig IIB) as compared to controls. Peak incorporation of thymidine was observed 48 hours after the onset of exposure to NO_2, uptake then declines to the control level around the fifth day. This decline has been interpreted by the authors to indicate that the new cells are relatively resistant to continuous exposure to the same concentration of NO_2. Using incorporation of radioactive thymidine as a marker for cell renewal Evans et al (1978) observed that the Clara cells in the bronchiolar region divide into a Clara cell and a ciliated cell, and in the alveolar region the Type II cell divides into a Type I cell and a Type II cell.

Evans and Freeman (1980) have concluded from their studies:

- the cells most vulnerable to NO_2 are the ciliated bronchiolar and Type I alveolar cells (both cell types have much larger surface area exposed to NO_2 than the more resistant Type II and Clara cells);
- the epithelium can repair itself in the presence of sublethal levels of NO_2; and

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* In these experiments incorporation of thymidine into nuclear DNA is intrepreted as a marker for the synthesis of new DNA and thus cell division.

- the new cells are relatively resistant to continuous exposure to the same concentration of NO_2.

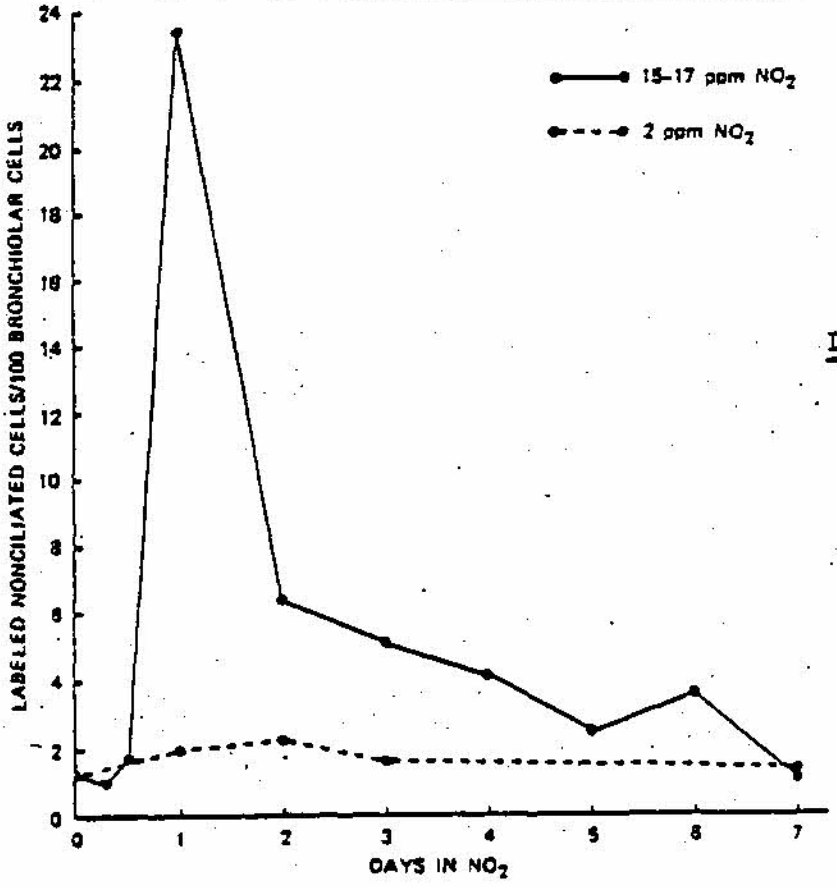
Discussion

The investigations by Evans et al (1971;1972;1975;1976;1978;1980) clearly show that lung bronchiolar ciliated cells and alveolar Type I cells are damaged by nitrogen dioxide. This effect stimulates cell division in the lung. The amount of cellular turnover appears to be dependent on the concentration of NO_2. The rate of cellular turnover, however, appears to be independent of the concentration of NO_2 to which the animal was exposed. The observed Type II cell proliferation is consistent with the biochemical observation (see next section) showing increased amounts of surfactant in lungs of animals exposed to nitrogen dioxide.

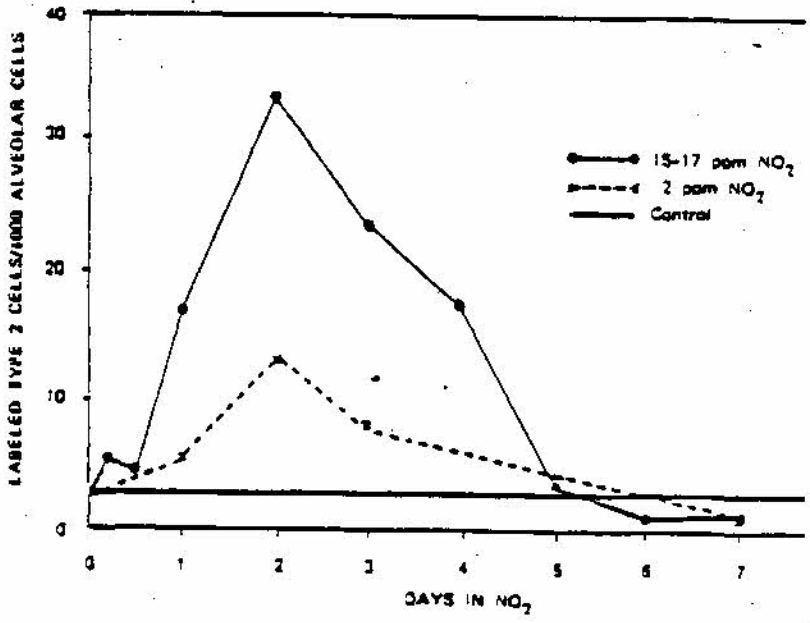
In the research done to date, animals have been exposed to 2 ppm or more NO_2 in order to demonstrate the effect on cell turnover. An extrapolation of the available data suggests that it would be difficult to demonstrate observable/detectable cellular damage at less than 2 ppm NO_2 in the short term. The methods used in these studies assume that damage is universal, thus no provision is made to survey the tissue in a manner which would detect focal damage. Exposure to less than 2 ppm nitrogen dioxide could cause local/focal damage which would not be detected by the method.

FIGURE II CELLULAR RENEWAL DURING CONTINUOUS EXPOSURE TO NO_2

as demonstrated by tritiated thymidine incorporation/labeling



IIA: Nonciliated (Clara) cells



IIB: Type II cells

Evans and Freeman (1980)

Biochemical

Nitrogen dioxide is probably toxic because it acts as an oxidant. In order to assess the effect of NO_2 interaction with tissue, biochemical studies have focused on changes in glucose metabolism, lipid peroxidation and alteration in sulfhydryl (glutathione) metabolism as a measure of cellular antioxidant activity.

Ospital et al (1981) have reported an increase in glutathione metabolism and an increase in the activity of some enzymes involved in glucose metabolism in lung tissue homogenates prepared from rats exposed continuously to 5 or 15 ppm NO_2 for 1-7 days. After one day of exposure to 15 ppm NO_2 the non-protein sulfhydryl compounds (a major pool of reducing equivalents in cells) increased 30% ($P<0.02$) above the control value and after 4 days it increased 140% ($P<0.01$) above the control. In addition, the total activity of the enzymes glutathione reductase, glutathione disulfide reductase and glutathione peroxidase significantly increased in lung tissue. Lung tissue from rats exposed for 7 days to 5 ppm NO_2 underwent significant increases, about 30% ($P < 0.05$), in glutathione reductase and disulfide reductase activity as well as nonprotein sulfhydryl compounds. There were also significant increases in the enzymes glucose-6-phosphate dehydrogenase, 6-phosphogluconate dehydrogenase and NADP-specific isocitrate dehydrogenase in rat lung tissue exposed to 15 ppm NO_2. The increase in activity of all three enzymes peaked (75-175%, $P < 0.01$) and remained elevated after 4 days exposure. After 7 days exposure to 5 ppm of NO_2 only glucose-6-phosphate dehydrogenase activity was significantly increased (33.5%, $P < 0.02$). Ospital et al (1981) suggested that the increase in glucose metabolism enabled lung cells to generate increased

amounts of NADPH (a reduced nicotinamide) which is used to maintain reduced glutathione. Similarly, the increase in glutathione reductase and peroxidase activities reduces toxic lipid peroxides. The authors suggest that the lack of dramatic changes following exposure to 5 ppm NO_2 might be due to less extensive and/or focal injury as well as the dilution of affected tissue with normal tissue during the homogenization process.

Sagai et al (1981) in a brief communication reported on the effect of NO_2 exposure, 10 ppm for up to 14 days, on rat lung tissue lipid peroxidation, glutathione reductase and glutathione peroxidase activity. They found that lipid peroxidation peaked between days 3-5 of the exposure period and then slowly declined to the control level by day 14. A detectable increase in glutathione reductase and peroxidase activities was seen around day 4, peaked by day 5 and remained elevated. The authors believe that the increase in lipid peroxidation is a measure of tissue damage; and the increase in glutathione reductase and peroxidase activities are a measure of an induced protective mechanism by the lung; thus the subsequent decline of lipid peroxidation is interpreted as a demonstration of the effectiveness of the protective mechanism.

Ichinose and Sagai (1982) have reported on the antioxidative protective systems in rat lung tissue which has been exposed to nitrogen dioxide. Lung tissue, from rats (6 per group) continuously exposed to 0, 0.4, 1.2 or 4 ppm NO_2 for 1-16 weeks was assayed for lipid peroxidation, glutathione reductase, glucose-6-phosphate dehydrogenase and superoxide dismutase. There was an increase in glucose-6-phosphate dehydrogenase activity which peaked, and remained elevated, after 4 weeks of exposure. The peak increase was 5% above control in lung tissue exposed to 0.4 ppm NO_2, 15% at 1.2 ppm and 46% at 4 ppm

(significantly different from control at 4 ppm). There was also an increase in glutathione reductase, which peaked at 1-2 weeks, in lung tissue from rats exposed to NO_2 (significantly different at 4 ppm NO_2). The peak activity of lipid peroxidation was reached after one week of exposure (significantly different from controls at 1.2 and 4 ppm NO_2) and then rapidly declined. After 8 weeks of exposure, lipid peroxidation again began to increase, and continued to do so until the end of the experiment at 16 weeks (significantly different from controls at 4 ppm NO_2). The authors interpreted the increase in superoxide dismutase activity as indirect evidence that peroxides are the source of lung tissue damage by nitrogen dioxide. They also suggested that the increase in glucose-6-phosphate dehydrogenase and glutathione reductase protect the cells from lipid peroxide damage through the formation of NADPH and reduced glutathione. The authors also attribute the increase in nonprotein sulfhydryls to the increase in glutathione-related enzyme activities. Ichinose and Sagai (1982) concluded that the results of their study show that the protective ability against oxidative stress decreases with long-term exposure to nitrogen dioxide and that lipid peroxidation increases inversely with the protective mechanism.

DeNicola et al (1981) have reported on significant changes in the amount of various enzymes as well as the cellular composition of pulmonary lavage fluid* in Syrian Hamsters (6 per group) exposed to 12, 17, or 22 ppm NO_2 from 48 hours. After 48 hours exposure to 12 ppm NO_2

* At the end of the exposure period the animal is sacrificed, and the lungs removed with the trachea attached. The lungs are then gently filled with a saline solution via a tube inserted into the trachea. The fluid (pulmonary lavage fluid) which is then removed by gentle suction contains unattached cells and the protein and lipids found in the terminal airways.

there was a slight (20%) but significant increase in acid phosphatase activity in lavage fluid. After 48 hours exposure to 17 ppm NO_2 there were significant increases in acid phosphatase (59%), glutathione reductase (38%), glutathione peroxidase (316%) and lactic acid dehydrogenase (273%) activity in lavage fluid. In addition, there were significant increases in sialic acid (870%) and protein (725%) content of the lavage fluid. Changes in enzymatic activity in lung tissue were much less dramatic. There were significant increases in the number of macrophages and neutrophils in pulmonary lavage fluid after 24 hours of exposure to 12 ppm NO_2. The number of cells found in lavage fluid increased with duration and level of exposure. The authors concluded that the increase in lactic acid dehydrogenase, glutathione reductase, and glutathione peroxidase activity reflects cell injury via the leakage of cytosolic enzymes into alveolar spaces (lavage fluid). The increase in lysosomal enzymes (acid and alkaline phosphatases) indicates an increase in the phagocytotic activity of macrophages and neutrophils, and the increase in sialic acid and protein, components of blood serum, are consistent with the observed edema.

The alveolar region of the lung is lined by a surface-active layer (surfactant**) which lowers and stabilizes surface forces during respiration (Clements, 1967). The essential component of the surfactant is dipalmitoyl lecithin which is synthesized in, and secreted by the Type II cell of the lung. Many investigators believe that lung compression/expansion destroys the surfactant and thus new material is

** Surfactant is essential for lung function. This conclusion is supported by the finding that immature infants suffering from Respiratory Distress Syndrome have reduced amounts of dipalmitoyl lecithin (the surface active phospholipid) in their lungs.

continually being discharged onto the alveolar surface (Clements, 1967). Because NO_2 toxicity results in initiation of Type II cell division there has been interest in the effect of NO_2 on surfactant.

Thomas and Rhoades (1970) have studied surfactant lecithin turnover in rats exposed to 5 ppm NO_2 continuously for two weeks. At the end of the exposure period the rats were injected (i.p.) with C^{14} radioactive palmitate. Three animals were sacrificed at 1, 2, 15 and 24 hours after injection of the radioactive palmitate and the radioactivity in the lung tissue dipalmitoyl lecithin determined. Thomas and Rhoades (1970) found that the biological half-life (the time it takes for half the radioactivity incorporated into a molecule to be removed) of surfactant dipalmitoyl lecithin was 12 hours in the control and 21 hours in the treated group of rats. The authors suggest that this decrease in renewal of surfactant diplamitoyl lecithin may account for some of the damage observed in the NO_2 exposed lung.

Blank et al (1978) have exposed rats to 15, 30 or 40 ppm NO_2 for five hours and then measured lung cell DNA synthesis, using radioactive thymidine incorporation as a marker; and surfactant lecithin content. Following exposure to 40 ppm NO_2 the rats were injected with tritiated thymidine one hour before sacrifice. They found that thymidine incorporation into total lung DNA can first be detected at 12 hours post exposure, peaks between 24-36 hours, and then declines. The authors interpreted the increase in thymidine incorporation as consistent with observed histological changes. Histological changes were first detected at 12 hours, and by 24 hours there was extensive hyperplasia. The hyperplastic reaction peaked at 30 to 48 hours and then noticeably declined by 72 and 96 hours. At 10 days the lungs of animals exposed to NO_2 and control animals were similar.

The amount of lung tissue lecithin was determined 24, 48 and 72 hours after exposing groups of rats to 15, 30 or 40 ppm NO_2 for 5 hours (Blank et al,1978). The data show that: a) the amount of lecithin in lung tissue significantly increases ($P<0.05$) following exposure to NO_2; b) the increase is proportional to the NO_2 exposure; and c) by 72 hours post exposure the amount of lecithin in the tissue begins to decline. Blank et al (1978) suggest three possible mechanisms to explain their findings. The lecithin accumulation in the lungs after exposure to NO_2 may result from: a net increase in synthesis; decreased removal; or an increased number of Type II cells. The relative contribution of each mechanism is currently not known.

Recently, Wright and Mavis (1981) and Wright et al (1982) have reported increases in levels of phospholipid biosynthetic enzymes in rat lung following in vivo exposure to 40 ppm NO_2 for five hours. Forty-eight hours post-exposure the rats were sacrificed and the activity of four enzymes involved in the biosynthesis of surfactant phospholipids were measured. The activity of two enzymes, glycerol-3-phosphate acyltransferase and glycerol phosphate phosphatidyl-transferase, significantly increased ($P<0.05$). As a control, the same enzymes were measured in the liver from the animals exposed to nitrogen dioxide and no change was observed. In the other study (Wright et al, 1982) Type II cells were isolated and assayed for enzymatic activity. The activity of all phospholipid biosynthetic enzymes significantly increased in Type II cells from rats exposed to 40 ppm NO_2. The authors concluded that their data are consistent with the finding that NO_2 causes Type II cell proliferation and an increase in surfactant phospholipids.

Williams et al (1971) exposed 12 rats to 15 ppm NO_2 for 28 days and then measured the surface-active properties of the material isolated from the alveolar spaces. The amount of lecithin isolated from the lungs of NO_2 exposed rats increased by 62% ($P<0.05$) as compared to the control. There were some small changes in the shape of the surface tension curve (a change in the total area of the curve or hysteresis loop) which the authors speculate might result from a direct action of NO_2 on the surfactant or contamination of the surfactant with material (protein) from injured cells.

Dowell et al (1971) exposed beagles to concentrations of NO_2 ranging from 3 to 16 ppm for one hour. At the end of the exposure period the animals were sacrificed and the amount of surface active lecithin in the lung alveolar space was determined. The data obtained suggest that the amount of surfactant lecithin declined in dogs exposed to 5 ppm or more NO_2 compared to controls. Exposure to 3 ppm NO_2 resulted in bleb (flaccid vesicle) formation in the alveolar endothelium, and exposure to 7 ppm or more resulted in intraalveolar edema in most dogs. The data from this experiment are difficult to assess since only one animal was used at each concentration of NO_2.

Dowell et al (1971) also found that <u>in vitro</u> NO_2 exposure (10,000 ppm for 0-30 minutes) had no effect on the surface active properties of surfactant. This observation is consistent with the fact that the major constituent of surfactant is dipalmitoyl lecithin, a compound which would not be affected by the oxidative effect of NO_2.

<u>Discussion:</u>

Investigations on the biochemical effects of NO_2 on lung tissue have been in two areas: the antioxidative defense system; and the

surfactant phospholipid/lecithin system. To date, the research on the antioxidative defense system has focused on the initiation of enzymes which would enhance the level of reduced glutathione in lung tissue (Ospital et al, 1981; Sagai et al 1981; and Ichinose and Sagai, 1982). Glutathione is the major nonprotein sulfhydryl compound in most cells, and plays an important role in the protection of cells against oxidative damage (Rouzer et al,1982).

Data from studies on the antioxidative system are consistent in the finding that, at 4 ppm NO_2 and above, there are significant increases in lung tissue glutathione reductase and peroxidase as well as other enzymes indirectly involved in maintaining increased levels of reduced glutathione. In addition, in the studies which measured oxidative products (Sagai et al, 1981; Ichinose and Sagai,1982) it was found that the level of oxidative products in the lung declined to control levels after induction of the antioxidative defense mechanism.

There are no data showing significant changes in lung tissue enzyme levels following exposure to less than 4 ppm NO_2. Ichinose and Sagai (1982), however, did obtain data showing an increase (not significant) in the level of some reducing enzymes in rat lung following exposure to 0.4 and 1.2 ppm NO_2.

The available data do not permit an evaluation which would include an exposure level at which there would be no detectable lung tissue damage. The methods currently used to assess lung tissue damage are limited in sensitivity because the assays relate change to the total

lung (enzyme activity/lung) while morphological studies indicate only lung surface cells are injured by NO_2 (the bronchiolar region and the alveolar region each comprise about 10% of the tissue mass). Assuming, that the antioxidative system is only induced in surface cells, and the increase in enzyme activity is dose dependent, then the increase in enzyme activity observed would have to be of a sufficient magnitude to overcome the diluting effect made by non-stimulated tissue since the enzyme activity is determined in whole tissue homogenates.

The observation by Ichinose and Sagai (1982) showing that there is a decline in the ability of the antioxidative system to protect the lung may be of particular importance in assessing chronic exposure to nitrogen dioxide. It should be noted, however, that the experiments conducted by these investigators were relatively short, 16 weeks, and the level of exposure at which the loss of the protective system was observed was high, 4 ppm NO_2. If further experiments do show similar trends, following chronic exposure to less than 1 ppm NO_2, in animals, the data will be important in evaluating the potential for adverse health effects for humans.

DeNicola et al (1981) found that there is a significant increase in the amount of various enzymes and blood constituents in pulmonary lavage fluid from hamsters following exposure to nitrogen dioxide. This suggests that this method may increase the sensitivity of detection of changes in lung tissue following exposure to NO_2 since there is no dilution by unaffected tissue.

Data from a number of laboratories have shown that following exposure to NO_2 there is both an increase in the amount of surfactant lecithin in the lung and an increase in the enzymes involved in its biosynthesis. These data also show that the increases in enzymatic

activity are probably confined to the Type II cell in which surfactant is synthesized. To date, there are insufficient data to determine if the increased enzyme levels are due to an increased number of cells or a net increase in the amount of enzyme in the cells. Also, the data are insufficient to determine if the increase in the amount of surfactant in lung results from an increased synthesis or decreased turnover. Nitrogen dioxide does not act directly on surfactant, since following in vitro exposure to very high concentrations there were no changes in its surface-active properties.

To date investigators have not suggested relationships which would attribute adverse or beneficial health effects to the increased amount of lung surfactant seen in some experiments following exposure to nitrogen dioxide. The increased amount of surfactant may simply reflect the increased number of Type II cells in the lung following exposure to NO_2.

Other

Resistance to Respiratory Infection

A model system has been developed by Coffin and Gardner (1972) to test an animal's resistance to respiratory infection following exposure to a pollutant. The test is relatively simple: groups of 20 mice are exposed to a pollutant for a period of time, then exposed to an aerosol of viable microorganisms for 15 minutes and mortality is then observed for 15 days. The enhancement of mortality, as compared to that of a control group* of mice which breathe only clean filtered air prior to exposure to the microorganism, is used for measuring the toxic effect.

Ehrlich and Henry (1968) reported that mice have an increased susceptibility to Klebsiella pneumoniae following exposure to 0.5 ppm NO_2. After three months exposure to 0.5 ppm NO_2 there was a significant ($P < 0.05$) increase in mortality of treated mice 60.5% (127/210 = dead/total) vs control mice 40.6% (72/180). Mortality increased to 70% (84/120) in the treated group compared to a control value of 46.3% (51/110) ($P<0.05$) after six months exposure. The authors also found that intermittent exposure, 6 hours/day, for six months significantly ($P< 0.1$) increased mortality in the treated group to 63.3% (38/60) vs a control value of 45% (27/60).

Gardner et al (1979) have reported on the effect of continuous and intermittent exposure to NO_2 on resistance to respiratory infection. In this study, groups of 20 mice were exposed to 0.5-28 ppm NO_2 for various periods of time ranging from a few minutes to several months, and then challenged with Streptococcus pyrogenes. A summary of some of their data is shown in Table I. A regression analysis of the data (all

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* The exposure to the microorganism is adjusted so that mortality is approximately 20% in the controls.

concentrations of NO_2 tested) was statistically significant at the P< 0.05 probability level. The data show that at each concentration (0.5-28 ppm) tested there was a linear correlation between length of exposure and mortality. Gardner et al (1979) also did a comparative analysis to determine the relationship of the toxic effect to concentration x time (CxT). The comparative data generated for a CxT = 7 is shown in Table II. As can be seen, concentration has a greater effect than time on mortality, i.e. mortality is greater following exposure to 28 ppm for 15 minutes compared to 3.5 ppm for 2 hours.

Gardner et al (1979) compared mortality following continuous and intermittent exposure. They found a statistically significant increase in mortality (about 15%) following continuous exposure to 1.5 ppm NO_2 for less than 24 hours. However, a significant increase in mortality was observed only after seven intermittent exposures (7 hrs/day) to 1.5 ppm NO_2. There was no difference in mortality in the two groups (continuous vs intermittent) after two weeks of exposure to 1.5 ppm NO_2. They observed no difference in mortality between intermittent and continuous exposure groups when the NO_2 concentration was increased to 3.5 ppm. Lastly, they calculated the relative mean survival time (time to death following exposure to S. pyrogenes) and found that survival time decreased following exposure to increasing concentrations of NO_2.

TABLE I

EFFECT OF VARIOUS CONCENTRATIONS OF NO_2 ON MOUSE MORTALITY AFTER EXPOSURE TO S. PYROGENES

NO_2	Exposure	Mortality
0.5 ppm	7 days	0.8%
	14	5.1
	30	5.0
	60	10.0
	90	19.0
	180	23.7
	270	12.3
	365	23.4
1.5 ppm	2 hrs	6.7
	5	0.0
	8	24.4
	18	25.0
	336	31.3
3.5 ppm	0.5	10.0
	1.0	8.0
	384	65.0
7.0 ppm	0.5	20.8
14.5	0.5	23.3
28.0	0.1	41.6

from Gardner et al 1979

TABLE II
INFLUENCE OF CONCENTRATION x TIME (CxT=7) ON ENHANCEMENT OF MORTALITY RESULTING FROM NO_2 AND S. PYROGENES

NO_2	Exposure	Mortality
1.5 ppm	4.7 hr.	6.4%*
3.5	2.0	18.7
7.0	1.0	30.2
14.0	0.5	21.7
28.0	0.25	55.5

* These are predictive values obtained from regression analysis from Gardner et al 1979

Gardner et al (1979) concluded from their study that differing concentrations and exposure times produce significantly different mortality responses. Concentration is the more important factor. To explain their findings they suggest that there may be more than one mechanism. At high levels, for short periods of time, the primary affect may be destruction of alveolar macrophages*, while long-term, low-level exposures may result in anatomical and biochemical changes.

In another study, Gardner (1980) has reported on animal resistance to respiratory infection following very low level exposures to NO_2. In this study, mice were continuously exposed for 14 days to 0.05, 0.5 or 1.2 ppm NO_2 for 14 days. During the exposure period the concentration of NO_2 was increased daily for one hour to twice the chronic exposure level. At the end of the exposure period the mice were challenged with microorganisms and mortality was observed. The data from this experiment are shown in Table III. The authors reported that according to the Chi square method exposure to 0.5 ppm plus a spike of 1.0 ppm or exposure to 1.2 ppm plus a spike of 2.5 ppm for 14 days resulted in a

* Alveolar macrophages, which function as the chief pulmonary defense, are cells found floating free in the alveolar space of the lung. They engulf bacteria which are then enzymatically digested.

significant increase in mortality as compared to controls, but exposure to 0.05 ppm plus a spike of 0.1 ppm did not produce a significant increase in mortality. However, if these data are recalculated according to the Fisher exact method the mortality following exposure to 0.05 ppm plus a spike of 0.1 ppm is significant ($P < 0.04$, two-tail test).

Table III

EFFECT OF VARIOUS LOW LEVEL CONCENTRATIONS OF NO_2 ON MOUSE MORTALITY

NO_2	Spike	Dead/Total
Control		93/319
1.2 ppm	2.5	135/315 +*
Control		55/239
0.5 ppm	1.0	85/240 ++*
Control		155/479
0.05 ppm	0.1	185/476 +++

* Chi square, significantlty different from control ($P < 0.05$). Fisher exact + = 0.0004; ++ = 0.004; +++ = 0.04. (from Gardner, 1980)

Larsen et al (1979) using the data of Gardner et al (1979) have developed a mathematical model which can be used to predict the excess mortality of mice following exposure to NO_2. These investigators found that excess mortality was proportional to the NO_2 concentration multiplied by exposure raised to the 0.33 power. The authors point out that the model is useful in predicting only mouse mortality. They do, however, suggest that similar exposures (0.5 ppm-28 ppm) would "probably only cause excess morbidity in men."

Larsen et al (1979) also reviewed the intermittent exposure studies of Gardner et al (1979) to determine if the data are applicable to a predictive model. They found that the model could be used if intermittent exposure was plotted as cumulative exposure. Cumulative exposure at 1.5 ppm NO_2 for less than 49 hours (7hr/day, 7 days/wk)

caused significantly less mortality than did continuous exposure. At 3.5 ppm NO_2 there was no difference between cumulative exposure and continuous exposure. This finding suggested to the authors that intermittent exposure to 1.5 ppm or less for short periods of time may permit the mice to partially recover from lung injury between exposures.

A number of laboratories (Giordano and Morrow,1972; Goldstein et al, 1973) have investigated the mechanism by which enhancement of mortality follows exposure to NO_2 and bacteria. The studies have focused on two possibilities: decreased ability of the lung to clear bacteria; and decreased ability to kill bacteria. Data, reported to date, have shown that there is decreased clearance (mucociliary transport) and bactericidal (alveolar macrophage function) activity following exposure, to 2 ppm NO_2. The data reported by these authors, however, show no change, compared to controls, in mucociliary transport nor bactericidal activity following exposure to less than 2 ppm NO_2, and thus give no insight into why animals are more susceptible to respiratory infection following exposure to less than 1 ppm nitrogen dioxide.

In a paper by Blair et al (1969) on induction of emphysema-like lesions in mice exposed to 0.5 ppm NO_2 (6 hr/day for 3 months) they observed a marked reduction in lung tissue response to bacterial challenge (the pneumonic lesion appeared to be more severe in mice exposed to NO_2, and the pneumonic process did not appear to be involved in areas which had marked lesions). This would suggest that the body defense mechanisms other than the mucociliary transport or the bactericidal activity of macrophages have been compromised.

Discussion

The resistance to respiratory infection as an end point is particularly useful since it assesses the effect of pollutants on the general health of the animal. The assay is unique because it gives no information as to how pollutants affect animals but rather answers the question: has the animal's total system been compromised.

The research by Gardner and his colleagues shows that mice continuously exposed to 0.05 ppm NO_2 (plus a one hour daily exposure to 0.1 ppm NO_2) or more are sufficiently compromised to be susceptible to increased bacterial infection. There is a significant increase in mouse mortality (6.6%) following exposure to 0.05 ppm NO_2 (plus a one hr/day spike of 0.1 ppm). At 0.5 ppm NO_2 mortality increased 10% after exposure for 60 days and to 20% after exposure for 90 days. A significant increase in mortality was observed in only two weeks when the mice were exposed to 0.5 ppm plus a one hour daily spike of 1.0 ppm NO_2. Their data suggest that intermittent and continuous exposure are similar when calculated as cumulative exposure. At low level exposure (1.5 ppm) observed mortality following intermittent exposure is less than the predicted value suggesting partial recovery.

Data from experiments on resistance to respiratory infection show that adverse health effects can be observed following exposure to 0.5 ppm NO_2. At the same exposure, assessment of morphological and biochemical data suggest that there is an adequate defensive response to the oxidant effects of NO_2 at least following subacute exposure. These differences suggest that NO_2 may affect as yet unidentified systems or a variety of systems at undetectable levels which in turn compromise the health of the test animal.

Non-Respiratory Effects

There are a limited number of reports in the literature on exposure to nitrogen dioxide and adverse effects not directly associated with respiratory function. There are two reports on kidney damage in guinea pigs exposed to 0.4-0.5 ppm for up to 14 days; a report on alteration in pentobarbital-induced sleep in mice exposed to 0.25 ppm; one report on alteration in brain enzyme levels in guinea pigs exposed to 0.53 ppm for 8 hours/day for 180 days; and a report on blood enzyme levels indicating liver and heart damage in guinea pigs exposed to 0.5 ppm for four months.

Function

In 1978 the American Thoracic Society prepared a comprehensive review of the health effects of air pollution. Included in the section on nitrogen dioxide, under animal studies, was the observation that mice exposed continuously to 0.5 ppm NO_2 developed emphysematous lesions. There are a large number of studies which have examined a variety of lung function parameters following exposure to NO_2. Animals exposed to NO_2 have been used as a model system to study emphysema.* Some investigators have reported a positive correlation between lung function and exposure to NO_2 while others have obtained no change in lung functions. Differences, seem to depend, to a large extent, on the parameters measured, and the length of exposure. For the purpose of

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* Emphysema once initiated is a progressive disease which is manifested by destructive changes in lung structure. The result of the changes is loss in ability of recoil and thus the lung stays in a position of inspiration. The etiology of emphysema is unknown, hence the lesions seen after exposure to NO_2 are referred to as emphysema-like.

this review positive studies, in which animals were exposed to low levels of NO_2, will be cited in detail, as well as any negative studies which utilize a similar protocol.

Gillespie et al (1980) exposed six month old beagle dogs to either 0.64 ppm NO_2 (plus 0.16 ppm NO) or 0.14 ppm NO_2 (plus 1.1 ppm NO) for 68 months. Lung function was assessed at the end of the exposure period and two years post exposure. As can be seen in Table IV, there were changes in some lung functions but not others. Significant changes were found in diffusion capacity, and dead space ventilation but not respiratory resistance. Some changes in lung function were only found two years post exposure. At the end of the exposure period there was no change in inspiratory lung compliance, but at the end of two years the NO_2 (high) group had increased from 30 ml to 40 ml ($P<0.025$) in lung compliance. Control and treated dogs had similar residual lung volumes at the end of the exposure period. However, two years post exposure in both NO_2 low & high groups residual lung volume of the dogs increased significantly. The residual volume in the low exposure group increased from 150 ml to 225 ml ($P<0.035$) and in the high exposure group it increased from 150 to 275 ml ($P<0.015$). The authors concluded that their observations were consistent with, and can be explained by parenchymal injury in dogs exposed to NO plus NO_2. The continued change in lung function post exposure is equated to a progressive emphysematous lesion.

TABLE IV

CHANGES FOLLOWING 68 MONTHS EXPOSURE TO NO_2

CONTROL vs NO_2 (Low) and NO_2 (High)

Measurement	Change (effect)
Pulmonary resistance	None
Total respiratory resistance	None
Chest wall resistance	None
Lung and chest wall compliance	None
Physiological dead space	NO_2 (low) significantly greater
Dead space ventilation	NO_2 (low) significantly greater
Pulmonary Diffusion Capacity/ Total lung capacity	NO_2 (low & high) significantly decreased
Pulmonary Diffusion Capacity	NO_2 (high) significantly decreased

from Gillespie et al (1980)

Kleinerman and Ip (1979) have reported that Syrian hamsters exposed to nitrogen dioxide have a net decrease in lung collagen and elastin.* Thirty-two hamsters were exposed to 30±5 ppm NO_2 for 22 hours/day. At 1, 4, 7, 10, 14 and 21 days after initiation of exposure five animals were sacrificed. A statistically significant net decrease in lung collagen was observed by the fourth day and elastin within 10 days. After 14 days of exposure total lung collagen returned toward pre-exposure levels. Total lung elastin did not return to normal levels until 10-15 days after termination of NO_2 exposure. Kleinerman and Ip compared their data to the evidence on elastase-induced experimental emphysema. They concluded that the alteration in lung elastin can best be explained by an increased elastase (enzyme which hydrolyzes the break down of elastin) secretion from alveolar macrophages.

Blair et al (1969) reported that mice exposed to 0.5 ppm NO_2 for 6 hours/day for 3 months have changes in lung tissue consistent with the development of emphysema-like lesions. Other groups of mice exposed to 0.5 ppm NO_2 for 18 and 24 hours/day for 6, 9 and 12 months also had early local emphysema-like lesions. The authors give no information on number of animals per group nor the incidence of lesions in each animal. The discussions by the authors suggest that there is an increase in the incidence of lesions with longer exposures.

Kosmider et al (1972) have reported that guinea pigs exposed to nitrogen oxides (mixture not described) for six months have a significant incidence of emphysema. Eighty guinea pigs were divided into two groups: 50 in the control group and 30 chronically exposed to

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* Collage is a structural component of the tissue and elastin is involved in the tissues ability to recoil after being expanded.

1 ppm nitrogen oxides. At the end of the exposure period the animals were sacrificed and the lung tissue examined for pathological changes. In the control group 15/50 animals had small emphysema foci, 15/50 had small atelectatic foci and 2 had an increase of mucus accumulation in the bronchi. In the exposed group 25/30 animals had small and large emphysema foci, as well as atelectatic foci, 9/30 had evidence of mucus accumulation in the bronchi and 12/30 had evidence of bronchial pneumonia. Consistent with these anatomical changes was the observation of increased hydroxyproline (the major amino acid in collagen and elastin) in the urine of guinea pigs exposed to nitrogen oxides. The authors concluded from their study that chronic exposure to nitrogen oxides caused emphysema-like lesions in guinea pigs. In addition, they suggest that the emphysema is the result of damage to lung collagen as evidenced by the increase in urine hydoxyproline.

In a similar experiment to that reported by Kosmider et al (1972), Drozdz et al (1977) exposed guinea pigs to nitrogen dioxide for six months. There were 90 animals, 30 in the control group and 60 exposed to 1.1 ppm NO_2 8 hours/day for 6 months. At the end of the exposure period, the guinea pigs exposed to 1.1 NO_2 showed pathological evidence of lung emphysema (numbers not given). Biochemical studies showed a statistically significant ($P<0.01$) decrease in lung collagen (control= 93.6mg vs exposed= 82.6mg; values are mg/gram of wet tissue). In addition, there was a significant increase in serum hydroxyproline (control = 44.7/ml vs exposed = 201.3/ml), and urine hydroxyproline (control = 1.4 micromoles vs exposed = 5.2 micromoles). The authors concluded from their study that chronic low level exposure to NO_2 results in lung emphysema, and the increased levels of blood and urine hydroxyproline is consistent with the loss of lung collagen.

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Discussion

A number of studies have shown that animals, chronically exposed to nitrogen dioxide develop emphysematous-like lung lesions. The data are, however, not entirely consistent. Kleinerman and Ip (1979) showed a loss of lung collagen and elastin in hamsters following exposure to 30±5 ppm NO_2 for three weeks, however, post-exposure follow-up indicated complete recovery. Kosmider et al (1972) and Drozdz et al (1977) showed emphysematous-like lesions in guinea pigs exposed to around 1 ppm NO_2 for 6 months. These investigators did not assess the ability of the lung to recover. It should be noted that Kleinerman and Ip showed a trend toward recovery: normal amounts of collagen were found in lung tissue after the second week of a three week exposure to nitrogen dioxide. The data from chronic exposure of guinea pigs to NO_2 would suggest that collagen destruction continued throughout the exposure period (Kosmider et al, 1972; Drozdz et al, 1977). The reason for the different results in these two studies is not readily apparent from the data.

The study by Blair et al (1969) indicate that exposure to 0.5 ppm for 6 hr/day for 3 months results in emphysema-like lesions.

The study by Gillespie et al (1980) suggests that chronic exposure of dogs to low levels of nitrogen oxides (0.14 ppm NO_2 plus 1.1 ppm NO) for 68 months resulted in some loss of lung capacity. Furthermore, the destructive damage which occurs during exposure to nitrogen oxides in dogs was not repaired and progressed to emphysema-like pathology 24 months post exposure. This observation supports the contention by some scientists that chronic exposure to nitrogen dioxide results in emphysema-like lesions.

HUMAN

Controlled Studies

Orehek et al (1976) exposed twenty asthmatics to 0.1 ppm NO_2 for one hour and then measured specific airway resistance and the bronchoconstrictor effect of carbachol (acetylcholine type drug). Each subject served as his/her own control. Exposures to unpolluted air and NO_2 were randomized in a single-blind fashion (the subject did not know exposure sequence) and were carried out on the same day. Each subject was tested twice. In 13/20 subjects there was a moderate (about 15%) but significant increase in specific airway resistance. In the same 13 subjects, carbachol produced a two-fold increase in specific airway resistance. In contrast, NO_2 neither modified the initial specific airway resistance nor the bronchoconstrictor effect of carbachol in the other seven asmathics. The authors concluded from their observations, "that some asthmatics exposed to low concentrations of NO_2 have moderate bronchial obstruction and a marked increase in their bronchial sensitivity to a bronchoconstrictor."

Kulle (1982) exposed twenty subjects with asthma and chronic bronchitis and 10 normal healthy subjects to 0.5 ppm NO_2 for two hours and then tested pulmonary function. The subjects acted as their own controls. No significant changes in pulmonary functions were observed in the subjects with asthma or bronchitis. However, when the subjects were analyzed as a combined group there was a significant decrease in lung compliance. Normal, healthy subjects also showed the same changes in lung function after exposure to NO_2. The author believed that the limited changes in pulmonary function were relatively insignificant. He

did speculate that slightly longer exposures, 4-6 hours, could result in significant changes in pulmonary function.

Sensitive Population Groups

In August 1982 the EPA Office of Air Quality Planning and Standards published a Review of the National Ambient Air Quality Standards for Nitrogen Oxides. Based on available health effects information the EPA staff concluded that "children, chronic bronchitics, asthmatics and individuals with emphysma appear to be most sensitive to exposures to nitrogen dioxide. Also, other individuals with impaired and/or sensitive respiratory or nasopharyngeal systems (e.g. individuals with symptoms of hay fever) may be more sensitive to NO_2. In addition, there is reason to believe that persons with cirrhosis of the liver or other liver, hormonal, and blood disorders, or those under going certain types of drug therapies may also be more sensitive to NO_2 because of the implications from animal studies showing increased systemic, hematological, and hormonal alteration after exposures to NO_2." The data cited by the EPA staff to support concerns about sensitive populations are summarized in Table V.

TABLE V

SUMMARY OF POTENTIALLY SENSITIVE GROUPS**

Sensitive Group	Supporting Evidence	References for Supporting Evidence	Population Estimates
Children	Children under age 2 exhibit increased prevalence of respiratory infection when living in homes with gas stoves. Children up to age 11 exhibited increased prevalence of respiratory infections when living in gas stove homes.	Speizer et al, 1980 Melia at al, 1979	age 0-5 17.5 million* age 5-13 36.6 million*
Asthmatics	Asthmatics reacted to lower levels of NO_2 in controlled human exposure studies	Kerr et al, 1979 Orehek et al, 1976	6.0 million*
Chronic Bronchitics	Chronic bronchitics reacted to low levels of NO_2 in controlled human exposure studies	Kerr et al, 1979 Von Nieding et al, 1971 Von Nieding et al, 1970	6.5 million*
Emphysematics	Emphysematics have significantly impaired respiratory systems. Because studies have shown that NO_2 impairs respiration by increasing airway resistance, it is reasonable to assume that emphysematics may be sensitive to NO_2.	Von Nieding et al, 1971 Beil and Ulmer, 1976 Orehek et al, 1976	1.3 million*
Person with Tuberculosis, Pneumonia, Pleurisy, Hay Fever or Other Allergies	Studies have shown that NO_2 increases airway resistance. Person who have or have had these conditions may be sufficiently impaired to be sensitive to low levels of NO_2	Von Nieding et al, 1971 Beil and Ulmer 1976 Orehek et al, 1976	unknown

Persons with Liver, Blood or Hormonal Disorders	NO_2 induces changes in liver drug metabolism, lung hormone metabolism, and blood biochemistry.	Menzel, 1980 Miller et al, 1980 Posin et al, 1979	unknown

*1970 U.S. Bureau of Census and 1970 U.S. National Health Survey
**All subgroups listed are not necessarily sensitive to NO_2 exposure at low levels.

Epidemiologic Studies

Kosmider et al (1972) examined a group of 70 workers exposed to 0.4-2.7 ppm NO_2 for 6-8 hrs/day for 4-6 years in the workplace. They compared a number of clinical findings (pulmonary function tests, chest x-rays, etc.) in this group of workers to those of a 80 workers not exposed to NO_2. There were no significant clinical symptoms of emphysema in the exposed group as compared to the control group. They did find a significant increase in hydroxyproline in the urine of these men as compared to the controls. Unfortunately, the amount of information supplied by the authors is not sufficient to fully evaluate their study and therefore diminishes its significance. The actual values and level of significance were not given. The authors made no attempt to compare the amount of hydroxyproline excreted with the level or length of exposure to NO_2. The NIOSH (1976) also reviewed this study and expressed concern about the lack of sufficient information.

The EPA Clean Air Science Advisory Committe has recently reviewed the EPA staff health assessment on NO_2. The comments by the Committee on adverse health effects in humans following exposure to nitrogen dioxide are summarized below (see Appendix A for complete discussion).

"The Committee concluded that none of the controlled human exposure studies offer definitive evidence that adverse health effects occur at levels below one part per million..... Community epidemiologic studies

... do not provide quantitative evidence of identifiable public health effects linked to specific ambient air concentrations of NO_2. .... The studies do provide, however limited qualitative support for the hypothesis that higher levels of NO_2, in association with other pollutants in the ambient air, may affect lung function and/or the onset of respiratory illness in children. .... The 'gas stove' studies and related studies in which NO_2 was measured in homes utilizing gas stoves suggest that repeated short-term peaks in the range of 0.15 - 0.30 ppm may be of concern for children and thus should be limited in the ambient air."

The Health Sciences staff (see Appendix C) have also reviewed the epidemiologic studies on the relationship between homes that use gas stoves for cooking and respiratory illness. The analysis notes, that for a variety of reasons there is no study which unambiguously links exposure to NO_2 and respiratory illness. Examples of two of the observations and the difficulties encountered are summarized below. The data from one study showed that there is a positive correlation between exposure to gas stoves and respiratory illness in very young children. In a follow-up study four years later, there was no positive correlation between exposure to gas stoves and respiratory illness in the same children. This difference raises a number of questions. Was the first observation valid? Are young children more sensitive than older children? Are young children adversely effected because they spend more time inside the house?

In another study, the incidence of respiratory problems in non-smoking males was significantly greater in houses with gas stoves compared to electric stoves. However, in the same study there was no

effect on women under similar conditions. Again, apparent inconsistencies raise a number of questions. Was the correlation between respiratory problems in non-smoking men in houses with gas stoves an artifact of data collection? Did the background level of respiratory problems in women mask a positive correlation? Should occupational exposure to pollutants be a factor in evaluating the incidence of respiratory illness in men?

Given these kinds of uncertainties and apparent contradictions the staff concluded: "The studies of cooking energy source indicate a trend of increased respiratory illness associated with the use of gas."

SUMMARY

There is sufficient evidence from animal studies to show that exposure to nitrogen dioxide can result in adverse health effects. The primary target for nitrogen dioxide exposure is the lung. Morphologic and biochemical studies show that following exposure to more than 1 ppm NO_2 there is a sequence of reactions in lung tissue. First is the loss of ciliated cells in the bronchiolar region, followed by the loss of Type I cells in terminal alveolar sacs. Cell death peaks around 24 hours post exposure and is then followed by cellular renewal. Data show that the new cells, exposed to the same concentration of NO_2, are more resistant to damage. It is believed that the reason for this resistance is an increase in the amount of the enzymes associated with protecting the tissue from the oxidative effect of NO_2 by maintaining a high level of reduced glutathione. In addition to cellular renewal and an increase in the amount of reduced glutathione there is also an increase in the amount of surface-active material covering the cells in the alveolar sacs. Only lung cells directly exposed to the atmosphere appear to be affected by NO_2. Since these cells comprise only a small portion of the total lung tissue, detection of alterations in metabolism is limited by the diluting effect of normal tissue.

Animals exposed to NO_2 have a significant decrease in their resistance to respiratory infection. Scientists have also shown that there is a relationship between length of exposure, continuous or intermittent, and increased mortality from bacterial infection. Mice continuously exposed for 14 days to 0.05 ppm NO_2 (plus a one hr/day spike of 0.1 ppm) have a 6.6% increase in mortality following exposure

to bacteria. Mice continuously exposed to 0.5 ppm NO_2 for 60 days have a 10% increase in mortality following exposure to bacteria. The mortality incidence increases to 20% if the exposure to 0.5 ppm NO_2 is for 90 days. These findings have suggested to investigators that humans exposed to nitrogen dioxide might have an increased morbidity.

Lastly, animal studies have shown that exposure to low levels (0.14 ppm + 1.1 ppm NO) of NO_2 for long periods of time can result in emphysema-like lesions in lung tissue. Gillespie et al (1980) found that dogs exposed continuously to about .14 ppm NO_2 (+1.1 ppm NO) for 68 months and then allowed to breathe unpolluted air had progressive changes in lung function consistent with emphysema-like lesions. In addition, a study by Drozdz et al (1977) showed that guinea pigs exposed to 1.1 ppm NO_2 for 8 hrs/day for six months also developed emphysema-like lesions. This latter observation is of particular significance and concern since it suggests that an 8 hour exposure to 1 ppm NO_2 followed by a 16 hour recovery period also results in irreversible damage to lung tissue.

There are a limited number of studies which have attempted to relate either chronic exposure in homes and occupational settings or known exposure studies in laboratories to NO_2 with adverse health effects in humans. These studies have two limitations, lack of long term follow-up to assess the risk of irreversible emphysema-like lung damage and the inability to distinguish the effect of NO_2 from other pollutants in epidemiologic studies. In the known exposure studies people have been exposed to low levels of NO_2, 1 ppm or less, for a few hours and then had lung function tests. Depending on the parameter measured the results have been either negative or positive. Despite the limitations of the various studies, the data collected to date suggest

that individuals who are at special risk, such as asthmatics, might be adversely affected by low level exposures to NO_2 for short periods of time. This concern is supported by the observation of Orehek et al (1976) that some asthmatics exposed to 0.1 ppm NO_2 of one hour have increased airway resistance.

There is one study, although containing very limited data, which does relate findings in humans which are similar to those reported in animal studies. Kosmider et al (1972) found no significant clinical symptoms of emphysema in a group of 70 men exposed to 0.4-2.7 ppm NO_2 for 6-8 hrs/day for 4-6 years. However, they did find a significant increase in hyroxyproline in the urine of these men as compared to the controls. The latter observation is of particular interest because it can be related to the studies in which guinea pigs exposed to 1.1 ppm NO_2 for 8 hrs/day developed emphysema and had an increased urinary excretion of hydroxyproline (the major amino acid found in collagen and elastin).

Recently the EPA Clean Air Science Advisory Committee (CASAC) reviewed the controlled human studies and concluded that there was no definitive evidence that adverse health effects occur at levels below 1 ppm NO_2. They also concluded that the data should be considered along with other evidence when considering standards.

The CASAC also reviewed the epidemiological studies and concluded that the identified studies "do not provide quantitative evidence of identifiable public health effects linked to specific ambient air concentration of NO_2 . . . The studies do provide, however, limited qualitative support for the hypothesis that higher levels of NO_2, in association with other pollutants in the ambient air, may affect lung function and/or the onset of respiratory illness in children . . . The

CASAC concurs with the staff paper recommendation that an annual primary standard within the range 0.05-0.08 ppm will offer sufficient protection for the various welfare effects of concern".

The Health Science staff has also reviewed the available epidemiologic studies on exposure to NO_2 which results from the use of gas stoves and related health effects. The staff analysis concluded that the studies indicate a trend of increased respiratory illness associated with the use of gas. None of the studies definitively show a relationship between exposure to NO_2 and respiratory illness.

A concern previously expressed by the Health Science staff (1982) still remains: "A question still remains as to whether there is a threshold for a detrimental effect from low level, chronic exposure to NO_2. The fact that there is a measurable pulmonary effect, i.e. decreased airway resistance at 1.5 ppm NO_2 for 15-45 min., does not indicate the presence or absence of a lasting pulmonary response. NIOSH has suggested that NO_2 exposure should be kept to a minimum to avoid the possible development of chronic lung disease, but the threshold exposure above which such dysfunction would develop is not known."

Conclusion

The staff has reviewed the current literature on adverse health effects associated with exposure to NO_2, previous health assessment documents prepared by other federal agencies and the recent comments made by the EPA Clean Air Science Advisory Committee. Based on the available data from animal studies one can reasonably be concerned that exposure above 0.05 ppm NO_2 (+ a one hour/day spike of 0.1 ppm NO_2) for 6-8 hours/day could be associated with an increased incidence

of respiratory infection, and that chronic exposure above 0.14 ppm (+1.1 ppm NO) or 0.5 ppm NO_2 could result in irreversible lung damage. In addition, animal studies which compare continuous vs intermittent exposure indicate that intermittent exposure can be associated with an increased susceptibility to respiratory infection. Furthermore, the intermittent exposure studies suggest that the level of exposure may be significant in determining the potential for adverse health effects since it has been shown that intermittent exposures are additive. There are two human studies which support the staff concern, the observation that some workers exposed to 0.4 - 2.7 ppm NO_2 for 8-10 hours/day have an increased urinary excretion of hydroxyproline suggesting a breakdown of lung tissue, and some asthmatics are adversely effected by a one hour exposure to 0.1 ppm NO_2.

The staff believes that there is a need for further research which will define the levels of exposure to NO_2 which are not associated with adverse health effects.

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APPENDIX C

Analysis of Epidemiological Studies Involving NO_2

A thorough search of the literature has revealed a number of epidemiological studies which relate adverse health effects to the presence of suspected sources of pollutants such as the use of gas for cooking. An analysis of these studies is given, with specific attention paid to the role of NO_2 as a potential causative agent.

A set of studies by Melia et al. (1-7) deals with the effects of the use of gas for cooking on the respiratory system in children. The adverse effects investigated include morning cough, day or night cough, wheeze, colds going to chest, asthma and bronchitis, collectively termed respiratory illness. The control group consisted of children (6-11 years old) living in homes where electricity was used for cooking. For their original 1973 cohort, 3204 children living in homes where electricity was used for cooking and 2554 in homes where gas was used were identified. Although the authors made many comparisons in their analyses of the raw data, the best comparison is after certain differences between the two groups have been taken into account, such as effects of social class, age and location. The result was that there was a significant association ($p < 0.05$) between the use of gas and respiratory illness for girls as compared to the use of electricity, but not for boys ($p \approx 0.3$). When the data were adjusted for

social class and age, but not location, the association between gas cooking and respiratory illness for boys was greater (p less than 0.07; for girls p was less than 0.001). It should be noted that no correction was made for smoking in this study (1).

In 1977, these authors did two subsequent analyses (2). First, the 1973 cohort was followed up; second, a new cohort of 3017 children 6-11 years old from homes using electricity and 1810 from those using gas was identified. By the same methodology as used before, the authors found, in the follow-up of the 1973 cohort (2408 of the 5758 were able to be followed up), that on the whole, use of gas for cooking was still more associated with respiratory illness than use of electricity. There was some variation, however; in some cases, the relative risk, which was greater than 1 in 1973, fell below 1 during follow-up (below 1 means that the illness in electric homes was greater than that in gas homes). Though there is no way to discover the source of this variation, one reason could be that younger children are more susceptible to the effects of using gas for cooking. For the new cohort, weighting for age and social class, the relative risks were 1.25 for boys (p less than 0.05) and 1.19 for girls (marginal significance, p approximately 0.07); again, relative risks over 1 mean that gas homes have a higher respiratory illness rate than electric homes with respect to cooking. The authors extended the analysis to take into account other factors such as location, smoking, outdoor pollutant levels, overcrowding, and others, and found that respiratory illness was still associated with gas cooking in boys (p less than 0.02) but not significantly in girls (p approximately 0.15). Only 1032 boys and 950 girls had sufficient

information to be included in the latter analysis.

In related papers (3-4), the authors showed that NO_2 levels were higher in kitchens and children's bedrooms in homes where gas was used for cooking as compared to electricity. They also identified a cohort of 431 children, 6-7 years old, from gas cooking homes and 96 from electric cooking homes. They found, after adjustment for various factors, that there was a weak, but nonsignificant, association between gas cooking and respiratory illness (p approximately 0.11). In another analysis, they found no association between gas cooking and changes in lung function. Also, a borderline correlation (p approximately 0.05) was found between bedroom NO_2 levels and illness in 103 of the children. The authors state, however, because of inconsistencies between their study and others, it is possible that the NO_2 levels were a proxy for another factor more related to respiratory illness such as temperature or humidity (5).

In a final set of papers, these authors explored the factors of temperature and relative humidity, initially finding no association between the use of gas vs electric cooking and either of these factors in a 40 home study. They did, however, confirm earlier results by finding levels of NO_2 significantly higher (p less than 0.05) in gas cooking homes. In a larger home sample (337 homes), no correlation was found between NO_2 levels and either temperature or relative humidity (6). A study was then performed on 183 of the 337 homes where gas was used for cooking and complete information was available. After adjusting for age, sex, social class, smoking, temperature and relative

humidity, no relation was found between level of NO_2 in the bedroom or living room and respiratory illness in 5-6 year old children (179 children total). A significant association, however, was found between relative humidity and respiratory illness. The authors concluded that if NO_2 causes such illness, its effect is weak and may not be easy to detect in small samples (7).

A final analysis of the Melia et al set of studies leads to a strong suggestion that the use of gas for cooking is related to respiratory illness, but such an association is by no means proved. The studies do not allow a definitive cause-effect relationship to be established because of variations between and within the studies, such as finding significant effects in boys but not girls one time, the opposite another time, and even finding some negative associations (i.e. favoring electricity for cooking related to respiratory illness). Whether or not adjustments were made for various factors and which factors were adjusted for seemed to make large differences in the significance of the results; the more adjustment made, the lower the level of significance seemed to be. Still, though the results are not significantly consistent in one direction, an overall trend towards the association between respiratory illness and gas cooking is indicated. From these studies, neither NO_2 nor other factors can be ruled out or accepted as the sole or contributing cause of the respiratory illness observed.

Two papers by Keller et al also examine the relationship between gas vs electricity for cooking and respiratory illness. Various comparisons

were made for subsamples of a 1952 member study sample from 441 families (8). No relationship was found between the use of gas cooking and respiratory illness or overall lung function for any comparison; for mothers, the incidence of such illness was higher, though not significantly, in electric homes. Also, no relationship was found between NO_2 level and respiratory illness rates in gas cooking households (83 home sample). A second study of 60 gas cooking homes matched demographically with 60 electric cooking homes, again found no relationship between gas cooking and respiratory illness in mothers, fathers, children 12-18, or children under 12 (9). In fact, in one case (children 12-18), the illness rate was significantly higher in electric homes. In analyzing these studies, it should be noted that the 120 homes in the latter study were also part of the former, and though no significance was found to associate gas cooking or NO_2 with respiratory illness, the sample size was relatively small to detect a weak association (i.e. Melia et al had difficulties showing a trend with larger samples).

Speizer et al (10,11) have performed a large study of 8120 children again looking for the effect of gas vs electric cooking on respiratory illness, and differences in NO_2 level. As in some previous studies, a significantly higher level of NO_2 was found in gas cooking homes vs electric cooking homes. Before adjustment for various factors, no significant association was found between the use of gas for cooking and bronchitis, respiratory illness before age 2, or respiratory illness during the last year. After adjustment for factors such as smoking and socioeconomic status, the authors found a significant association (using

statistical tests looking for correlation) between serious respiratory illness before age two and the use of gas for cooking. Such illness was also found to be significantly associated with social class, parental smoking, and city-cohort (six different cities were included in the study). When rates of respiratory illness before age two were corrected for smoking and city-cohort, the adjusted rates were 249 and 214 cases of illness per thousand in males for gas and electric homes, respectively; for females the rates were 205 and 175 gas gas and electric, respectively. Though the authors mention this difference, no discussion is given as to the statistical confidence of these values. Additionally, the authors found an association between gas cooking (as compared to electric) and lowered forced vital capacity (FVC) and forced respiratory volume (FEV_1). In summary, this study indicates a relationship between gas cooking and respiratory illness, but the illness may be related to other factors as well. Though no mention of statistical confidence is made with respect to the adjusted rates of respiratory illness, these rates are always higher in gas cooking homes as compared to electric, even within specific cities. This result would be a very unlikely one if there was, in reality, no association between respiratory illness and the use of gas for cooking. Thus, a trend is definitely indicated, and the changes noted in the lung function tests may also be important. With regard to NO_2 as a causative factor, however, neither it nor other factors can be ruled out or accepted as the sole or contributory cause on the basis of this study.

A paper by Comstock et al (12) examined the relationships between respiratory symptoms (cough, phlegm, wheeze, breathlessness and chest illness), the use of gas vs electricity for cooking, and smoking among a

cohort of 1724 people. In ex-smokers and current smokers, although the relative risks associated with gas as compared to electricity were greater than one for most of the symptoms, none of them were significant. For 68 male non-smokers in gas homes vs 319 non-smokers in electric homes, the relative risks of 2.44, 1.70 and 4.31 for cough, wheeze and breathlessness, respectively, were significant (p less than 0.05). In non-smoking women, however, a relative risk of 0.01 for breathlessness was significant (i.e. electricity for cooking causing more of the symptom than gas - 71 women in electric homes vs 33 in gas). Thus, this study, although showing some respiratory ailments associated with gas cooking in male non-smokers, is overall too limited to lead to a definitive conclusion. A study using larger sample sizes might clarify the results.

A study by Dodge (13) compared the prevalence of various respiratory problems in homes where gas vs electricity was used for cooking. A cohort of 427 Arizona children, mostly 8-10 years old, was identified. Rates of asthma, wheeze, and sputum production were elevated, but not significantly, among children in gas homes. The rate of cough, however, was significantly elevated in gas as compared to electric homes. The author found that there were no differences in lung function (FEV_1) or lung growth between the two groups. One problem with the study is that it is difficult to tell how matched the two groups were; even though parental smoking was also determined, neither this nor any other factor was mentioned by the author as being taken into account in the statistical analysis. The author concludes that the study provides evidence that home cooking does not produce serious respiratory problems. It should be noted, however, that the study size was relatively small and might not be able to detect such effects.

Lung function tests ($FEV_{0.75}$) were performed on 11,211 children aged 6-13 in a study by Hasselblad et al (14). The authors found a significant change in lung function among girls aged 9-13 living in homes where gas is used for cooking as compared to electric, but such a finding was not observed in girls aged 6-9 or in boys in either age group. It was proposed that this might reflect an increased amount of time spent in the kitchen in this particular group, or that it might be due to the effects of an interaction between possible increased smoking in this group and exposure to emissions from gas stoves. In any event, the authors consider the observed change to be small. Also, the problem of matching described above in other studies would apply here. Therefore, though the study may be indicating a relationship between the use of gas for cooking and altered lung function, no firm conclusion can be drawn from the study data.

A set of papers by Shy et al (15-16) examined the relationship between adverse health effects and NO_2 released by an industrial facility near the tested community. Ventilatory performance (FEV) was significantly (p less than 0.05) impaired among 306 children in the test area, with higher average NO_2 levels, as compared to 489 children in control areas with lower average NO_2 levels, although the authors state that the differences were small in magnitude (15). When studying the relationship between respiratory illness and high NO_2 vs two separate control areas, the authors found a statistically significant excess respiratory illness rate among all family segments (fathers, mothers, siblings, second graders) in the high NO_2 area. On the average, the biweekly respiratory illness rates (per 100 persons) were 17, 14 and 15 in the high NO_2, control 1, and control 2 areas respectively (total 4043 individuals in the study). Such a relationship was generally not found

for a high particulate area vs controls. The study was complicated by outbreaks of influenza, but after correcting for this, the authors still found that the association between high NO_2 and respiratory illness was significant (16). The study suffers from the lack of information on other potential causes of the observed symptomology, such as smoking, since adjustments were not made for many of the factors of the type addressed in the gas vs electric cooking studies. The authors mention that parental smoking had no significant effect on respiratory illness in second graders, but in the high NO_2 area, the average biweekly respiratory illness rate per 100 persons increased from 20.7 (non-smoking) to 22.2 (smoking), with no such increase among controls. Thus, correction for smoking and other factors should have been made for the analysis of the entire cohort.

Another problem with the study is whether or not other outdoor pollutants were present (e.g. released from the factory in the high NO_2 area). Additionally, there is a problem with the determination of a subject's NO_2 exposure. NO_2 was measured at sites representing areas near schools. Three sites near three schools in the high NO_2 area were monitored; they averaged 0.109, 0.078 and 0.062 ppm NO_2. The two control sites averaged 0.063 and 0.043 ppm. Thus, one high NO_2 site was almost identical to a control area and a second NO_2 site was not too different. Within the high NO_2 area, no dose-response relationship between NO_2 level and respiratory illness was observed. In other words, there was no difference between the illness rates from the 0.062 and the 0.109 ppm sites in the high NO_2 area. Yet, if one looks at the data, there seems to be a large difference between the illness rates from the 0.062 high NO_2 and the

0.063 control in all four family member categories (second graders, 20.4 vs 18.0; siblings, 19.1 vs 15.6; mothers, 13.4 vs 11.8; fathers, 12.1 vs 8.8; 0.062 high NO_2 vs 0.063 controls respectively). This difference in illness rates at similar NO_2 concentrations may be due to the fact that the method used to determine NO_2 levels was later found to be unreliable (17). Thus the relationship between NO_2 level and illness rate or ventilatory performance in the entire study is questionable. Other reasons for the discrepancy may include location of subjects with respect to measured site (and accompanying NO_2 level differences), percentage of time of subjects in the immediate area, relationship between indoor and outdoor NO_2 levels, other pollutants, etc. Therefore, although the study suggests a correlation between elevated NO_2 levels and respiratory illness, the relationship is far from being proved conclusive.

A subsequent study on the same geographical area (17) was performed to determine if, four years later, the elevated NO_2 levels and the authors' previous findings concerning respiratory illness continued. This time a multivariate analysis was conducted to correct for smoking, crowding, educational status, history of disease, and the use of gas stoves for cooking. The cohort (175-275 families) exhibited, in all four categories (fathers, mothers, school children, preschool children), increased respiratory illness in the first half of 1972 in the high NO_2 area as compared to lower NO_2 areas. In September to December, 1972, or in January to April, 1973, this was not the case, however. The authors found the respiratory illness rate to be significantly higher in early 1972 as compared to early 1973. One reason for the latter findings was a strike at the industrial facility from January to March of 1973, but

this does not explain the late 1972 findings. The NO_2 levels given in the paper lead to confusion, because several different methodologies of NO_2 level determination were used. Reliable measurements were not conducted under contract for the authors before September 1972; there was no consistent difference between high NO_2 and other area average NO_2 levels anytime from September 1972 to March 1973 (in fact, "high NO_2" area levels were lower than "low NO_2" area levels in early 1973). Levels for early 1972 were termed not reliable by the authors, which is the time period when elevated respiratory illness rates were observed in the high NO_2 area. Therefore, due to this and the always present problem of the role of other outdoor pollutants, this study cannot be used to associate increased NO_2 level with a higher rate of respiratory illness, as is admitted by the authors when they state "it was not possible to attribute the excesses in illness to specific pollutants". The authors stated that due to the dramatic decrease in illness coinciding with the strike, short-term exposure may be more important than long-term exposure.

A similar study was conducted by Kamat et al (18) who tried to correlate NO_2 and other pollutant levels in various urban and rural areas with respiratory effects. The authors found that NO_2 level significantly correlated with frequent colds, cough and breathlessness in a cohort of over 4000 subjects. The problem with this type of analysis, however, is that correlation, not comparison of test and control populations, was the statistical technique used. This type of analysis, plus the fact that many other factors could be the cause of differences in respiratory illness rates when urban/rural areas are compared with each other, leads to the conclusion that this study is not useful in establishing a

relationship between NO_2 and respiratory illness.

The final study reviewed is an examination of the relationship between exposure to nitrogen oxides (including NO_2) and changes in lipid metabolism (19). Chemical factory workers (70 men) exposed to 0.4 to 2.7 ppm nitrogen oxides 6-8 hours daily for 4-6 years were compared to a control group of healthy, nonexposed workers (80 men) in the same age group. The authors found that compared to controls, total cholesterol and esterified cholesterol in blood serum were significantly decreased ($p < 0.001$) in the exposed workers. The authors, however, note that this effect is opposite of what is found for guinea pigs exposed to nitrogen oxides. No differences between the two groups were observed for free cholesterol levels. Among exposed workers, total lipids, and beta and gamma lipoproteins in serum were significantly increased ($p < 0.01$-0.001) and alpha lipoproteins were significantly decreased ($p < 0.001$), compared to controls. The major concern about this study is that other pollutants might have been present in the chemical factory so that the effect cannot be attributed to nitrogen oxides. Additionally, the relationship between these altered lipid levels and adverse health effects remains to be elucidated.

SUMMARY

Generally, the studies reviewed attempt to relate NO_2 exposure with an increased rate of respiratory illness or other symptoms. The studies of cooking energy source strongly suggest that increased respiratory illness is associated with the use of gas. This association is, however, not definitively proven since there are no consistent findings

of a significant nature within or between studies indicating that the use of gas alone causes increased respiratory illness. Much variation also occurs as a result of the type of statistical analysis performed, including what factors were or were not adjusted. Additionally, other pollutants (such as NO, formaldehyde, particulates) may be released from gas stoves so that any health effects could not be attributed solely or partially to NO_2, even though NO_2 levels are increased, based on these studies. The fact that NO_2 is a component of the released gases suggests that it may be a causative factor.

The studies correlating outdoor NO_2 levels with adverse health effects do not provide much more than a weak indication that there may be a relationship between the two. Major problems include inconsistencies in measurement methodology, the relationship between measured levels and those actually experienced by the cohort (both indoors and outdoors), the fact that increased NO_2 levels do not always correlate with increased respiratory illness rates, and the contribution of other potential sources to the illness rate.

Though the results from negative studies are also reported in this paper, it should be clear that none of them provide a conclusive indication that NO_2 is not associated with adverse health effects, again due to the limitations of these studies.

In summary, though no conclusive determination can be made, the reviewed studies suggest that NO_2 is potentially a causative factor for respiratory illness.

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UNITED STATES GOVERNMENT

Memorandum

U.S. CONSUMER PRODUCT SAFETY COMMISSION
WASHINGTON, D.C. 20207

TO: The Commission
THROUGH: Sadye Dunn, Secretary
THROUGH: Martin Howard Katz, General Counsel
THROUGH: Edward Morgan, Executive Director
THROUGH: Andrew G. Ulsamer, Acting Associate Executive Director for Health Sciences
FROM: Sandra Eberle, Program Manager
Chemical Hazards Program

NOV 27 1984

SUBJECT: Status Report on Kerosene Heaters

BACKGROUND

In June, 1983 the Commission staff submitted a comprehensive analysis of the health and safety hazards associated with the use of unvented kerosene heaters. A substantial portion of the CPSC staff efforts and concurrent industry studies dealt with the issue of indoor air quality. The staff report included consumer use survey data, laboratory generated emission rate data, expected levels of pollutants in homes under various situations generated by mathematical modeling techniques, and actual observed levels of pollutants in homes where consumers used kerosene heaters. This information, along with in-depth assessments of the potential health effects of the various pollutants, formed the basis for the staff conclusions on the expected health consequences of consumer use of kerosene heaters.

As a result of that staff report, and an industry-prepared report issued in July, 1983, a number of efforts to improve the overall safety of kerosene heaters have been undertaken by the industry and by Underwriters Laboratories. In the National Kerosene Heater Association's resolution addressing the staff recommendations it was agreed that the industry would work with the Commission staff and U.L. to address the need for an emission standard on nitrogen dioxide, the pollutant the staff concluded was of greatest health concern.

PURPOSE

The purpose of this report is to update the information contained in the June, 1983 briefing package and to report on the progress made in improving the indoor air quality performance of kerosene heaters.

HEALTH ASSESSMENTS

The staff has reviewed the health effects studies on nitrogen dioxide and carbon monoxide published since the in-depth assessments were prepared for the June 1983 briefing package. The new studies, in general, add further support to the staff conclusions.

NOTE: This document has not been reviewed or accepted by the Commission.
Initial [illegible] Date 11/27/84

For example, new data indicate that toxic events such as cellular damage, previously only shown after exposures to high levels (2 ppm) of nitrogen dioxide, are detectable in animals after exposures to about 0.4 ppm. Additional studies suggest that lung tissue of older animals may be more susceptible to the adverse effects of nitrogen dioxide. Exposure to nitrogen dioxide is also associated with alteration in immunological function in other studies, which might begin to explain the observed decrease in resistance to respiratory infection. Some new data on nitrogen dioxide indicate that there is a relationship between nitrogen dioxide uptake and the excretion of nitrosamines. Because nitrosamines are potent carcinogens these studies raise new concerns regarding the effects of low level chronic exposure to nitrogen dioxide.

NEW TECHNOLOGY AND KEROSENE HEATERS

The emissions testing of kerosene heaters has continued at CPSC. The testing protocol is the same as that used for the testing reported in June 1983. Two advances in technology have been investigated (TAB A):

- a radiant and a convective heater each with a new design utilizing a dual combustion chamber, and,
- add-on catalytic devices.

The results from the advanced technology heaters are impressive. Nitrogen dioxide emission rates for both the radiant and convective heater were reduced by 75 to 80 percent over those tested in 1983. Carbon monoxide emission rates showed a 90 percent reduction from the blue flame (generally radiant heaters) and about a 50 percent reduction compared to the white flame (convective heaters) tested in 1983. This testing was, however, limited to two models of each of the two advanced technologies designs.

The staff tested three types of catalytic add-on devices. Although these models may not be representative of the entire market, the staff preliminarily concludes that, as a class, these devices reduce nitrogen dioxide emission rates by 40 to 50% and carbon monoxide emission rates by 10% for blue flame heaters and 60% for white flame heaters.

Table 2. Emission Rates for Kerosene Heaters 1984

Heater type	Average Emission Rate (cc/kJ) 1983 Data	1984 Data Catalysts (3 types)	1984 Data Dual Chamber (2 Heaters)
Radiant and convective blue flame			
NO_2	0.0025	0.0013 (range 0.001 to 0.002)	0.0004
CO	0.11	0.09 (range 0.001 to 0.014)	0.01
Convective white flame and wickless			
NO_2	0.0038	0.0015 (range 0.001 to 0.002)	0.0008
CO	0.02	0.01 (range 0.009 to 0.01)	0.01

The technological advance represented by the dual combustion chamber substantially reduces concern for adverse health effects among the general population from nitrogen dioxide or carbon monoxide emitted by these heaters. Although a full modeling analysis has not been done, it is unlikely that even in a closed room carbon monoxide concentrations could exceed eight parts per million or that nitrogen dioxide levels would exceed 0.1 ppm.

At this time we are not aware of other innovations in burner designs by other manufacturers, but the dual chamber models tested were on the market late last year and will be marketed this season in the suggested retail price range of $160 to $180 (ECON memo TAB C).

The add-on catalytic devices provide an option for consumers to improve the indoor air quality performance of previously purchased heaters. However, even with these devices the public is well advised to use the heater in rooms at least partially open to the rest of the house. The staff will continue to monitor the market and to test the performance of these devices during FY1985.

VOLUNTARY STANDARDS ACTIVITY

Under the auspices of Underwriters Laboratories, Inc., a task force with representatives from the kerosene heater industry, CPSC staff, U.L., and the consuming public (represented by a technically skilled academic from the public health community) have been meeting to assist U.L. in the development of consensus standards on indoor air quality issues for use in the U.L. certification test program.

During the three meetings eld so far the primary item under consideration has been the dev opment of a test methodology for the measurement of various pollutar gases. The start of this program has been delayed due to the lack oi appropriate instrumentation. U.L. notified us by letter dated 10/ 5/84 that it has now obtained. The task force is encouraging testing tc determine the practicability and feasibility of using a hood met od for measuring kerosene heater nitrogen dioxide emission rate . Presently, the staff believes that chamber derived emission rates are most applicable for modeling the pollutant concentrations to be xpected in homes where heaters are used. However, U.L. would like to be ble to use a faster and less expensive test method than the controlle environmental chamber. Thus, comparative studies are needed of emission rates derived from the various sampling strategies, i. . chamber, probe and hood techniques. Studies of this type have now en initiated at U.L. (TAB B, letter from U.L. dated 9/28/84) under the idance of the task group. Studies which may provide some additional da a of a similar nature have been undertaken by the Environmenta Protection Agency and the Lawrence Berkeley Laboratory through th coordination efforts of the federal Committee on Indoor Air Qualit . CPSC will also undertake some chamber to hood coorelation studies us g our chamber at the Engineering Sciences Laboratory.

Controversy continues ove the appropriate emissions level at which to set the nitrogen dioxide st dard. Discussions between CPSC staff, U.L., and the industry are on- oing and seek agreement on a process by which a consensus position can e reached. The memorandum submitted by the National Kerosene Heater A sociation (TAB D) is part of this process.

SUMMARY REPORT ON NIEHS HEALTH AND HEATING SURVEY

It was through funding of a small portion of this major study by the National Institute of Envi onmental Health Sciences that we obtained the in-home monitoring data re ortad in the June 1983 report. The study, conducted by the John P erce Foundation of Yale University measured concentrations of nit ogen dioxide in 155 homes with kerosene heaters and 148 homes without erosene heaters over six two-week periods from January - March, 1983.

The Pierce study (TAB A) onfirmed earlier data and assumptions that nitrogen dioxide concentr tions are relatively equal throughout the house, and that most people do not use kerosene heaters in a room closed off from the remainder of the ouse.

The difference in polluta t output observed in the CPSC laboratory between convective and radiant heaters was also confirmed by the whole house data. Convective heater resulted in nitrogen dioxide concentrations (0.11 ppm) grea er than radiant heaters (0.03 ppm). Monitoring in ninety-five home with one kerosene heater found that the indoor air in 84 percent of th m did not exceed 0.25 ppm nitrogen dioxide, the concentration tha the staff believes to be the level where

adverse health effects would occur. The maximum concentration, adjusted for hours of use, was 0.85 ppm nitrogen dioxide in a home with a convective heater. In contrast, for radiant heaters, the maximum concentration of nitrogen dioxide, adjusted for hours of use, was 0.12 ppm.

Preliminary analysis of the health effects data from the Pierce Study indicate an association between acute respiratory illness and the use of kerosene heaters. The population studied consisted of 174 families with kerosene heaters and 173 families without kerosene heaters. These families had, respectively, 59 children under the age of 13, and 62 children under the age of 13. The test and control families had the air in their homes monitored for nitrogen dioxide concentrations and were interviewed bi-weekly from January through March, 1983. Monitoring data demonstrated that children in homes with kerosene heaters could experience average long-term exposures to nitrogen dioxide three to four times as high as those of children in non-heater homes (0.025 vs. 0.006 ppm). Exposed children had significantly more days of illness than non-exposed children.

Average hours of heater use showed a significant positive association to days of illness ($p<0.05$) when controlling for variables such as the number of cigarettes smoked in the home, type of cooking fuel, age and sex. Further, a history of prior respiratory illness also showed a positive association to respiratory illness during the study period ($p<0.05$) suggesting an increased sensitivity to NO_2 associated respiratory illnesses for those with a history of such illness. Age on the other hand showed an inverse association to days of respiratory illness suggesting an increased resistance to NO_2 induced respiratory illness for older children ($p<0.05$). These findings are consistent with previous epidemiologic studies, but it should be noted that this was a small study with several factors which introduce uncertainties into the analysis of the data.

CONCLUSIONS

Testing of new technology kerosene heaters has demonstrated a substantial decrease in nitrogen dioxide and carbon monoxide emissions. The new dual combustion chamber heaters based on the very limited data available are not likely to cause adverse health effects under normal use conditions. Testing of add-on catalytic devices demonstrate that they are effective in reducing pollutant emissions from the kerosene heaters that were the subject of our June 1983 briefing package.

A thorough review of the new health effects studies found them to be consistent with the earlier literature. The staff conclusions remain the same as those presented in June, 1983; that nitrogen dioxide levels in the home should not exceed 0.25 ppm and that carbon monoxide levels should not exceed 15 ppm, averaged over eight hours, or a 25 ppm peak exposure for one hour.

The Pierce study confirmed earlier staff assumptions on the use of kerosene heaters and the exposure of individuals in the home. The Pierce study also found increases in respiratory illness among sensitive populations in kerosene heater homes in a manner anticipated by the staff and consistent with earlier epidemiologic studies.

Efforts are underway with U.L. and the kerosene heater industry to develop a standard for nitrogen dioxide emissions. Progress has been made in developing a certification test procedure. Discussions are underway to develop a consensus standard for nitrogen dioxide emission rates from kerosene heaters. The kerosene heater industry continues to be fully supportive of the appropriateness of developing an emission standard.

TAB A – HS Memo

UNITED STATES GOVERNMENT

Memorandum

U.S. CONSUMER PRODUCT SAFETY COMMISSION
WASHINGTON, D.C. 20207

NOV 23 1984

TO: Sandra Eberle, Program Manager

THROUGH: Peter W. Preuss, Associate Executive Director for Health Sciences

FROM: Warren K. Porter, Jr., Director, HSHL

SUBJECT: Status Report, Kerosene Heaters

SUMMARY:

Three areas of the kerosene heater study have received further review and investigation. These are further analyses of the Pierce Foundation Study; CPSC testing of additional kerosene heaters, both new technology and use of catalytic add-on devices; and voluntary standards development activities with Underwriter Laboratories, Inc. These additional activities discussed in the following sections in more detail, have shown some progress in reduction of emission of pollutants. The Pierce Foundation Study of occupied residences with and without kerosene heaters studied exposure levels of nitrogen dioxide, a pulmonary irritant. In addition the study attempted to determine if the observed exposures to NO_2 resulted in increased respiratory illness. The study indicated the following:

- Heaters were used a median time of 4.5 hours per day,
- Heaters were generally used with the heater room open to the remainder of the house,
- Convective heaters resulted in higher concentrations of nitrogen dioxide (0.11 ppm) than radiant heaters (0.03 ppm).
- Of 95 homes with one kerosene heater, 84% did not exceed the 0.25 ppm concentration the staff believes to be the level where adverse health effects would occur; however, in one house the maximum concentration adjusted for hours of use was 0.85 ppm.
- Preliminary analyses of the health effects associated with kerosene heater use indicate an association between kerosene heater use and increased incidence of respiratory illnesses. The association was strongest in children, and those people with a past history of respiratory illnesses. Symptoms decreased with increasing age. It should be noted that the population studied was volunteers as opposed to randomly selected residences and the population was small (121 children in 347 homes). Further, a great deal of publicity surrounded kerosene heater safety during the study period. These factors introduce uncertainties in the assessment of health effects but the association of NO_2 and adverse health effects found by the Pierce Foundation is consistent with previous epidemiological studies.

The CPSC heater testing program followed the basic protocol used during the 1982-1983 testing study. The heaters tested consisted of two new technology heaters using double combustion chambers and evaluation of the effectiveness of three types of catalytic devices intended to reduce emissions of carbon monoxide and odors.

The current testing has demonstrated the following:

- The new technology heaters, both convective and radiant, produce nitrogen dioxide at about 20 to 25% of the emission rates determined for old technology blue flame and white flame heaters,

- The new technology heaters resulted in carbon monoxide emissions being reduced to 10% of the emission rates determined for old style blue flame heaters and to about 50% of the emission rates determined for old style white flame heaters,

- Preliminary testing of catalytic devices indicate nitrogen dioxide production is about 50% of the emission rates observed for similar blue or white flame heaters while carbon monoxide production is 90% of that determined for blue flame radiant heaters and about 50% of the emission rates determined for similar white flame convective heaters.

The CPSC staff, Underwriters Laboratories (U.L.), the kerosene heater industry and academia representing consumers have been meeting to develop a concensus standard for U.L. certification programs addressing pollutant emissions from kerosene heaters. To date the discussions have been directed toward identifying instrumentation and testing protocols for determining emission rates of nitrogen dioxide carbon monoxide, nitric oxide and carbon monoxide. The following activities are underway:

- U.L. has capability for calculating emission rates of carbon monoxide and carbon dioxide,

- U.L. has ordered and is awaiting delivery of the nitrogen dioxide and nitric oxide measuring instruments,

- Comparison tests of various methodologies are planned to determine if small scale test devices duplicate emission rates determined in large laboratory chambers or residences.

Pierce Foundation Health and Heating Survey

A portion of this study, funded by the National Institute of Environmental Health Sciences, studied health effects associated with kerosene heater use. CPSC provided funding for the costs associated with the performance of a field study of pollutant concentrations observed in occupied dwellings in the New Haven area. The results of the monitoring were discussed in the June 1983 Kerosene Heater briefing package. Nitrogen dioxide (NO_2) was the most thoroughly investigated pollutant since inexpensive passive monitors were available. The concentration of NO_2 in 155 homes with kerosene heaters and 148 homes without kerosene heaters were measured over six two week periods from January through March. During this period heater use ranged from two hours per day to 24 hours per day with a median use of 4.5 hours per day. Two week average concentrations of NO_2 for the various homes monitored are presented in Table 1. Also presented in Table 1 are the NO_2 concentrations adjusted for hours of use of the kerosene heater. The adjusted value is believed to represent an estimate of average concentration during the period the heater was in use but does not estimate peak or steady state concentrations reached during use. Further controlled laboratory studies are necessary to define the usefulness of assessing steady state or peak exposure concentrations from passive monitors.

Table 1 NO_2 Concentrations measured over six two-week periods

Type of Use	NO_2 Concentration		
	n	Two Week Average	Average During Heater Use*
		(ppm)	(ppm)
No Heater, No Stove	145	0.004	----
One Heater, No Stove	95	0.018	0.079
No Heater, One Stove	42	0.017	----
One Heater, One Stove	18	0.033	----
Two Heaters, No Stove	13	0.035	----
Two Heaters, One Stove	3	0.043	----

*Adjustment was made only for those homes with one heater and no stove in order to avoid errors associated with the presence of multiple NO_2 sources. The range of adjusted concentrations was from about 0.005 ppm to 1.0 ppm.

The distribution of NO_2 concentrations throughout different areas of the houses was nearly equal; this was observed both for control houses, and for houses with kerosene heaters. This fact indicates that the hot air from the heater disperses rapidly throughout the house, confirming the staff's assumption in the 1983 briefing package that for purposes of mathematical modeling the house could be treated as a single large room when interior doors remain open. Further, these data

substantiate the finding of the kerosene heater industry's user survey that most people do not use the heaters in closed rooms.

No clear trend was observed between NO_2 concentrations and rated heat output of the heaters. This is not surprising, since the size of the heaters purchased were either scaled to the size of the room to be heated or the heaters, if too large for the room, were probably periodically turned on and off in order to avoid over-heating the particular room in which the heater was located. There was, however, a distinct difference associated with NO_2 concentrations and use of radiant or convective heaters, the convective heaters producing higher concentrations of NO_2. The maximum NO_2 concentration, adjusted for hours of use, for radiant heaters was about 0.12 ppm (the average of 20 observations was about 0.03 ppm). In contrast, convective heaters produced maximum concentrations, adjusted for hours of use, of about 0.85 ppm (the average of 75 observations was about 0.11 ppm). Although the average concentrations of NO_2 produced by convective heaters is above the 0.05 ppm National Ambient Air Quality Standard, it is not above the level (0.25 ppm) where the staff believes adverse health effects would result for normal healthy adults. It should be noted, however, that about 20% of the NO_2 concentrations that were observed with the use of convective kerosene heaters were greater than 0.25 ppm.

Respiratory Illness

This phase of the Pierce Foundation's Study on Health and Heating followed the association of acute respiratory illness and the use of

kerosene space heaters. The population studied consisted of 174 families with 59 children under the age of 13, in homes with kerosene heaters, and 62 children under 13 years of age in 173 homes without kerosene heaters. Of the total of 347 families about 87% of the households had the air monitored for at least one two-week period for NO_2. The test and control families were followed by telephone interview once every two weeks from January through March 1983. The telephone interview assessed heater use and respiratory symptoms for the oldest female, and if present, the child less than thirteen years old and closest to five years of age. The mean age of the children was 6.8 years in a mean household of 4.2 people. The average and ranges of NO_2 concentrations in the heater and non-heater homes are presented in Table 3.

Table 3. Two week average NO_2 concentrations in ppm (range) for test and control homes

Class	Sampler Location			
	Outdoor	Kitchen	Living Room	Bedroom
Heater House*	0.008 (0.002-0.023)	0.026 (0.002-0.112)	0.025 (0.002-0.082)	0.025 (0.002-0.119)
Non-heater	0.007 (0.0-0.014)	0.007 (0.0-0.042)	0.006 (0.0-0.034)	0.006 (0.0-0.034)

* Average heater use was 7.7 hours per day (range 0 to 24 hours per day).

It is seen from these data that children in homes with heaters could experience NO_2 exposures of three to four times those experienced in non-heater homes while they were present in the homes.

A statistical analysis of these data indicates that exposed children had significantly more days of illness than non-exposed children ($p<0.05$). Average hours of heater use showed a significant positive association to days of illness ($p<0.05$) when controlling for variables such as the number of cigarettes smoked in the home, type of cooking fuel, age and sex. Further, a history of prior respiratory illness also showed a positive association to respiratory illness during the study period ($p<0.05$) suggesting an increased sensitivity to NO_2 associated respiratory illnesses for those with a history of such illness. Age on the other hand showed an inverse association to days of respiratory illness suggesting an increased resistance to NO_2 induced respiratory illness for older children ($p<0.05$).

These results are preliminary but are consistent with epidemiologic studies reviewed in the 1983 Kerosene Heater Briefing Package. It should be noted that the study population was small and potential uncertainties include use of a convenience population sample and the large amount of publicity surrounding the safety of kerosene heaters during the time that the study was performed.

Kerosene heater testing program -- CPSC

The testing of kerosene heaters has continued at the CPSC. The testing protocol adopted was the same as that used during the 1983 testing phase of the kerosene heater investigation. Heaters were fueled with 1K kerosene, burned for a minimum of 20 hours prior to testing and subsequently tested in an exposure chamber for a burn time of two hours or until the chamber temperature reached 85° F, which-ever occurred

combustion. The coolant, however, was not allowed to fall below the dew point temperature in order to ensure that water soluble pollutants would not be lost. The air exchange rate was maintained at about 1.5 air changes per hour. The pollutants were monitored continuously and recorded at one minute intervals. The data, stored on magnetic disks, was transmitted to the Parklawn Computer Facility where it underwent reduction to concentration vs time formatting. The pollutants monitored were carbon monoxide (CO), nitric oxide (NO), nitrogen dioxide (NO_2), nitrogen oxides (NO_x), carbon dioxide (CO_2), and oxygen depletion.

The heaters which were tested were two new technology heaters which use a double combustion chamber design which is intended to emit a minimum amount of nitrogen oxides and carbon monoxide as well as old technology heaters which had catalytic converters added to them. Both radiant blue flame heaters which typically produce higher concentrations of CO and lower concentrations of NO_2, and convective heaters were tested. The new technology heaters represented both a radiant and convective type; however it should be noted that both had dual combustion chambers of similar operating principles.

In general, the dual combustion chamber design resulted in a reduction of NO_2 to about 20 to 25% of the emission rates observed for blue flame radiant and white flame convective heaters during the 1983 testing program. These heaters also resulted in a reduction of 90% in the CO emission rates when compared to blue flame radiant heaters tested in 1983 and a reduction of at least 50% when compared to white flame convective heaters tested in the 1983 program. The catalytic add-on

devices have not been fully tested at this time; however, preliminary data indicate that as a class they result in NO_2 emission rates of about 50 to 60 percent of those observed in the 1983 testing program while CO emission rates are 90% of those determined for blue flame heaters and about 50% of the emission rates determined for white flame heaters. A summary of these data is presented in Table 2.

Table 2. Emission Rates for Kerosene Heaters 1984

Heater Type	Average Emission Rate (cc/kJ)		
	1983 Data	1984 Data	
		Catalysts (3 types)	Dual Chamber (2 Heaters)
Radiant and conventive blue flame			
NO_2	0.0025	0.0013 (range 0.001 to 0.002)	0.0004
CO	0.11	0.09 (range 0.001 to 0.014)	0.01
Conventive White flame and wickless			
NO_2	0.0038	0.0015 (range 0.001 to 0.002)	0.0008
CO	0.02	0.01 (range 0.009 to 0.01	0.01

These data and future data obtained when the current testing program is completed will be used in preparing an exposure assessment. Several observations of a general nature, however, can be made at this time.

- The emission rates observed with the two chamber combustion heaters are low enough for CO that it is unlikely that the predicted CO concentrations in a room would exceed eight parts per million, even under closed room conditions.

- The emission rates for NO_2 for the dual combustion chamber design are low enough that the predicted NO_2 concentrations would be unlikely to exceed 0.1 ppm even in a closed room.

- The catalytic add-on devices appear to lower the emissions of NO_2 to about ½ their original levels. Thus, in most instances, they would be effective in allowing heater use in semi-open rooms. However, in some closed room situations the heaters would still result in NO_2 concentrations in excess of the 0.25 ppm recommended by the staff.

- The catalytic add-on devices appear to lower CO emissions by about 10-60%, thus the heaters could probably be used in most open and semi-open room situations. However, with high emitting heaters, the catalytic add-on devices may not be effective enough to allow use of all heaters in closed room situations.

Voluntary Standards Activity

The staff of the CPSC, Underwriters Laboratories (UL), the kerosene heater industry and academia representing consumers have been in the process of developing a consensus standard for use in certification programs for kerosene heaters. These minimum standards would ensure that under proper use most of the population could use the heaters with a suitable degree of safety.

Three meetings of the voluntary standards work group have been held

since June 1983. The primary concern of the group to date has been addressing the issue of test methodology for measurement of emission rates of various pollutants. The pollutants under consideration are carbon monoxide (CO), nitric oxide (NO), nitrogen dioxide (NO_2) and for technical reasons, i.e. data reduction to emission rates, carbon dioxide, nitrogen oxides (NO_x) and oxygen depletion.

The basic instrumentation for measuring pollutant levels in air samples are well established. A variety of well established methods are available for the determination of nitrogen compounds and new technologies are evolving; however, the particular choice of instrument must be matched to the physical test system designed for the certification testing. At present UL is in the process of purchasing a chemiluminescent nitrogen oxides detector. The instrument considered should have adequate sensitivity and flexibility to permit monitoring either by use of chambers, hoods or probes with a minimum of interference from water and carbon dioxide. When the instrument is in place a comparison between various sampling strategies will be undertaken (chamber, hood and probe). The goal of the testing will be to determine if chamber derived emission rates are the same as hood or probe derived emission rates. Presently, the staff believes that chamber derived emission rates are most applicable for modeling the concentrations to be expected in homes where heaters are used. This belief is based on the work of the Lawrence Berkeley Laboratories and the Pierce Foundation studies presented in the 1983 briefing. One comparison of a hood derived emission rate to a monitored home was provided by the work of Weston Associates and was discussed in the 1983

briefing package. It should be noted, however, that although the hood derived emission rate obtained by Weston agreed with the staff's chamber derived emission rate and also allowed prediction of the concentrations of pollutants in the test home, the Weston hood test was performed in the test home which unlike a laboratory had significant levels of NO, NO_2,CO and CO_2 present. This fact may be important in that the contaminated combustion air present n the home may have affected the emission rates. Had the hood tests been performed in a laboratory where the combustion air would have been relatively free of pollutants, the emission rates may have been different. Thus, comparative studies of hood, probe and chamber derived emission rates must be performed in order to ensure development of both reproducible and accurate test data as well as technically sound recommendations on tolerable pollutant emission rates.

In addition to the activities at UL additional comparisons of emission rates derived from chamber and test stand methods are being undertaken by the U.S. Environmental Protection Agency (EPA). These studies will compare chamber to test stand derived emission rates for unvented gas fired space heaters and for kerosene heaters. The major goal of the EPA study is directed toward particulates and the presence of polyaromatic hydrocarbons on the particulates. Preliminary data from this program should be available in 1985.

Health Effects Updates

Since the 1983 briefing 15 new articles on the health effects of nitrogen dioxide on humans and animals have been found. An update of

the health effects presented in the 1983 briefing is attached. The new literature, at least half of which was published since 1982, generally supports the staff recommendation of controlling NO_2 emissions to prevent concentrations in homes from exceeding 0.25 ppm for the healthy population. The newer literature is consistent with previous epidemiologic studies associating NO_2 exposure with respiratory illness. Further, more sensitive techniques for assessing lung and tissue damage following exposure to low concentrations of NO_2 are described. Additional research, however, is necessary in order to determine if the observed effects are permanent or transient in nature.

An update of carbon monoxide (CO) health effects is attached. Literature published since the 1983 briefing has not led the staff to change its original recommendation that exposures should not exceed an eight hour average of 15 ppm or a one hour peak exposure of 25 ppm.

NITROGEN DIOXIDE HEALTH ASSESSMENT, 1984

INTRODUCTION

In 1983* the Health Sciences staff completed an in-depth review of data on the health effects associated with exposure to nitrogen dioxide. The purpose of this document is to update the 1983 staff review.

BACKGROUND/SUMMARY*

In 1983 the Health Sciences staff concluded that there was sufficient evidence from animal studies to show that exposure to nitrogen dioxide can result in adverse health effects. The results from morphologic and biochemical studies show that following exposure to more than 1.0 ppm NO_2 there is a sequence of reactions in lung tissue which includes: cell death, and then cellular renewal. New cells, exposed to the same concentration of NO_2 are more resistant to damage. Other data indicate that the reason for this resistance is an increase in the amount of reduced glutathione which protects the tissue from the oxidative effect of nitrogen dioxide.

Animals exposed to NO_2 have a significant decrease in their resistance to respiratory infection. There appears to be a relationship between length of exposure, continuous or intermittent, and increased mortality from bacterial infection. Mice continuously exposed for 14 days to 0.05 ppm NO_2 (plus a one hour/day spike of 0.1 ppm) have a 6.6%

*1983 Kerosene Heater Briefing Package. The background/summary is a summation of the health assessment made by the Health Sciences staff. References can be found in the 1983 Kerosene Heater Briefing Package.

increase in mortality following exposure to bacteria. Mice continuously exposed to 0.5 ppm NO_2 for 60 days have a 10% increase in mortality following exposure to bacteria. The mortality incidence increases to 20% if the exposure to 0.5 ppm NO_2 is for 90 days. These findings have suggested to some investigators that humans exposed to nitrogen dioxide might also result in an increased morbidity.

There are a limited number of studies which have attempted to relate either chronic exposure to NO_2 in homes and occupational settings or known exposure studies in laboratories with adverse health effects in humans. These studies have two limitations, lack of long term follow-up to assess the risk of irreversible emphysema-like lung damage and the inability to distinguish the effect of NO_2 from other pollutants in epidemiologic studies. Despite the limitations of the various studies, the data collected to date suggest that individuals who are at special risk, such as asthmatics, might be adversely affected by exposure to low levels of NO_2 for short periods of time.

There is one study in which investigators found no significant clinical symptoms of emphysema in a group of 70 men exposed to 0.4 - 2.7 ppm NO_2 for 6-8 hours/day for 4-6 years. However, they did find a significant increase in hydroxyproline* in the urine of the exposed men as compared to controls. This observation is of particular interest because it can be related to studies in which guinea pigs exposed to 1.1 ppm NO_2 for 8 hours/day had an increased urinary excretion of hydroxyproline and developed emphysema. In addition, dogs exposed continuously to about 0.14 ppm NO_2 for 68 months and then allowed to breathe unpolluted air had progressive changes in lung function

*The major amino acid found in collagen and elastin.

beginning at 24 months post exposure and emphysema-like lesions at autopsy.

The available epidemiologic studies on exposure to NO_2 from the use of gas stoves and related health effects indicates a trend toward increased respiratory illness associated with the use of gas appliances. None of the studies, however, show a definitive relationship between exposure to NO_2 and respiratory illness.

The EPA Clean Air Science Advisory Committee (CASAC) in 1982 also reviewed the epidemiological studies and concluded that the identified studies "do not provide quantitative evidence of identifiable public health effects linked to specific ambient air concentrations of NO_2.... The CASAC concurs with the 'EPA' staff paper recommendation that an annual primary standard within the range 0.05 - 0.08 ppm will offer sufficient protection for the various welfare effects of concern."

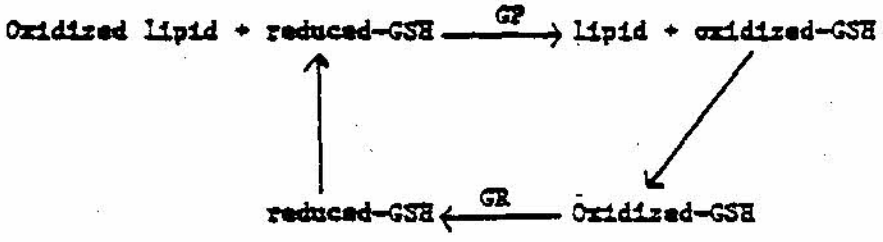
The current report will review all health studies associated with chronic or acute exposure to less than 1.0 ppm nitrogen dioxide which have been published since the 1983 Health Sciences review. In addition, this health assessment will include a review of the systemic health effects attributed to exposure to NO_2 which have not been previously discussed.

EFFECT OF NITROGEN DIOXIDE ON THE LUNG

EFFECT OF AGE

Biochemical

Because NO_2 is an oxidant, a variety of studies have been conducted to assess how the lung responds to oxidant stress. Two pulmonary antioxidant defense mechanisms, superoxide dismutase and the glutathione system, have been postulated to offer varying degrees of protection against oxidant-induced lung injury. The enzyme superoxide dismutase catalyzes the dismutation of the superoxide radical (O_2^-) to hydrogen peroxide (H_2O_2). The enzyme catalase then converts the hydrogen peroxide to water and oxygen ($2H_2O_2 \longrightarrow 2H_2O + O_2$). The glutathione system is thought to limit the lipid peroxidation initiated by the superoxide radical by the mechanisms shown in the equations below.



GSH = glutathione; GP = glutathione peroxidase; and
GR = glutathione reductase

Yam et al (1978) measured the activity of superoxide dismutase (SOD), catalase (CA), glutathione peroxidase (GP), glutathione reductase

(GR), and reduced glutathione (GSH) in lung tissue from Sprague-Dawley rats of various ages (2 days premature to 70 days old). No significant differences were found in the various enzyme activities or GSH level between male and female rats. The pulmonary SOD activity and GSH level of the 70-day-old rats were approximately 200%* of that found in the neonatal rats (0-21 days old). Similarly, the GP and GR activities showed a progressive increase with age from day 12 to 70 (at day 70 the GP activity increased to 200% and the GR activity increased to 150% of that observed in the neonate). Conversely, the catalase (CA) activity increased dramatically during the first 10 days of life (increase of about 100%) and then began a steady decline to 50% of that found in the neonatal rat lung.

The authors conclude, based on their data and that of others, that the ability of an animal to tolerate oxidant induced lung injury is governed by a delicate balance between the generation of cytotoxic agents and the capabilities of the various pulmonary defense mechanisms. Furthermore, the ability of the lung to offset oxidant challenge will be determined not only by the total activities of antioxidant enzymes but by the capability of the lung to increase its level of antioxidant enzymes in response to oxidants and the proximity of the protective systems to the site of cytotoxicity. Lastly, the authors point out that

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*Yam et al (1978) give no statistical data, however, the data in the figures indicate that the values are highly significant.

the decline in CA activity in lung tissue in the adult rat may be extremely critical and further contributs to the susceptibility of the lungs to oxidant-induced damage.

Tyson et al (1982) exposed Sprague-Dawley rats of various ages (5 to 180 days old) to 14 ppm NO_2 for 72 hours. At the end of the exposure period, the animals were sacrificed and the glucose-6-phosphate dehydrogenase, glulathione reductase, and glutathione peroxidase activity (collectively known as the GSH - shuttle enzymes) in whole lung tissue were determined. The 72 hour exposure period was selected because it was believed that the animals would be in the adaptive recovery phase* following NO_2 exposure. The values obtained are summarized in Table I. The data show that post-natal and neonatal (0-20 days old) animals do not show significant increases of monitored enzymes following an acute exposure to a high concentration of nitrogen dioxide (14 ppm). The weanling (20-40 days old) and young adult (40-180 days old) have significant increases in lung tissue GSH-shuttle enzymes. Furthermore, the data suggest that the increase in enzyme activity may be age related since the change in young adults, greater than 60 days old, is greater than that seen in weanling rats. The study also included an additional group of animals (13 days old at sacrifice) which were exposed to 50 ppm nitrogen dioxide. This high concentration of NO_2 resulted in a significant elevation in the activity of the GSH-shuttle enzymes in the lungs of these rats.

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*Studies from a number of laboratories have shown that the lung goes through an adaptive process following exposure to NO_2. Initially there is cell death which peaks at 24 hours, followed by cellular renewal which peaks at 48 hours plus an increase in the activity of various protective enzymes.

Table I EFFECT OF AGE ON CHANGES IN ENZYME ACTIVITY IN RAT LUNG FOLLOWING 72 HOUR EXPOSURE TO 14PPM NO_2

Age at Sacrifice		G6PD*	GR*	GP*
8 days		+12	0	-9
13		+18	+15	+2
18		0	+ 7	+3**
28		+166**	+85**	+79**
38[1]		+146**	+77**	+65**
60-90[1]	c	+377**	+187**	+155**
90[2]	c	+343**	+158**	+83**
180[2]		+415**	+198**	+150**
180[1]		+301**	+113**	+113**
180[1]	c	+151	+71	+33

Values are % difference from control (IU/lung). Each value is a comparison of more than 5 animals in each group (control vs exposed) unless otherwise indicated (c = less than 5 animals/group).
*G6PD = glucose-6-phosphate, GR = glutathione reductase, GP = glutathione peroxidase. 1 = nursing dams, 2 = males only.
**$P<.05$ or greater. Adapted from Tyson et al (1982).

The authors concluded that the most consistent findings were that neonates showed the least response to oxidant stress. They pointed out that the unresponsiveness in the exposed neonate lungs was probably not due to differences in the time course of injury and the proliferative phases resulting from it because, when the level of NO_2 was increased to 50 ppm, the activity of the GSH-shuttle enzymes was significantly increased. Tyson et al (1982) also point out that the difference in response can not be explained by higher basal levels of enzymes since studies from a number of laboratories have shown the activity to be similar in lung tissue from neonatal and adult animals. The reason(s) for the observed difference is yet to be elucidated.

Morphological

A number of studies clearly show that lung bronchiolar ciliated cells and alveolar Type I cells are damaged by 2 ppm or more nitrogen dioxide (see CPSC Kerosene heater 1983). When Type I cells are damaged they are replaced by Type II cells which divide into a Type I cell plus a Type II cell. There is also an overall hyperplasia of Type II cells in response to damage by nitrogen dioxide.

Recently, Sherwin and Richters (1982) have attempted to quantitate Type II hyperplasia of the lung following exposure to 0.34 ppm nitrogen dioxide. These investigators have developed a new technique based on the fact that Type II cells have relatively more lactate dehydrogenase (LDH) activity than other cells of the lung. They stained the tissue for LDH activity and then counted the number of Type II cells on the histological slide (using a method called quantitative image analysis). Sixty Swiss Webster male mice were exposed to 0.34 ppm NO_2 for 6 hours/day, 5 days/week, for 6 weeks, and sixty control animals were exposed to filtered air. For each animal eight slides were prepared each from the left lung, the right upper lobe, and the left lower lobe (total of 24 slides/animal). Each slide was divided into four quadrants for analysis. Thus, there were 96 image analyses/animal and a total of 11,520 analyses in the experiment. The results of the study show a small (less than 10%) but highly significant increase in the number of Type II cells in the lungs of the animals exposed to nitrogen dioxide. Thus, it would appear that intermittent exposure to low levels (0.34 ppm) of nitrogen dioxide can result in detectable

indication of lung damage. At present, however, these studies do not allow for an assessment which would indicate if the damage detected is transitory or reflects the beginning of an irreversible process.

Cabral-Anderson et al (1977) and Evans et al (1977) have reported on the effects of high concentrations of NO_2 on the lungs of aging rats. Groups of 27 HLS-SD rats of various ages (one to 25 months old) were exposed to different concentrations of nitrogen dioxide. The NO_2 concentration was decreased for older animals to prevent excessive deaths. The incidence of mortality related to acute exposure to NO_2 is summarized in Table II.

TABLE II EFFECT OF ACUTE NO_2 EXPOSURE ON AGING RATS

Age Month	NO_2 Exposure ppm	Comments
1	18	No deaths
3	17	No deaths
11	14	2 deaths after 48 hrs.
19	14	12 deaths after 48 hrs. 3 deaths after 72 hrs.
25	12	2 deaths after 48 hrs.

There were 27 rats in each group. Because of excessive mortality in the 19 month old group additional animals were added. Cabral-Anderson et al (1977)

During the exposure to NO_2 groups of three rats were sacrificed at 8 hours, 12 hours, and on day 1, 2, 3, 4, 5, 10 and 15 and lung tissue was qualitatively examined by light and electron microscopy for injury. Comparison of the morphological response to NO_2 among different aged rats revealed no differences in the types of cells injured, the manner of cell injury, nor the capacity for repair of injured tissue. There were larger amounts of tissue damage in the alveolar region of the lungs

The data show that peak turnover occurred on day 3 in the 11, 19 and 25 month old animals. The data are summarized in Figure I.

These data support the morphologic observation that tissue repair in one month old animals is more rapid than that in the 11, 19 and 25 month old animals. Once the initial tissue repair was complete there were no apparent signs of residual damage. The authors did note, however, that the ^{3}H-thymidine incorporation into cells was still elevated compared to controls. This suggested to the authors that a damage/repair process might be functioning but could not be defined morphologically.

Kyono and Kawai (1982) also studied the relationship between age and morphological changes associated with exposure to nitrogen dioxide. They exposed groups of female JCL-SD strain (SPF) rats (7 animals/group) to 0.1, 0.5, 3.0 or 10 ppm nitrogen dioxide continuously for one month. The rats were 1, 3, 12 or 21 months-old at the onset of the exposure period. The responses of the lung to NO_2 exposure showed age-related differences. Kyono and Kawai (1982), using electron microscopic morphometry, found a trend toward a dose-dependent increase in the arithmetic mean thickness of the air-blood barrier in all age groups examined. Based on the morphometric index, the response tends to decline from one to 12 months of age but then increases as the animals age. Some of the morphometric data obtained by Kyono and Kawai (1982), are summarized in Figure II. The most noticable changes in lung morphology due to exposure to low levels (0.1 and 0.5 ppm) of NO_2 are observed at one and three months of age. Significant changes (see Figure II) are found in the arithmetic mean thickness of the air-blood barrier (AMT), volume density of the alveolar wall tissue (VdAWT), and surface area (Sa). At 12 months of age significant differences are

of older animals but not in the bronchiolar epithelium. There was less tissue damage in the 25-month old animals than in the 11 and 19 month old animals. The authors suggest that this difference may have resulted from decreased exposure concentration. The time to onset of tissue repair was 48 hours in the 1 and 3 month old rats whereas in the 11, 19 and 25 month old rats repair was observed on the third day. After 10 days of exposure to NO_2 there was no evidence of additional injury in any age group. The authors concluded from these observations that alveolar cells are more sensitive to injury by NO_2 in aging than in young rats.

In a second experiment, using exactly the same conditions the effect of NO_2 on cell turnover in lung tissue was determined by injecting groups of three rats at each time period with ^{3}H-thymidine*.

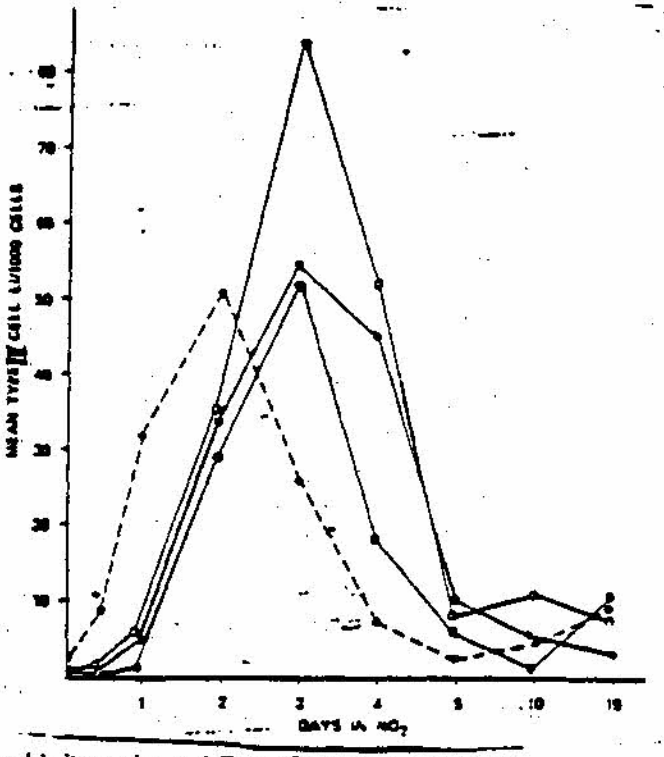


FIG. 1. Mean labeling indexes of Type II cells from rats of different ages exposed to NO_2: 1 month old (●——●); 11 months old (●——●); 19 months old (○——○); 25 months old (⊙——⊙).

(Evans et al, 1977)

* In these experiments incorporation of ^{3}H-thymidine into nuclear DNA is used as a marker for the synthesis of new DNA and thus cell division.

FIGURE II MORPHOLOGIC CHANGES IN LUNG TISSUE IN ANIMALS EXPOSED TO NITROGEN DIOXIDE.

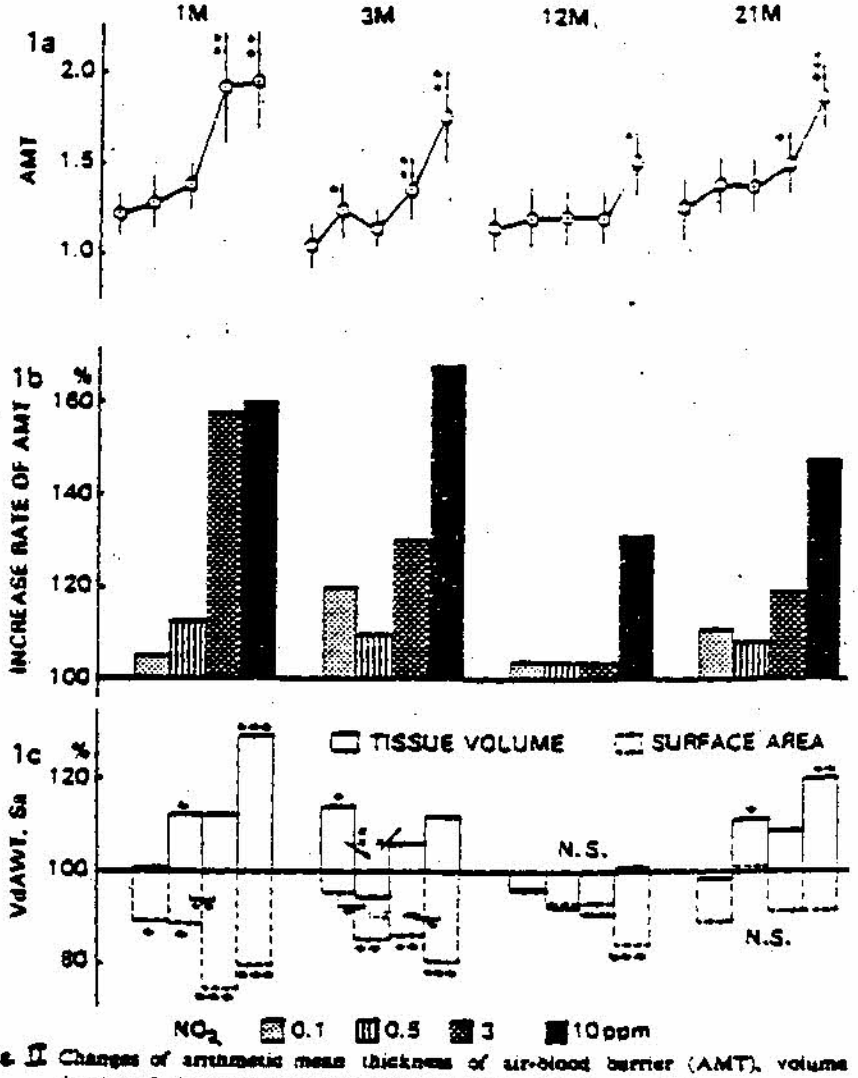


Fig. II Changes of arithmetic mean thickness of air-blood barrier (AMT), volume density of alveolar wall tissue (VdAWT) and surface area (Sa).
1a: Changes of AMT of each age group. Bar indicates 95% confidence interval of the mean.
1b: Increase of AMT, percentage of exposed against the control value.
1c: Changes of VdAWT and Sa.
t-test: * $p<0.05$ ** $p<0.01$ *** $p<0.001$ p value control versus experimental.
χ^2-test: ' $p<0.05$ '' $p<0.01$ ''' $p<0.001$ —/, compared to indicated dose groups
N.S.: non-significant.
All the values are expressed as percentage of exposed groups to controls of the same age.

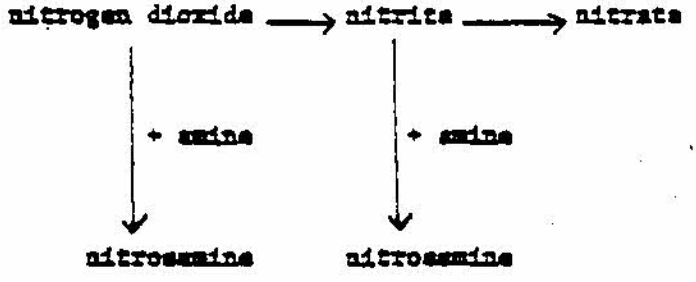
Kyono and Kawai (1982)

found only after exposure to 10 ppm nitrogen dioxide, and at 21 months of age the profile of morphologic change has a tendency to look like that of a young animal.

NITROGEN DIOXIDE METABOLISM

Recent reports have demonstrated a role for inhaled nitrogen dioxide in the biosynthesis of nitrosamines, which are a group of well established carcinogens * (National Academy of Sciences, 1981). N-nitrosamines are formed by the reaction of an amine or an amine derivative with a nitrosating agent. Nitrogen dioxide has been implicated as a nitrosating agent in the formation of N-nitroso compounds (National Academy of Sciences, 1981).

Nitrogen dioxide can react with tissues to form nitrate. This reaction occurs through a nitrite intermediate. The nitrite intermediate may then react with amines to also form nitrosamines.



*1983 Briefing Package on Nitrosamines in Pacifiers, Ongoing Investigation and Proposed Voluntary Standard.

There are few reports in the literature which demonstrate that animals metabolize nitrogen dioxide. For example, rabbits exposed to a high concentration of NO_2 had elevated levels of both nitrate and nitrite (a nitrosating agent) in the blood and urine (Svorcova and Kaut, 1971). Oda et al (1981) also found increased levels of nitrate and nitrite in the blood of mice exposed to nitrogen dioxide. The half-life for blood nitrate in the mouse was about one hour and the half-life for blood nitrite was a few minutes. The short half-life of the nitrite has been attributed to its reaction with oxyhemoglobin to form methemoglobin and nitrate (Goldstein et al 1977).

Igbal et al (1980) gave ICR mice (3 to 4 males/group) 2 mg of morpholine (an amine) by gavage. The animals were then exposed to 50 ppm NO_2 for one to four hours, sacrificed and the amount of nitrosomorpholine synthesized (total amount in the whole animal) determined. The data obtained indicated that the conversion of morpholine to nitrosomorpholine was linear over the time period studied. At four hours the amount of morpholine converted to nitrosomorpholine was 2230±138.6 ng per mouse. In a second experiment Igbal et al (1980) determined the amount of nitrosomorpholine synthesized as a function of NO_2 concentration. They studied the effect of 0.2 to 50 ppm NO_2 on the conversion of morpholine to nitrosomorpholine and found that the conversion was linear at exposures from 15 to 50 ppm. Conversion at 0.2 ppm NO_2 was less than half that expected. Igbal et al (1980) concluded from their data that mice biosynthesize nitrosomorpholine from NO_2 and morpholine.

Mirvish et al (1981) repeated the studies of Igbal et al (1980), however, they came to the conclusion that no significant

N-nitrosomorpholine was formed in rat stomach contents or blood or the whole mouse after exposure to morpholine (gavage) and nitrogen dioxide. This was because Mirvish et al (1981) were able to demonstrate that the method used by Iqbal et al (1980) to inhibit *in vitro* nitrosations was not effective, and thus gave false positive results. Mirvish et al (1981) did, however, observe that tissues from mice exposed to 23 ppm NO_2 for five hours produced a nitrosating agent *in vivo* that could nitrosate morpholine *in vitro* (Table III). Attempts at identifying the nitrosating agent were not successful (no data given).

TABLE III NITROSATING AGENT IN MOUSE TISSUE

Organ	N-NOR* ng/g tissue
Skin	70
Liver	28
Lung	63
Carcass	67

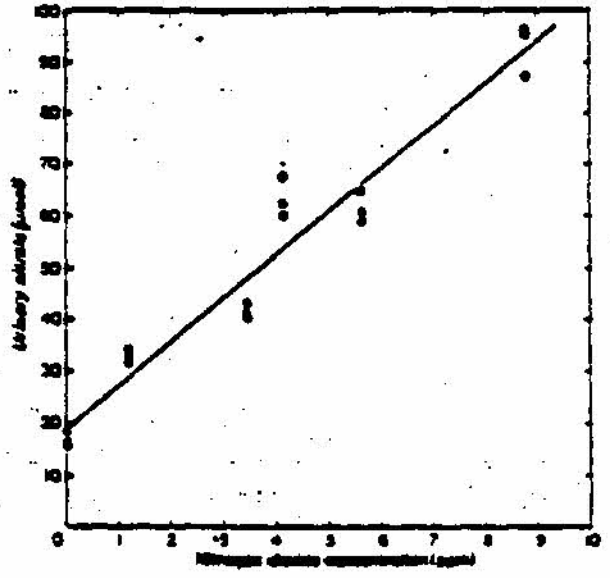
Tissue homogenates were incubated with 10 ng morpholine.
*NMOR = N-nitrosomorpholine control- whole mouse homogenate + morpholine yielded 9 ng/g tissue NMOR. Mirvish et al (1981).

The authors speculated that the nitrosating agent could be important biologically since it has been reported that lung adenomas were induced in mice exposed to 1-2 ppm NO_2 plus 1.0 gram of morpholine/liter of drinking water.

Saul and Archer (1983) exposed Sprague-Dawley rats to various concentrations of NO_2 (1.2 to 8.8 ppm) for 25 hours and then measured the amount of nitrate found in the urine for the next three days. All animals were fasted on the day of exposure since it was observed that NO_2 reacts with the food to form nitrates. The data obtained indicate that there was a dose response relationship between NO_2 exposure and

urinary nitrate (see Figure III). The authors calculated that for a 185 gram rat the rate of nitrate formation was 8.4±1.1 umole nitrate/ppm NO_2/24 hr exposure.

FIGURE III URINARY NITRATE OF RATS EXPOSED TO NO_2



Saul and Archer (1983)

Saul and Archer (1983) assumed that the inhaled NO_2 reacts with readily oxidizable tissue components such as proteins, lipids, glutathione or amines to form nitrate. Assuming that all nitrite formed is absorbed into the blood; they estimated, based on the observed rate of conversion of nitrite to nitrate, that 9.6 umoles of nitrite is formed per ppm inhaled NO_2/day/rat. The authors also estimated nitrite formation in the human. Based on a direct interspecies extrapolation*, they estimated that a 68.5 kg man with a resting minute volume of 8.73

*same efficiency of uptake and metabolism

liters/minute would form 79 umoles (about 3.6 mg) nitrite in the respiratory tract/day/0.1 ppm NO_2. Support for this assumption is based on the observation that there is a linear dose response relationship between injected nitrite and excreted nitrate, and the observation of Postlethwait and Mustafa (1981) using an isolated perfused rat lung preparation to study NO_2 metabolism.

Postlethwait and Mustafa (1983) have studied the conversion of morpholine to N-nitrosomorpholine (a known carcinogen) using an updated rat lung perfusion method. The lung preparation from a six week-old pathogen-free rat was incubated with 10mM morpholine in the perfusate plus 20 ppm NO_2 in the air taken into the lungs (exposed) or only air (control) for 60 minutes. The amount of N-nitrosomorpholine was determined in the lung tissue and lung perfusate. The results are shown in Table IV.

TABLE IV FORMATION OF N-NITROSOMORPHOLINE

Condition	N-nitrosomorpholine content	
	Lung tissue ug/lung	Perfusate total
Control	2.2	2.1
Exposed	6.2	147

Postlethwait and Mustafa (1983)

In a control experiment $NaNO_2$ (a known nitrosating agent) was added only to the perfusate and 285ug N-nitrosomorpholine were produced. The authors noted that since the concentrations of N-nitrosomorpholine was 4.3ug/g in the lung tissue and 4.2ng/ml in the perfusate it was unlikely that the tissue content could be explained by nitrate formation in the perfusion solution. The authors did note that their data did not

permit an evaluation of how much N-nitrosomorpholine was produced in the tissue and how much was produced in the perfusate solution. They stated that the in the lung only 0.0004% of the 10mM added morpholine was converted to N-nitrosomorpholine, and it is unknown whether formation of N-nitrosomorpholine outside the GI tract might play a role in NO_2-related carcinogenesis.

CANCER

Richters and Kuraitis (1981) and Richters (1981) have done several experiments in an effort to determine if injury makes the lung more susceptible to cancer metastasis. To test this hypothesis they first exposed mice to low levels of NO_2 (0.4 ppm, 8hrs/day, 5 days/week for 10 weeks or 0.8 ppm, 8hrs/day, 5 days/week for 12 weeks), and then infused the animals with cancer cells and then counted the number of metastatic cancers in the lung.

Male Swiss-Webster mice exposed to 0.4 ppm NO_2 for 10 weeks (8hrs /day, 5 days/week) and then infused with melanoma cells were sacrificed 10 or 21 days post infusion and the number of melanoma nodules in the lung counted. The data obtained in this experiment are summarized in Table V. They found a statistically significant increase in melanoma nodules in the lungs of mice exposed to nitrogen dioxide.

TABLE V FREQUENCY OF MELANOMA NODULES IN LUNGS OF MICE EXPOSED TO 0.4 PPM NO_2.

Time*	Treatment (# animals)	Mean No. Melanoma Nodules/ lobe (range)
10 days	Control (5)	5.9 (0-19)
	Treated (5)	13.0 (0-56)[1]
21 days	Control (8)	3.4 (0-15)
	Treated (6)	10.3 (0-47)[2]

*Time after infusion with melanoma cells. [1] $P < 0.5$; [2] $P < 0.005$.

Richters and Kuraitis (1981)

In a second experiment C3H3L/6J mice were exposed to 0.8 ppm NO_2 (8hrs/day, 5 days/week) for 12 weeks. The animals were infused with melanoma cells following the exposure to NO_2 and then sacrificed 21 days post infusion. The data obtained on the incidence of melanoma in the lung are summarized in Table VI. Again, they found a statistically significant increase in melanoma nodules in the lungs of mice exposed to nitrogen dioxide.

In the experiments above, one group of the control animals are exposed to filtered room air. The filter system removes particulates, NO_x and ozone. These investigators also had a second control group (not shown) which was exposed to room air. The room air control group of animals had an increased number of melanoma nodules/lobe compared to the filtered air control ($P < .05$)

TABLE VI MELANOMA NODULES IN LUNG OF MICE EXPOSED TO 0.8PPM NO$_2$

Treatment Group (No of animals)	Mean No. Melanoma Nodules/Lobe (range)
Control (23)	7.4 (1-17)
Treated (24)	19 (2-63)*

*P < 0.0025 Richters and Kuraitis (1982)

Richters (1981) also did an analysis of the distribution of melanoma nodules in each lobe of the lung of C57BL/6J mice. The data are summarized in Table VII.

TABLE VII DISTRIBUTION OF MELANOMA NODULES

Treatment Group	L	RU	RM	RL	C	Total
Control (23)*	52	33	30	40	17	172
Treated (24)	115	121	67	94	52	449

* (number of animals in each group)
L, left lobe; RU, right upper lobe; RM, right middle lobe; RL, right lower lobe; C, cardiac lobe. Richters (1981).

The data summarized in Table VII indicate that the metastatic process is observed approximately equally all lobes of the lung, and that it is a statistically significant increase from controls. The authors made no attempt to determine the mechanism(s) responsible for

the enhanced metastasis in NO_2 exposed animals. They do, however, speculate that one or more of the following are most likely: (a) increased pulmonary capillary permeability and endothelial cell injury; (b) suppression of host-defense systems; (c) alterations in clotting mechanisms; or (d) a combination of events. They also suggest that the model system described may be useful for detecting subtle, but potentially harmful alterations associated with inhalation of air pollutants.

SYSTEMIC EFFECTS

Mice exposed to low levels (less than 1.0 ppm) of NO_2 have reduced resistance to respiratory infection (Gardner 1980). In addition, data from some epidemiologic studies have suggested that young children exposed to NO_2 have an increased incidence of respiratory infection (Speizer et al. 1980). Because of these observations a number of investigations have been undertaken in an attempt to ascertain if the immunological defense system of the body is compromised by exposure to nitrogen dioxide.

Kuraitis et al (1981) evaluated the effects of NO_2 on the spleen of adult and newborn Swiss Webster mice exposed to 0.35 ppm NO_2 for eight hours/day, 5 days/week for 6 weeks. A total of 217 control animals and 217 NO_2 exposed animals were used. There was a significant ($P<0.0025$) increase in spleen weight in newborn but not adult mice, and an increase in the size of spleen lymphoid nodules ($P<0.01$) in all groups. The authors explain these observations as follows: the NO_2 induces changes

in the lung making it a more favorable environment for microorganisms, and thus the immune response could be responsible for the lymphoid nodule change.

Roy-Burman et al (1981) have investigated the effect of exposure to NO_2 on changes in retrovirus* expression in mice. These investigators exposed a group of six-week old male Swiss Webster mice (a strain of mice known to be a low retrovirus expressor) to 0.8 ppm NO_2 for 8 hours/day, 5 days/week for up to 20 weeks, and a group of 10-week old female AKR mice (a strain of mice known to be a high retrovirus expressor) to 0.3 ppm NO_2 for 8 hours/day, 5 days/week for up to 20 weeks. Groups of 10 to 12 animals were sacrificed at weeks 1, 3, 6, 12, 15, and 18, and pooled tissue from lung, kidney, thymus and spleen examined for retrovirus expression. Only the spleen, of the tissues examined, showed changes in retrovirus expression in response to the NO_2 exposure. In the low expressor mice (Swiss Webster) there was an elevated retrovirus expression at the end of the first and 18th weeks exposure. In the high expressor mice (AKR) there was an elevated retrovirus expression at the end of the first, eight, 12th and 15th weeks of exposure. The authors indicate that the elevation in retrovirus expression was significant but do not present statistical data to support their conclusion. The authors conclude that, "the observed alterations of genetic expression in the splenic tissue of two different strains of mice, together with the fact that the spleen is closely interrelated with circulating blood components, support the

* Retrovirus (endogenous type C RNA tumor viruses) exist naturally within the cellular genome in a number of vertebrate species. It is known that expression of these viruses can be induced by a variety of treatments.

emerging notion that the spleen is a target organ for the effects of ambient levels of inhaled nitrogen dioxide.

Holt et al (1979) assessed the immunolgical function of BALB/c mice exposed to 10 ppm NO_2 for 2 hours/day, 5 days/week for up to 30 weeks. After 10, 15, 23 or 26 weeks of exposure groups of animals were sacrificed and the splenic cell response to phytohemagglutinin determined (a standard test to determine immunologic activity). The phytohemagglutinin response was depressed 50%* or more at all time periods. The effect of NO_2 on in vivo T-cell** function was also determined. T-cell function was enhanced, at weeks 10 and 23, normal at week 20, and slightly depressed at week 26. These investigators also studied the effect of NO_2 exposure on tumor rejection. Tumor (produced by virus TKL5) rejection was approximately 66% in the control group vs 10% in the NO_2 exposed group (authors do not indicate length of exposure). Finally, pathological examination of mouse lung after 30 weeks exposure to 10 ppm NO_2 showed an increase in goblet cells, diffuse interstitial pneumonia, and an increase in the dimensions of the peripheral air spaces (emphysematous change).

Miller et al (1980) have reported that mice exposed to NO_2 for three hours have a significant increase in pentobarbital-induced sleeping time (a measure of the animal's drug metabolizing ability). These investigators exposed CD-1 mice to 0.125, 0.25, 0.5, 1.0 or 5.0

*No statistical evaluation is given by the authors. They do state that within all experiments standard deviations within individual replicate determinations were within 10% of the mean. ** T-cell = a cell from the thymus gland. The assay is known as the graft-virus-host reactions (GVH).

ppm NO_2 for three hours/day for 1-3 days and then measured sleeping time induced by pentobarbital. There was an increase in sleeping time in mice exposed to 0.25 ppm or more nitrogen dioxide. There was no detectable effect on sleeping time after exposure to 0.125 ppm nitrogen dioxide. The increase in sleeping times was not dose related nor did it persist after repeated exposures. The authors speculate that the NO_2 somehow alters the hepatic mixed function oxidase activity which metabolizes pentobarbital. They also suggest that the lack of persistance results from tolerance to nitrogen dioxide. The lack of a dose response relationship in these studies suggests that there may be alternative mechanisms to explain these observations. These investigators have made some attempts to better define how exposure to NO_2 alters hepatic function. They assayed the liver from CD-1 mice exposed to 5 ppm NO_2 for 3 hours and found no change in aniline hydroxylase, aminopyrine N-demethylase and p-nitroanisole O-demethylase activities, three enzyme activities associated with the mixed function oxidase complex in the liver. (Graham et al 1982). Thus, additional experiments will be required to define how exposure to NO_2 alters hepatic function.

OTHER

Sherwin and Layfield (1976), have reported on the effect of exposure to NO_2 on protein leakage from blood into the lung tissue of mice. Proteins in the blood were labeled with a fluorescent dye, non-specifically, and the amount of protein leakage into the lung tissue was then determined using a fluorometric assay. Eight male mice were continuously exposed to 0.47 ppm NO_2 plus filtered air for 10 days, six mice were exposed for 12 days and eight mice were exposed for 14 days.

Similar numbers of animals were exposed only to filtered air. The mean protein value of all exposed animals was higher than that of the control animals ($P<0.025$). The increase in fluorescence in the lung is believed to be a measure of serum protein leakage into the alveolar spaces of the lung and thus a measure of tissue damage. These experiments, however, should be considered preliminary since no data are presented which can be used to validate the transfer of fluorescently tagged protein from the blood to the lung.

HUMAN HEALTH EFFECTS

Acute Exposure

During the Apollo-Soyuz joint space mission three American astronauts were accidentally exposed to nitrogen dioxide (Hatton et al, 1977). The exposure was for four minutes and 40 seconds, the peak concentration of NO_2 was 750 ppm and the average was 250 ppm. All three crew members complained of tightness of the chest, inability to inhale deeply, and nonproductive cough. X-rays of the chest taken about four hours after the exposure showed nondetectable pulmonary involvement. The symptoms worsened the next day. X-rays revealed findings characteristic of a diffuse chemical pneumonitis. Twenty-four hours after exposure to nitrogen dioxide all three astronauts had a significant ($P<0.05$) increase (69%) in urinary hydroxylysine glycoside indicating collagen degradation. The astronauts became asymptomatic on the third day and their chest x-rays were normal on the fifth day. At day 28 post-exposure their urinary hydroxylysine glycoside level was normal. This study adds additional, well documented information to the

data base on clinically observed effects following exposure to nitrogen dioxide. Although the exposure was to a very high level of NO_2 and only for a few minutes it does show that clinical symptoms precede detectable x-ray changes and that collagen breakdown was not detectable by x-ray.

Chronic Exposure

Hasselblad et al (1981), measured lung function in 16,689 white children from 6 to 13 years old from seven geographic areas. They measured the $FEV_{0.75}$* of each child, and had each family fill out a questionnaire detailing the frequency of respiratory illness in the family. Data analysis were performed on four groups; boys 6-9, boys 9-13, girls 6-9, girls 9-13. A significant negative association between maternal smoking and the $FEV_{0.75}$ was observed. In addition, among older girls (9-13 years of age) there was a decrease in the $FEV_{0.75}$ associated with the presence of a gas cooking stove in the home. According to the authors, the decrease in the $FEV_{0.75}$ of the girls 9-13 years of age approached statistical significance ($p < 0.524$). The authors suggest two possible explanations for these findings. First, older girls may be in the kitchen longer than younger girls (or boys), i.e., cumulative exposure may be more important than the simple frequency of exposure. Alternatively, there may be an interaction between smoking and NO_2 exposure in older girls. The authors indicate that they obtained no information which would support or exclude the third explanation.

Gamble et al (1983) have examined workers from five salt mines to determine if there are adverse health effects associated with exposure to NO_2 and respirable particulates from the use of diesel engines. The examination of each worker included a questionnaire, chest x-ray and spirometry.

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*$FEV_{0.75}$ = Forced Expiratory Volume at 0.75 Seconds.

There were 259 white male workers in the study, 97 of whom had worked more than 10 years in underground mines. The cumulative exposure (ppm X months) to NO_2 was determined for each worker; 115 had a low exposure (<48 ppm months), 38 a medium exposure (48-100), and 89 had a high exposure (>100). The relationship of the independent variables (age, smoking, exposure to NO_2) to pulmonary function was assessed by multiple regression. The mean NO_2 exposure was 1.3 ppm, with a low of 0.2 ppm and a high of 2.5 ppm. The highest individual exposure was 4.3 ppm NO_2. A large proportion of workers were exposed to average concentrations greater than 1 ppm nitrogen dioxide. The authors found that none of the exposure variables showed any significant association with reduced pulmonary functions. The use of cumulative exposure limits the sensitivity of the Gamble et al (1983) study since exposure to 1.0 ppm NO_2 for one month is given equal weight to an exposure of 0.1 ppm for 10 months. Some people, for example, who are likely to be affected by 1.0 ppm NO_2 may never react to 0.1 ppm nitrogen dioxide.

Berwick et al (1984) have recently reported on the incidence of respiratory illness in children exposed to NO_2 from unvented kerosene heaters. The observation period was January - March 1983. Information on respiratory illness history and heater use pattern was obtained bi-weekly by telephone. The children were under 13 years of age (mean 6.8 years), 59 living in homes with kerosene heaters and 62 living in homes without. Average two-week integrated NO_2 samples were taken in 113 of the 121 children's homes. The mean kitchen level of NO_2 in homes with kerosene heaters was 0.02 ppm [46.9 ug/m^3 with a range of 3-211 ug/m^3] and 0.007 ppm [14.1 ug/m^3- with a range of 0-80 ug/m^3] in homes without kerosene heaters. The mean levels of NO_2 found in bedrooms in

homes with and without kerosene heaters were 0.02 ppm and 0.006 ppm respectively. The overall average use of kerosene heaters was 7.7 hours/day. There was more gas cooking in homes without kerosene heaters compared to the homes with kerosene heaters (46.5 vs 17.5 minutes/day). There was no difference in the mean number of cigarettes smoked/day (12.6 with vs 12.7 without).

Berwick et al (1984) found that children living in homes with kerosene heaters had significantly more days of illness than children living in homes without kerosene heaters ($p< 0.05$). Age had a significant, inverse association with days of illness ($p< 0.05$) and a history of respiratory illness was positively associated ($p< 0.05$). Average hours of heater use showed a significant positive association to days of illness ($p<0.05$) when controlling for variables such as number of cigarettes smoked in the home, type of cooking fuel, age and sex. The authors conclude that their data suggest that young children with a history of respiratory infection are most sensitive to the adverse health effects of kerosene heater exposure. They also emphasize that their data are preliminary that there are potential biases and that their sample size is small.

Ware et al (1984) published a reassessment of their data on the incidence of respiratory illness and effects on pulmonary function in children exposed to passive smoking and gas cooking. The data base for their previous reports provided by 8,120 white children 6-10 years of age indicated increased respiratory illness before the age of two and decreased pulmonary function levels in children exposed to gas stoves. The latest report (Ware et al, 1984) examined data collected on the complete cohort, 10,106 white children. An analysis of the data found

that none of the respiratory illness and symptoms studied was significantly associated with exposure to gas cooking. In addition, the estimated effect of gas stove use was reduced by approximately 30% after adjustment for parental education. The study does, however, suggest that exposure to gas stoves may be associated with reduced pulmonary function. The authors suggest that these changes in conclusions may have resulted from errors not completely understood such as the choice of exposure variables used in their analyses. They suggest that a better assessment of exposure may require epidemiologic studies that include the monitoring of personal exposure.

Results from the newly available data from indoor epidemiologic studies do not appear to provide additional evidence substantiating or strengthening earlier conclusions regarding health effects potentially associated with exposure to NO_2 in gas stove homes. An analysis of the various studies suggest that there are a number of difficulties relating chronic low level exposure to NO_2 to changes in lung function or respiratory illness. For example, Kosmider et al (1972) found no changes in pulmonary function in workers exposed to 0.4 - 2.7 ppm NO_2 but they did find a significant increase in urinary hydroxyproline which suggests a breakdown of lung collagen or elastin. The study by Ware et al (1984) may also be limited in its ability to detect differences in respiratory illness since the criteria used severely restricts the incidences counted (e.g. the child must be out of school for three days or have a cough for 3 months).

A review of the recent discussions by the EPA, Clean Air Science Advisory Committee (CASAC)* on health effects associated with low level exposure to NO_2 also indicates a lack of concensus. For example, some investigators have reported effects at 0.1 ppm NO_2 and others have reported no effect at 4.0 ppm. The CASAC in its discussion of these data were unable to reconcile these differences in their effort to advise the EPA on setting an NO_2 standard. Part of the problem, as pointed out by Dr. Shenker, a member of the CASAC, is: " the very earliest NO_2 studies of respiratory symptoms and illness in children were incidence studies where they followed these children prospectively and looked at illness occurring during ... a two year period ... that study hasn't ... been done again ... ... all of the more recent work has been cross sectional which we know to be much less sensitive and subject to a lot of problems, not the least of which is historical illness and current exposure."

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* See the EPA Transcript of CASAC meeting July 1984

DISCUSSION/SUMMARY

A number of studies have been recently published containing measurements of lung tissue response to nitrogen dioxide as a function of age at exposure (Yam et al, 1978, Tyson et al, 1982; Cabral-Anderson et al 1977; and Evans et al, 1977). Two types of data have been obtained; biochemical and morphological. In the biochemical studies induction of antioxidation enzymes which help to protect the tissue from oxidative stress were measured and in the morphological studies increased cellular turnover in response to tissue damage was measured. The data obtained suggests that induction of antioxidative enzymes in response to NO_2 injury begins at about one month of age in the rat, peaks at about one year and then declines with age. The very young animal, less than one month of age, has a high initial (control) level of antioxidative enzymes in lung tissue which declines with age. This could account for the lack of response to oxidant stress in lung tissue of very young animals.

Morphologic changes are seen in lung tissue of one and three month old rats exposed to less than 1.0 ppm nitrogen dioxide. The lung tissue of one year old rats does not show morphologic changes, but by 21 months of age there are again indications that exposure to NO_2 results in detectable changes. These observations suggest that the growing lung tissue in a young animal is relatively more sensitive to the oxidant stress of nitrogen dioxide and that as the ability to induce the antioxidant protective system in old animals diminishes the lung tissue again becomes more sensitive to nitrogen dioxide.

There are two recent studies which report cellular damage to animal lung tissue following exposure to less than 0.5 ppm nitrogen dioxide. Kyono and Kawai (1982) have reported morphologic changes in one month old rats exposed to 0.1 ppm NO_2 while Sherwin and Richter (1982) have reported Type II cell hyperplasia in mice exposed to 0.34 ppm nitrogen dioxide. Previously, Type II cell hyperplasia was detected only after exposure to 2 ppm nitrogen dioxide. It should be noted that these studies significantly lower the level at which this type of damage to lung tissue has been detected.

A number of studies have now been published that begin to explain the early observations by Gardner (1980) that mice exposed to NO_2 have a reduced resistance to respiratory infection. The studies by Kuraitis et al (1981), Roy-Burman et al (1981) and Holt et al (1979) have reported systemic effects, i.e. alteration in the immunological function of animals following exposure to nitrogen dioxide. The splenic changes observed, after additional research, may offer sufficient sensitivity to determine small but significant changes in an animals defense mechanism which can be correlated with a reduced resistance to respiratory infection.

There are now animal studies which clearly show that the uptake of exogenous NO_2 results in an increased excretion of nitrosamine (Saul and Archer, 1983). The data obtained indicate that the relationship between uptake and excretion is dose related when the dose is more than 1 ppm nitrogen dioxide. At 0.2 ppm NO_2 conversion is less than half that expected. In addition, the study by Richters and Kuraitis (1981) showing increased metastases of injected cancer cells might also add to the concern that NO_2 plays a role as an environmental carcinogen.

Lastly, a review of the most recently published epidemiologic studies do not appear to provide much additional evidence substantiating or strengthening earlier conclusions regarding health effects associated with chronic low level exposure to NO_2 and/or a history of respiratory illness or lung function levels.

CONCLUSIONS

The literature reviewed in this report adds further support to the health assessment in made 1983 by the Health Sciences staff that chronic exposure to low levels of NO_2 is associated with demonstratable health effects. In addition, some of the new data indicate that events such as cellular damage, previously associated only with exposure to high concentrations of NO_2, are now detectable after exposures to 0.34 ppm nitrogen dioxide. Recent studies also suggest that lung tissue of older animals may be more susceptible to the adverse effects of NO_2 than young animals. Lastly, there are some data from animal studies which suggests that exposure to NO_2 is associated with alteration in immunological function. A number of studies have shown changes in spleen weight or retrovirus expression in mice exposed to 0.125-0.35 ppm nitrogen dioxide. The results of these animal studies indicate that NO_2 exposure effects the immune function and thus increases the susceptibility to infection. However, the lack of methods for extrapolation of the animal data to man for analogous effects limits their usefulness.

The studies on the metabolism of NO_2 indicate that there is a relationship between NO_2 uptake and nitrosamine excretion. These studies raise a number of new concerns regarding the effects of low level chronic exposure to nitrogen dioxide.

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UNITED STATES GOVERNMENT

Memorandum

U.S. CONSUMER PRODUCT
SAFETY COMMISSION
WASHINGTON, D.C. 20207

DATE: 6/29/83

TO : Peter W. Preuss, Ph.D., AED/HS
THRU : Andrew G. Ulsamer, Ph.D., Director, HSHE AGU

FROM : Rita Orzel, Ph.D., Chief, Toxicology Review Branch RO/AGU
Warren Porter, Ph.D., Director, HSHL

SUBJECT: Status Report: Pollutants Generated by Unvented Gas Space Heaters

1. INTRODUCTION

In the course of its investigation of acute carbon monoxide poisoning resulting from the use of unvented gas space heaters, the Commission became concerned about possible health effects resulting from prolonged exposure to carbon monoxide and other pollutants emitted by such heaters. To address this issue, a study was conducted at the Lawrence Berkeley Laboratory (LBL) to estimate consumer exposure to a number of pollutants including carbon monoxide, carbon dioxide, nitrogen oxide, nitrogen dioxide, formaldehyde, and particulates emitted by unvented gas space heaters. Based on the findings of the LBL Study, an assessment of possible health hazards associated with exposure to pollutants emitted by unvented gas space heaters under various conditions of usage has been performed based upon existing reviews and standards. In addition, Health Sciences is conducting an in-depth evaluation of toxicity data on the possible human health effects resulting from exposure to these pollutants.

The present report summarizes the work completed at LBL and provides a discussion of existing health standards for pollutants emitted by unvented gas space heaters.

2. LBL STUDY PLAN

The LBL study was designed to estimate consumer exposure to pollutants generated by unvented gas space heaters and consisted of two phases: a laboratory chamber study and a field test study.

A) Laboratory Chamber Study (Phase One) (Appendix C)

The first phase of the LBL study involved the testing of eight heaters in a 27 m^3 laboratory chamber. The heaters were allowed to burn 5 ft^3 of natural gas under ventilation conditions ranging from 0.2 to 5.0 air changes per hour. Heater tuning was adjusted (well- or maltuned) by varying the air/fuel ratio before each test. The heaters were also tested at full or partial gas input. Oxygen, carbon monoxide, carbon dioxide and nitrogen dioxide were monitored continuously during the test. One-hour average measurements of formaldehyde and particulates were made after the heater was shut-off.

In addition, three heaters were also operated to steady-state conditions in the chamber to establish that steady-state values predicted mathematically from chamber tests were comparable to actual concentrations measured in steady-state chamber tests.

Further, using the emission rates specific for each heater (determined in both the chamber and field tests) and a mathematical model, steady-state pollutant concentrations were calculated for a number of well- and maltuned heaters for a 1150 ft^2 house at 0.5 air change per hour.

B) Field (Test House) Study (Phase Two) (Appendix D)

In the field study, measurements of the pollutants generated by operation of four selected heaters in well- or maltuned conditions were made in an unoccupied, one story house (1150 ft^2) with a volume of 8,500 ft^3. Heaters were operated to near steady-state conditions while ventilation rates were varied from 0.4 to 1.14 air changes per hour by opening doors and windows. Pollutant gases (carbon monoxide, carbon dioxide and nitrogen dioxide) and oxygen were measured at one minute intervals at several locations in the house. Particulate measurements were made every 10 minutes and formaldehyde levels were measured every hour.

Finally, predicted pollutant levels were calculated by utilizing emission rates obtained in chamber tests and the mathematical model, and compared to actual pollutant concentrations measured in the (1150 ft^2) house under similar ventilation conditions.

3. RESULTS AND DISCUSSION

A) Oxygen Depletion and Pollutants Generated by Heaters

Oxygen depletion and levels of carbon dioxide, carbon monoxide and nitrogen dioxide generated by well-tuned and maltuned heaters in the chamber were determined. These data were used in a mathematical model to predict levels that could exist in a house (1150 ft^2) at 0.5 air changes per hour while the heater would be in use. These predicted levels, the levels actually measured in the test house, and their correlation, as well as factors affecting the generation of these pollutants are summarized and discussed below.

a) Oxygen Depletion

Reductions in oxygen levels were observed in the chamber as well as in the model house. The normal oxygen level in the air is about 21%. Predicted steady-state levels for the house ranged from 19.0% to 20.3%. Actual measurements in the house ranged from 19.1% to 20.7%. Therefore, the measured levels of oxygen in the house correlated well with concentrations predicted by the model based on chamber data.

b) Carbon Dioxide:

Levels of carbon dioxide produced were uniform among heaters of the same size; the larger heaters produced higher concentrations. Tuning did not alter carbon dioxide emission characteristics. The normal level is about 0.03%. Predicted house levels of carbon dioxide ranged from 0.4% to 1.1% for well-tuned heaters and 0.5% to 0.9% for maltuned heaters. Measured levels in the house were 0.3% to 1.1% and 0.5% to 0.9% for well- and maltuned heaters, respectively. The projected carbon dioxide values for any one heater were within 25% of measured values.

c) Nitrogen Dioxide:

Levels of nitrogen dioxide are generally around 0.02 ppm in homes in the absence of home combustion sources. Projected house nitrogen dioxide levels based on chamber tests ranged from 0.4 to 1.46 ppm for well-tuned and from 0.8 to 1.4 ppm for maltuned heaters. Actual measurements in the test house varied from 0.4 to 1.5 ppm and 0.5 to 1.4 ppm for well-tuned and maltuned heaters, respectively. Maltuning increased nitrogen dioxide generation for some heaters. For specific heaters, measured values showed variations up to three-fold compared to predicted measurements based on chamber data. However, an intensive analysis of the field study data indicated that for some heaters the nitrogen dioxide emission rate changes with the length of time the heater is allowed to operate. Although this phenomenon is not completely understood, it is believed that the change in emission rate is due in part to the increase in temperature of the surfaces around the flame. As the temperature increases to

a constant level, the emission rate approaches a final constant value. This indicates that for modeling purposes both initial and steady state emission rates need to be determined to account for short term or long term heater use, respectively. When nitrogen dioxide levels were predicted based on the steady state emission rates, the predicted house level was in good agreement with the measured level.

d) Carbon Monoxide

Background levels of carbon monoxide are about 1 ppm. Predicted carbon monoxide levels in the house for well-tuned heaters ranged from 1.6 to 20.3 ppm. For maltuned heaters operated with excess fuel, predicted levels ranged from 2.8 to 12.8 ppm, while one heater operated with excess air was predicted to produce 92.4 ppm of carbon monoxide.

The near steady state values measured in the test house ranged from 1.6 to 20.3 ppm for well-tuned heaters. Measured values for maltuned heaters operated with excess fuel ranged from 2.8 to 12.8 ppm; the excess air maltuned heater emitted 92.4 ppm. Carbon monoxide emission rates changed with some heaters depending on the length of time the heater was allowed to burn. Thus, like nitrogen dioxide, both short and long term emission rates need to be determined for use in various predictive modeling scenarios representing either short or long periods of use. In the case of CO, modeled levels for long term use, using the long term emission rates, showed good agreement with levels measured in the field study house.

e) Formaldehyde

Emissions of formaldehyde were highly variable among heaters and between tests of the same heaters. The levels predicted using chamber data and the model were generally below 0.1 ppm; however, one maltuned heater operated with excess air was predicted to generate 2 ppm. All measured levels in the house for well-tuned heaters were consistently uniform and less than 0.1 ppm. The maltuned heater predicted to generate 2 ppm produced 1.3 ppm formaldehyde when operated in the house with excess air. The background levels for the tests in the field study house were generally below 0.02 ppm.

f) Particulates

Particulate emissions were uniformly low in both chamber and house tests and considerably below the 75 ug/m^3 long-term EPA outdoor standard. The highest level recorded in the house was about 4 ug/m^3.

B) Factors Affecting Pollutant Concentrations

The data obtained in the chamber tests and in the house indicated that a number of factors can affect pollutant generation. These factors are discussed below:

a) Mixing of Air

One of the requirements for the use of a mathematical model to reliably predict steady-state concentrations from short-term chamber tests to the house, is that the air in the space being modeled must be well-mixed.

Tests were conducted in the chamber and in the test house to establish adequacy of air mixing. In the chamber, a number of fans were needed to assure mixing. In the field house, with all interior doors open, and without the use of fans, mixing was rapid and complete, as indicated by similar pollutants levels in the rooms monitored. Thus, the mixing requirement of the model was met in both the chamber and the field house.

b) Air Change Rate

Laboratory testing was conducted in a chamber located in a large building. Under this condition, concentrations of pollutants correlated well (inversely) with air change rates. In the test house, which is subject to environmental factors such as wind velocity and direction, air currents, etc., it was difficult to achieve a desired air change rate or to keep it uniform for the duration of the test. Even though wide variations in air change rates were observed in the house, pollutant concentrations were, in most cases, inversely proportional to ventilation rates.

c) Tuning

Tuning of heaters was accomplished by adjusting the air shutter, thereby varying the air/fuel ratio to a setting where the carbon monoxide emission was at a minimum as measured by instrumentation and noted by visual observations of flame color and stability.

Tuning tests conducted on one high carbon monoxide-emitting heater indicated that very small changes in the air shutter opening produced large changes in carbon monoxide concentrations even though visually the flame characteristics did not appear to be subjectively different from the prior shutter setting. After proper tuning by instrumental means, this heater produced low concentrations of carbon monoxide.

These experiments indicate that visual observation of flame characteristics alone is not a reliable predictor of optimal tuning, at least for some heaters. As most heaters used in homes would probably be tuned in this manner and not by instrumental means, it is likely that many of the heaters in use may be maltuned to a lesser or greater degree. Tests conducted with maltuned and well-tuned heaters indicate that higher levels of carbon monoxide nitrogen dioxide, and formaldehyde may result under maltuned conditions.

d) Burner Design

Three heaters, all from the same manufacturer, were found in the chamber tests to be low emitters of carbon monoxide and relatively insensitive to tuning. The design of the burner element in these heaters is different from other heaters in that the burner uses slots which produce a feathered flame rather than small flamelets which form on the circular ports common to the other heaters. It appears that burner design has a significant effect on emission rate and sensitivity to tuning.

e) Partial/Full Gas Input

According to manufacturers' instructions, heaters are designed for operation at full gas input. By adjusting a pressure regulator on the heaters, the gas supply can be reduced. As this would provide some control of temperature, it is considered a forseeable use. A number of studies were therefore conducted in the chamber with heaters operated at partial gas input. Results indicate that carbon monoxide and formaldehyde emissions at full input were one to two-fold greater than for partial input; other pollutants were increased to a lesser extent. More complete combustion at partial input may account for this finding.

4. MODELING

The laboratory testing (Appendix C) demonstrated that three heaters from one manufacturer exhibited emission rates of carbon monoxide and nitrogen dioxide that were reproducible to within ± 15% overall for the three heaters tested. These heaters had a different design of burner than the remaining heaters. This manufacturer's heaters exhibited emission rates that were also less sensitive to the air/fuel ratio than the other manufacturer's heaters. The heaters from the remaining manufacturers did not exhibit the reproductibility of emission rates previously mentioned. Whether this is a result of changes in air/fuel ratios, length of burn times, or sensitivity to mechanical parameters, has not been determined from these limited studies. Using the mass balance equation described by Traynor (Appendix C) and the short term emission rates determined from chamber testing (Tables 4 and 12, Appendix C), steady state concentrations of the various pollutants and oxygen depletion were estimated for the house where the field study was performed. These levels predicted from short term emission rates are compared in Tables 1 and 2, to the levels predicted from long term emission rates determined in the field study house (Appendix D).

Generally use of either short term or long term emission rates would predict carbon dioxide oxygen and levels within ±25% of the measured concentration. Use of short term emission rates for predicting steady state carbon monoxide and nitrogen dioxide concentrations were less successful due to the variation of emission rate with longer burning times. However, predictions of steady state levels, based on the long term emission rates determined in the field study, were in good agreement with measured concentrations.

Nitrogen dioxide is a reactive gas which is removed from the air by factors other than just ventilation. Previously published studies (LBL-9522 and LBL-12563) indicate that, in addition to removal of nitrogen dioxide by ventilation, nitrogen dioxide removal by non-specific reactions varies from 0.15 air change per hour to 1.3 air changes per hour. In both the chamber and house studies reactive decay was about 0.3 air change per hour. Thus, in predicting nitrogen dioxide air levels, one should remember, that in the home environment, where many activities are occurring, the reactive decay of nitrogen dioxide may vary considerably. The net effect could be a 2 to 4-fold discrepancy between predicted and measured air levels at any given emission rate.

It is clear from the limited studies performed that modeling of home air levels based on data from laboratory chamber studies is promising, if certain constraints and caution are exercised. Thus, one must ensure the adequate mixing of air in the chamber and assume adequate mixing in the home (both of these conditions were met in both chamber and field studies). A constant and reproducible emission rate must be obtained. This constraint was met only for one manufacturer's heaters. To clarify the inability to obtain constant and reproductible emission rates from the remaining heaters was beyond the scope of work of these studies and would require an investigation of heater design. Finally, the loss of pollutants through reactive decay (nitrogen dioxide), as opposed to only ventilation losses, is variable and depends upon environmental conditions which are also not fully understood.

5. COMPARISON OF POLLUTANTS EMITTED BY HEATERS AND HEALTH STANDARDS

The range of pollutant levels produced by heaters in the house and existing/proposed standards for these pollutants in ambient/workplace air are summarized in Appendix A.

A comparison of these values indicates that the heaters produced levels of nitrogen dioxide which exceeded both EPA and World Health Organization ambient air standards. In a number of instances, carbon dioxide levels measured in the home were in excess of the Navy standard for continuous exposure in submarines. Carbon monoxide concentrations from several heaters in the house exceeded the EPA ambient air standard.

Several heaters produced levels of nitrogen dioxide (30A, 30C, 16B, 12A), carbon dioxide (30A and 30C at low air exchange rates), and carbon monoxide (30A maltuned, 16B well tuned) in the house which exceeded some of the existing/ proposed standards and thus, have the potential of adversely affecting the health of exposed individuals.

6. CONCLUSIONS

Based on chamber tests and house measurements, some general observations can be made with regard to emissions from unvented gas space heaters and effects on oxygen levels:

- All heaters produced carbon monoxide, carbon dioxide, nitric oxide, nitrogen dioxide, formaldehyde, particulates, and depleted oxygen levels.

- Carbon monoxide emissions were variable from heater to heater and among tests of the same heater. Generally, concentrations of less than 15 ppm were measured for well-tuned heaters; a maltuned heater operated with excess air produced 92.4 ppm in the house.

- Oxygen levels did not fall below 19% regardless of the state of tuning.

- Carbon dioxide emissions related well to heater size; larger heaters produced higher concentrations. The highest level measured in the house was 1.1%. Most heaters, however, produced levels of 0.8% or less regardless of the state of tuning.

- Measured nitrogen dioxide levels were variable and exceeded 0.4 ppm for all heaters regardless of state of tuning.

- Formaldehyde levels were generally below 0.1 ppm except for one heater which produced concentrations of formaldehyde above 1 ppm. A maltuned heater operated with excess air produced 1.3 ppm of formaldehyde in the house.

- Particulate emissions measured in the house were consistent and low.

Heaters were also observed to differ in emission rates and sensitivity to tuning. Heaters with a slot-type burner design emitted lower levels of pollutants and were relatively insensitive to tuning when compared to heaters with circular port type burners. It was also noted that precise tuning of heaters could not be accomplished by visual observation of flame characterictics alone, but required laboratory instrumentation. In the chamber, operation of heaters at partial gas input resulted in lower emission rates and, consequently, reduced levels of pollutants as compared to operation at full gas input. Other factors affecting pollutant concentrations were air change rates and distribution of the room air throughout the remainder of the house.

TABLE 1

Predicted and Measured Steady State Levels of Oxygen and Pollutants Generated by Heaters in a 1150 ft^2 House

Heater	Air Changes Per Hour	Short Term Emission Rates: Carbon Monoxide (PPM) P[a]	Short Term Emission Rates: Carbon Monoxide (PPM) M[b]	Short Term Emission Rates: Carbon Dioxide (%) P	Short Term Emission Rates: Carbon Dioxide (%) M	Long Term Emission Rates: Carbon Monoxide (PPM) P	Long Term Emission Rates: Carbon Monoxide (PPM) M	Long Term Emission Rates: Carbon Dioxide (%) P	Long Term Emission Rates: Carbon Dioxide (%) M
30 A (well-tuned)	0.37	7.7	5.1	1.0	1.0	5.0	5.1	1.0	1.0
30 A (well-tuned)	0.49	5.8	4.2	0.8	0.8	3.9	4.2	0.8	0.8
30 A (well-tuned)	1.1	2.7	1.6	0.4	0.4	1.6	1.6	0.4	0.9
30A maltuned[c]	0.45	40.5	9.5	0.9	0.9	9.2	9.5	0.9	0.9
30A maltuned[c]	0.57	32.0	12.8	0.7	0.7	12.0	12.8	0.7	0.7
30A maltuned[c]	0.70	26.0	2.8	0.5	0.6	3.1	2.8	0.5	0.6
30A maltuned[d]	0.76	78.0	92.4	0.5	0.5	94.9	92.4	0.5	0.5
30C (well-tuned)	0.36	3.5	4.5	1.1	1.1	3.8	4.5	1.1	1.1
16B (well-tuned)	0.46	21.9	20.3	0.4	0.5	19.1	20.3	0.4	0.5
12A (well-tuned)	0.39	13.4	3.1	0.4	0.3	4.4	3.1	0.4	0.3

[a] Predicted [b] Measured [c] Excess fuel [d] Excess air

TABLE 2

Predicted and Measured Levels of Oxygen and Pollutants Generated by Heaters in a 1150 ft^2 House

Heater	Air Changes Per Hour	Short Term Emission Rates: Nitrogen Dioxide (PPM) P	M	Short Term Emission Rates: Oxygen (%) P	M	Long Term Emission Rates: Nitrogen Dioxide (PPM) P	M	Long Term Emission Rates: Oxygen (%) P	M
30A (well-tuned)	0.37	2.0	1.5	19.0	19.1	1.5	1.5	19.5	19.1
30A (well-tuned)	0.49	1.7	1.1	19.5	19.4	1.1	1.1	19.3	19.4
30A (well-tuned)	1.1	0.9	0.4	20.3	20.3	0.4	0.4	20.3	20.3
30A maltuned[c]	0.45	1.3	0.8	19.1	19.2	0.8	0.8	19.2	19.2
30A maltuned[c]	0.57	1.1	0.8	19.7	19.5	0.8	0.8	19.8	19.5
30A maltuned[c]	0.70	0.9	0.5	19.9	19.9	0.4	0.5	19.8	19.9
30A maltuned[d]	0.76	0.9	1.4	20.2	20.0	1.4	1.4	20.0	20.0
30C (well-tuned)	0.36	1.0	1.0	19.1	20.0	1.2	1.0	18.9	20.0
16B (well-tuned)	0.46	1.0	0.9	19.5	20.2	0.8	0.9	20.1	20.2
12A (well-tuned)	0.39	0.8	0.4	20.2	20.7	0.4	0.4	20.5	20.7

[a] Predicted [b] Measured [c] Excess fuel [d] Excess air

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Appendix A

Measured Pollutant House Levels and Existing/Proposed Standards

Pollutant	House [a] Levels	Standards Proposed/Existing	Agency	Comments
Carbon monoxide	1.6-20.3 ppm[b] 2.8-92.4 ppm[c]	9 ppm 35 ppm 50 ppm	EPA NIOSH OSHA ACGIH	Protect entire population Protect Workers
Carbon dioxide	0.3-1.1% [b] 0.5-0.9% [c]	1% 1% 0.5% 1% 1.5%	NIOSH Navy " NASA ACGIH	Protect Workers Continous exposure in submarines 90-day exposure, nuclear submarines Manned spacecraft and skylab flights Protect Workers
Nitrogen dioxide	0.4-1.5 ppm[b] 0.5-0.8 ppm[c]	0.1-.17 ppm .05 ppm	WHO EPA	1-hour exposure Annual average
Formaldehyde	0.1 ppm[b] 1.3 ppm[c] (1 test)	0.1 ppm 3 ppm 1 ppm	ASHRAE OSHA NIOSH NIOSH	Ceiling for indoor air TWA occupational Recommended TWA (1976) Recommended HCHO be handled as potential carcinogen (1981)

[a] 8,500 ft^3 and 0.5 air change rate
[b] well-tuned
[c] maltuned

Peter W. Preuss, Ph.D., AED, HS

MAY 1 = 1983

Warren K. Porter, Jr., Director, HSHL
Paul White, HS

Kerosene Heater Emissions: Estimation of Exposure.

Background

Various reports (1, 2, 3, and 4) indicate the production of certain pollutants from the use of kerosene space heaters. These pollutants, carbon monoxide (CO), carbon dioxide (CO_2), nitric oxide (NO), nitrogen dioxide (NO_2), and sulfur dioxide (SO_2), if present at high levels, can cause adverse health effects. A discussion of the health effects resulting from exposure to CO, CO_2, NO_2, NO_2, and SO_2 has been forwarded to the Chemical Hazards Program. Earlier, Health Sciences, estimated potential human exposure from the use of kerosene heaters in a closed room through application of a mass balance equation to the relatively limited emission factors published in the scientific literature.

This report provides our estimates of exposure based on twenty-four tests performed by the CPSC staff on twelve individual heaters. Duplicate tests of each heater were performed and are described in the Engineering Test Manual (Tab A). In addition four heaters were tested under conditions of improper settings, i.e. high fuel consumption rate, and low fuel consumption rate, which were accomplished by raising or lowering the wick. From the data obtained, emission rates for the pollutants were calculated and these rates used in three models to estimate exposure under:

a. steady state conditions and
b. dynamic concentration conditions for one room size (250 ft^2) typical of the living room described in the Kerosun and National Kersene Heater Association (NKHA) use survey.

5) After extinguishing the heater, decay of pollutants was monitored for about one hour.

6) Fuel consumption was monitored.

7) The same lot of fuel was used for all tests; it contained 0.01% sulfur.

The data obtained from the kerosene heater testing were treated by regression analysis to determine pollutant generation rates and reactive decay rates for NO_2. The pollutant generation rates, Table 1, indicate that the wick-type blue flame heaters, whose flame temperature is lower than the wick-type white flame heater, consistently generated less NO and NO_2 than the white flame heaters or the wickless heater. In general, the cooler blue flame heaters generated about one half the amount of NO_2 produced by the white flame convective or wickless, heaters. However, the lower temperature of the blue flame heaters, which results in lower combustion efficiency, produced CO at nearly five times the rate of the white flame convective or wickless heaters. Near steady state pollutant concentrations (measurements and predictions based on the calculated emission rates) are presented in Table 2.

Analysis of Emission Rates

Because of limited resources for testing the staff decided to perform only duplicate tests on each heater. Thus for the 24 scheduled tests of heaters operating according to the manufacturers recommendations, twelve heaters could be tested. Although this approach would not permit estimating the reproducibility of emissions from an individual heater, it would allow estimation of reproducibility of emission rates for kerosene heaters as a group. The first group consisted of blue flame convective and radiant heaters, white flame connective heaters and one wickless heater were the second group. The wickless heater was

All calculations are done for a heater within a 1150 ft^2 ranch style home as described by Traynor (5). Even with these restrictions a number of factors may affect the projected pollutant levels.

1) The use geometry - the size of the room where the heater is located and whether the room is closed, has a door ajar, or is open to the rest of the house;
2) The air change rate of the home with the outdoors and the heating requirements of the home (how well insulated);
3) The outside temperature and the temperature being maintained in the areas of the home adjacent to the heater room;
4) The heater BTU output; and
5) The heater pollutant emission rate per BTU.

In order to address these variables we have taken the following steps in our modeling:

1) Three basic model types are used:
   A. Closed Room - The heater is used in a room with doors and windows closed. Air does not mix with the remainder of house, but does exchange with the outdoors. The room size is scaled to the heater size to maintain 70°F temperature.
   B. Semi-Open Room - The heater is used in a room with the door opened two inches and air mixing with the remainder of the house. The rate of mixing was calculated using the mass air flow equation as developed by Ingersoll and supplied to CPSC by S. Winter and D. Lavery (7). It should be noted that this equation was developed for a 3 foot wide door; however we made a linear extrapolation to a 2 inch cracked door. Thus, since no data are available

(BTU/Ft^2-hr-^oF) calculated from the data available from studies conducted by the Department of Energy's (DOE) Residential Energy Conservation System (RECS) program (9), American Gas Association (AGA) (10) and the National Association of Home Builders (NAHB) (11). The DOE and AGA estimates, based on residential gas consumption, estimated heat load requirements of 0.37 and 0.32 BTU/Ft^2-hr-^oF for the U.S. housing stock in existence in 1979 and 1980 respectively. The NAHB estimates, based on an analysis of current building practices using the DOE 2 computer model, estimated a heating load requirement of 0.21 BTU/F^2-hr-^oF.

The lower NAHB estimate would reflect currently used tighter construction practices, as well as the contribution of solar heating.

B) The second example was modeled assuming an air exchange rate of 0.8/hr which is a typical value for U.S. homes taken from the NAS report on Indoor Pollutants (12). This home is assumed to have a heating requirement of 0.49 BTU/Ft^2-hr-^oF. This assumption is believed to be reasonable in that it is based on a Rand Corporation report (1975) (13) that estimated heat load based on fuel consumption and efficiency estimates for gas heated homes. Further, the value is in the range of a National Bureau of

4) The heaters tested by the CPSC staff fell into three groups: smaller heaters (n=6) which averaged 6800 BTU/hr*, medium sized heaters (n=3) which averaged 10,400 BTU/hr* and larger heaters (n=2) which averaged 15,600 BTU/hr*. Calculations were performed on the basis of these three heater sizes.

5) Heater pollutant emission rates were calculated exclusively from averages of the data from the Engineering Laboratory tests (Table 4).

Under these modeling conditions for each room geometry, heater size, and heater type there are two outside temperature conditions and two heating load assumptions used for a total of four scenarios. In the closed room and semi-open room models, each of these scenarios is associated with a different room size scaled to maintain a 70°F temperature. These room sizes are compatabile with those cited in the NKHA and Kerosun user surveys. When calculated room size exceeded 650 ft^2 these models were judged not likely to represent a common home situation and pollutant levels were not modeled. In order to condense this data we show only the highest and lowest of the pollutant levels predicted for the different outside temperature and heating load assumptions. This range is given for each room geometry, heater size and heater type.

Tables, 5 and 5a give the projected pollutant levels for each of these cases. The details of the calculations are given in Appendix A.

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* There heat outputs were calculated from the average fuel consumption rate in grams per hour and a conversion of 41.24 BTU/g of fuel.

Indoor Air Quality

House: 1150 square feet, single story, ranch style, hot water heat

Kerosene Heater Type: 6800 BTU Blue Flame.

Case 1: Outdoor Temp 0°F, Thermostat 50°F, Heater Room 70°F

Loose, poorly Insulated House 0.8 Air Changes per hour

Steady State Concentration			
Pollutant	Door Open	Door Ajar	Door Closed
NO_2 (ppm)	0.070	0.19	0.47
CO (ppm)	4.66	11.34	34.27
CO_2 (%)	0.15	0.36	1.09
Room Size ft^2	1150	121	147

If the heater were the same size but a white flame type:

Steady State Concentration			
Pollutant	Door Open	Door Ajar	Door Closed
NO_2 (ppm)	0.12	0.34	0.87
CO (ppm)	1.11	2.7	8.16
CO_2 (%)	0.15	0.36	1.08
Room Size ft^2	1150	121	147

Important Points:

- Carbon Monoxide levels are appreciably lower for a white flame heater when compared to blue flame heaters under comparable circumstances.
- Nitrogen dioxide levels are nearly double the levels observed with blue flame heaters under comparable circumstances.
- Both blue flame and white flame heaters result in excessive NO_2 levels when the door is closed.

Case 3: Same House as Case 1; however the outdoor temperature is warmer.

Outdoor temp 32°F, Thermostat 60°F, Heater Room 70°F.

Loose poorly insulated house 0.8 air changes per hour.

	Steady State Concentration		
Pollutant	Door Open	Door Ajar	Door Closed
NO_2	0.066	0.12	0.25
CO	4.66	9.83	17.89
CO_2	0.15	0.31	0.57
Room Area ft^2	1150	259	276

If a white flame heater of 6800 BTU/hr output were substituted for the blue flame heater:

	Steady State Concentration		
Pollutant	Door Open	Door Ajar	Door Closed
NO_2	0.12	0.23	0.47
CO	1.11	2.34	4.26
CO_2	0.15	0.31	0.57
Room Area ft^2	1150	259	276

Important Points:

- CO levels are lower than under the colder outside temperatures of Case 1 and Case 2. This is a result of the fact that the 6800 BTU/hr heaters can heat larger areas.
- NO_2 reaches levels where concern should be shown with the white flame heater is used in a closed room.
- NO_2 levels may be of concern to sensitive individuals for white flame heaters in a room with the door ajar or in closed rooms when a blue flame heater is used.

Modeling Pollutant Levels in a House (dynamic)

Since during the burning time of a heater pollutants increase in concentration to a level closely approaching steady state, we performed dynamic modeling for six different conditions for the three pollutants CO_2, CO, and NO_2. Pollutant levels were modeled for a 10,400 BTU/hr white flame convective heater and for a 6,800 BTU/hr blue flame heater. The emission rates for the pollutants were averages of the data from the CPSC testing of blue and of white flame heaters. The three models used here are the open house, semi open room (door open 2 inches) and closed room.

The following model parameters were used:

Emission Rates:

6,800 BTU/hr Heater (Blue Flame)

CO_2	41 cc/kJ
CO	0.125 cc/kJ
NO_2	2.2×10^{-3} cc/kJ

10,400 BTU/hr Heater (White Flame)

CO_2	40 cc/kJ
CO	0.017 cc/kJ
NO_2	3.2×10^{-3} cc/kJ

Air exchange rate	0.8 per hour
Room size	250 ft^2 (56.6m^3)
House size	1150 ft^2 (240m^3)
q_{12}	80.4 m^3/hr

temperatures in the room unless the outdoor temperature was 32°F and the house was tight and reasonably well insulated. The 10,400 BTU/hour white flame convective heater in the semi-open configuration would, on the other hand, cause the temperature in the heater room to be over 70°F for all cases except for an outdoor temperature of 0°F and a loose, poorly insulated house.

The following chart presents the predicted 8 hour average exposures and indicates the equivalence of the dynamic and steady state models for the reported most common use pattern, i.e. door open.

Effect of Other Pollutant Sources

The contribution of other sources of pollutants to the indoor environment has been discussed by Traynor et al (14). In a study of gas ranges and ovens in an unoccupied house, (the one used by CPSC in our previously discussed modeling) they monitored CO and NO levels in the kitchen, living room and bedroom of the house during and after use of a range and oven. The protocol consisted of operation of the oven ($350^{o}F$) and two burners (high setting) with a pot of water on each burner. The burn time was about 35 minutes. Since our model is based on the test house used by Traynor (5,8) and since the oven/range source strength would be additive to the kerosene heaters emissions for the period of time the range/oven was in use, the final pollutant levels would be the sum of those produced by kerosene heaters and range/oven during and near the time their uses coincided.

Thus, if the heater alone was operated in an open house situation and the oven was used for cooking for one and a half hours in the late afternoon when the heater would be likely to be operating, we predict the general levels of CO and NO_2 under the following conditions:

Parameters:

Air change 0.8/hr

Reactive Decay 0.3/hr

Oven Emission Rates

CO 1655 cc/hr

NO_2 45.3 cc/hr

Heater on at 4:00 PM

Oven on at 5:00 PM

Oven off at 6:30 PM

Heater off at 12:00 Midnight.

Sulfur Dioxide

The concentrations of surfur dioxide measured during the CPSC kerosene heater testing program were observed to be erratic and did not correspond to levels calculated for combustion of kerosene containing 0.01% sulfur. Thus a special chamber calibration was performed by adding SO_2 to the chamber such that a steady state concentration of about 1 ppm would result. In the first test with no heater present, the concentration achieved was only 50% of the calculated level while in a second test when an operating heater was also present in the chamber only 30% of the calculated level was obtained. The concentration vs time profile for the latter test was erratic indicating that the SO_2, other pollutants, and water vapor were interacting with each other or the chamber materials or both in a non-specific manner. Thus, the staff believes at this time, that levels of SO_2 to which consumers are exposed, will by necessity have to be obtained from the studies in homes such as that being performed by the Pierce Foundation in New Haven Connecticut.

9. J.C. Ribot et al (1982). Monitored Low Energy Houses in North America and Europe: A Compilation and Economic Analysis. LBL report 14788 for the U.S. Department of Energy.

10. American Gas Association (1983). Actual and Forecasted Conservation in the Residential Gas Space Heating Market through 1990. An Energy Analysis prepared by the American Gas Association.

11.

12. National Academy of Sciences (1981). Indoor Pollutants prepared by the Committee on Indoor Pollutants.

13. S.H. Dole (1975). Energy use and conservation in the residential sector: a regional analysis. Rand Corp. report for the National Sciences Foundation.

14. R. Chenshaw et al (1982). Optimal Westhirization of Law-Income Housing in the U.S.: A Research Demonstration Project.. A NBS report for the Community Services Administration.

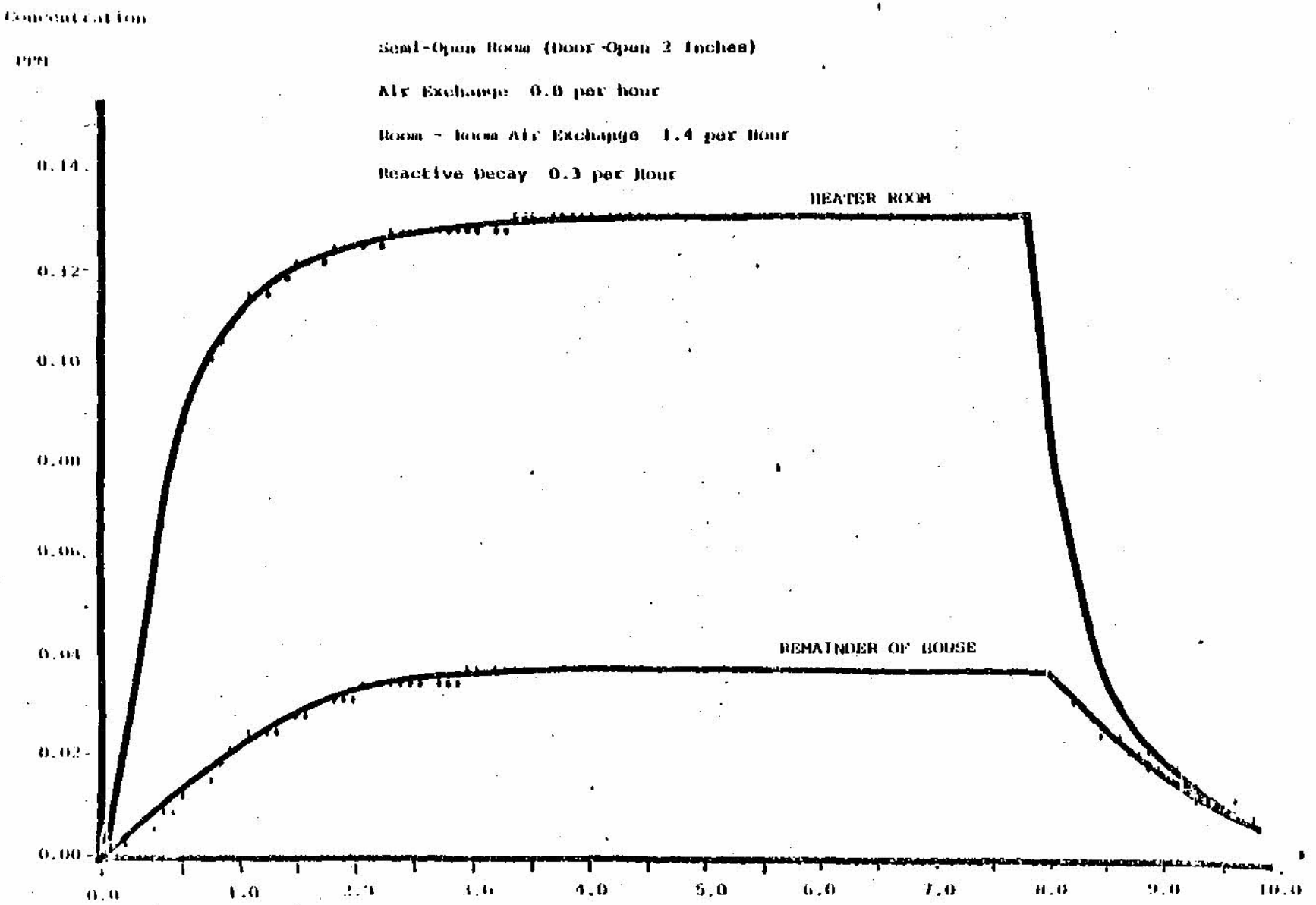


Figure 2 TWO COMPARTMENT MODEL FOR NITROGEN DIOXIDE 6800 BTU/HR BLUE FLAME HEATER

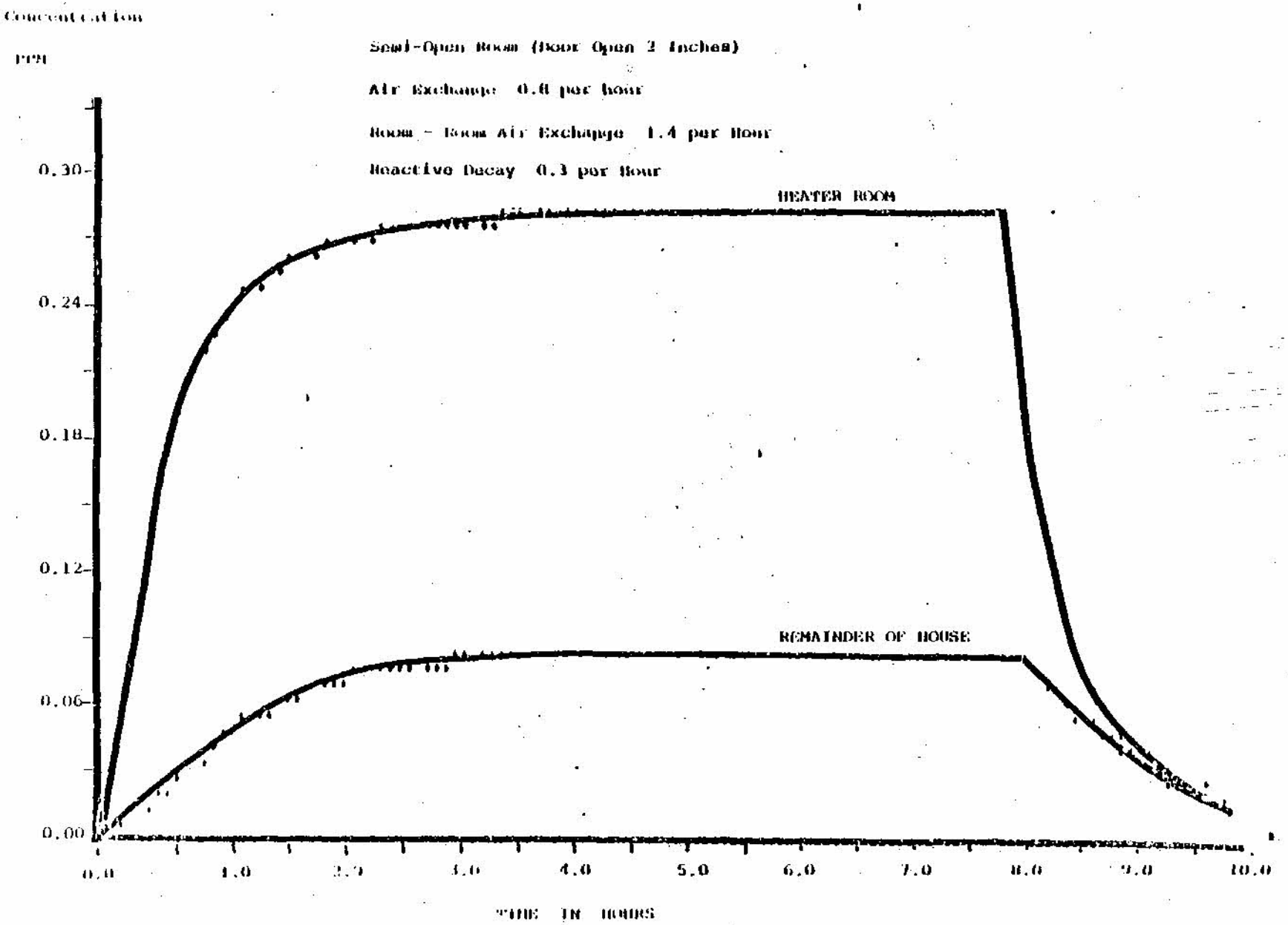


Figure 4 TWO COMPARTMENT MODEL FOR NITROGEN DIOXIDE 10400 BTU/HR WHITE FLAME CONVECTIVE

9. J.C. Ribot et al (1982). Monitored Low Energy Houses in North America and Europe: A Compilation and Economic Analysis. LBL report 14788 for the U.S. Department of Energy.

10. American Gas Association (1983). Actual and Forecasted Conservation in the Residential Gas Space Heating Market through 1990. An Energy Analysis prepared by the American Gas Association.

11.

12. National Academy of Sciences (1981). Indoor Pollutants prepared by the Committee on Indoor Pollutants.

13. S.H. Dole (1975). Energy Use and conservation in the residential sector: a regional analysis. Rand Corp. report for the National Sciences Foundation.

14. R. Chenshaw et al (1982). Optimal Westhirization of Law-Income Housing in the U.S.: A Research Demonstration Project.. A NBS report for the Community Services Administration.

TABLE 1: Kerosene Heater Emission Rates

Heater Number	Reactive Decay (hr^{-1})		Pollutant Emission Rates (cc/kJ)				
	NO	NO_2	NO	NO_2	CO	CO_2	O_2
F-805-3858	a	0.15	a	2.09×10^{-3}	0.13	40.39	-71.24
F-805-3858	a	0.21	a	2.07×10^{-3}	0.11	39.58	-55.04
F-598-0161	a	0.10	a	2.00×10^{-3}	0.13	45.77	-63.47
F-598-0161	a	0.11	a	2.40×10^{-3}	0.13	45.99	-61.72
F-598-0161(high)	a	0.13	a	2.14×10^{-3}	0.11	38.8	-52.9
F-598-0161(low)	a	0.09	a	2.79×10^{-3}	0.24	41.3	-49.2
F-805-3860	0.11	0.18	1.81×10^{-3}	2.39×10^{-3}	0.08	38.96	-51.02
F-805-3860	a	0.09	a	2.80×10^{-3}	0.20	40.08	-49.4
F-598-0160	a	0.03	a	2.76×10^{-3}	0.171	43.10	-55.30
F-598-0160	a	0.12	a	2.43×10^{-3}	0.118	40.40	-55.90
F-805-3862	0.13	0.14	1.01×10^{-3}	2.63×10^{-3}	0.09	39.97	-53.70
F-805-3862	0.06	0.11	1.04×10^{-3}	2.84×10^{-3}	0.10	42.54	-54.63
F-805-3859	a	0.11	a	3.29×10^{-3}	0.24	38.14	-50.78
F-805-3859	a	0.15	a	2.98×10^{-3}	0.25	40.67	-52.35
F-805-3861**	0.11	0.11	16.4×10^{-3}	3.87×10^{-3}	0.02	32.38	-42.56
F-805-3861**	0.24	0.18	18.0×10^{-3}	5.08×10^{-3}	0.02	35.31	-48.75
F-598-0162	0.06	0.04	16.1×10^{-3}	3.50×10^{-3}	0.03	41.32	-55.23
F-598-0162	0.07	0.18	16.4×10^{-3}	6.85×10^{-3}	0.04	42.02	-57.12
504	0.04	0.17	8.93×10^{-3}	3.08×10^{-3}	0.02	40.4	-51.0
504	0.07	-0.16	8.08×10^{-3}	2.31×10^{-3}	0.01	37.8	-51.9
504(low)	-	-	5.6×10^{-3}	2.5×10^{-3}	0.03	39.6	-48.1
504(high)	0.11	-0.18	10.2×10^{-3}	2.5×10^{-3}	0.01	39.6	-50.0
508	0.07	-0.19	11.02×10^{-3}	2.96×10^{-3}	0.03	42.8	-60.2
508	0.00	-0.06	8.66×10^{-3}	2.45×10^{-3}	0.01	36.8	-51.1
508(low)	-	-	5.8×10^{-3}	6.4×10^{-3}	0.04	44.3	-56.9
508(high)	0.01	0.02	12.3×10^{-3}	1.6×10^{-3}	0.01	44.4	-58.6
0181	0.06	-0.06	8.85×10^{-3}	3.18×10^{-3}	0.02	41.6	-58.1
0181	0.00	0.06	5.30×10^{-3}	4.98×10^{-3}	0.03	41.6	-55.4
510*	0.00	0.12	0.89×10^{-3}	1.85×10^{-3}	0.10	38.5	-50.3
510*	-0.04	0.10	2.02×10^{-3}	2.38×10^{-3}	0.08	43.1	-57.5
F-598-0160(low)	-	-	0.24×10^{-3}	2.50×10^{-3}	0.14	39.5	-50.0
F-598-0160(high)	0.08	0.08	1.93×10^{-3}	2.46×10^{-3}	0.09	39.5	-51.2

Notes: *Blue flame radiant with a catalytic deodorizer disc.

**Wickless heater

a) Nitric oxide levels were low and variable. No attempts were made to estimate emission rates of NO for these experiments.

TABLE 2 KEROSENE HEATER TEST DATA

	Air Ex. Rate CO_2	Air Ex. Rate O_2	Final Concentration (PPM) Predicted					Measured						
	CO_2	O_2	CO_2	CO	NO_2	NO	O_2(%)	CO_2	CO	NO_2	NO	O_2(%)	Output-kJ/hr	Date
F-805-3858(R)[1]	1.46	1.31	7331	25.3	0.36	0.05	20.0	7497	24.9	0.35	0.05	20.1	7268	12/13/82
F-805-3858(R)	1.52	1.55	7588	30.9	0.36	0.08	19.9	7486	30.1	0.34	0.08	19.9	7595	12/27/82
F-598-0161(R)	1.50	1.69	7510	26.3	0.37	0.08	20.0	7509	20.3	0.36	0.09	20.0	7387	12/20/82
F-598-0161(R)	1.54	1.52	7540	22.0	0.37	0.08	19.8	7510	21.3	0.35	0.08	19.9	6560	12/30/82
F-598-0161(high)	1.49	1.55	7334	20.7	0.36	a	19.9	7251	20.2	0.36	0.07	19.9	7420	3/10/83
F-598 0161(low)	1.52	1.48	5374	32.6	0.34	a	20.2	5405	31.7	0.33	0.01	20.3	5352	3/14/83
F-805-3860(HC)	1.45	1.48	8299	16.9	0.46	0.36	19.8	8135	16.9	0.44	0.35	19.9	8248	12/14/82
F-805-3860(HC)	1.52	1.51	7001	34.6	0.47	a	20.0	6857	33.6	0.43	a	20.1	6960	1/05/83
F-598-0160(HC)	1.62	1.57	6611	37.1	0.45	a	20.0	6207	26.3	0.47	a	20.1	6616	1/04/83
F-598-0160(HC)	1.50	1.53	7329	21.4	0.41	a	20.0	6592	19.6	0.37	a	20.0	6677	1/11/83
F-805-3862(R)	1.50	1.50	7501	17.6	0.46	0.18	19.9	7549	17.5	0.44	0.18	20.0	7252	12/16/82
F-805-3862(R)	1.50	1.47	8093	19.6	0.51	0.19	19.9	7727	19.2	0.46	0.19	19.9	7432	12/29/82
F-805-3859(HC)	1.51	1.49	12498	22.8	1.01	a	19.2	12500	76.5	0.91	a	19.3	12903	1/03/83
F-805-3859(HC)	1.56	1.58	13733	23.4	0.83	a	19.4	11879	23.4	0.79	a	19.4	13144	1/10/83
F-598-0162(HC)	1.55	1.56	7464	4.6	0.78	3.4	19.9	7940	5.6	0.64	2.77	20.1	7261	12/15/82
F-598-0162(HC)	1.59	1.59	7268	6.2	1.03	2.8	20.0	7067	6.0	0.98	2.65	20.1	7135	1/05/83
F-805-3861[2]	1.49	1.63	9643	4.8	1.04	4.75	19.7	9557	4.5	1.07	4.66	19.8	87421	12/28/82
F-805-3861	1.49	1.55	13825	6.9	2.54	6.89	19.1	13486	6.8	1.73	6.00	19.3	15201	12/31/82
508(HC)	1.59	1.62	15715	4.9	1.04	3.89	18.8	15088	4.3	0.96	3.81	18.9	15606	2/28/83
508(HC)	1.53	1.63	11964	3.5	0.80	2.78	19.3	11533	4.3	0.78	2.75	19.4	17781	3/2/83
508(HC)(low)	b	b	11145	10.9	1.20	1.07	19.3	12444	10.9	1.29	1.07	19.3	12332	3/16/83
508(HC)(high)	1.59	1.61	13230	8.2	1.39	2.23	19.1	13888	8.2	1.39	2.23	19.1	14928	3/16/83
504(HC)	1.59	1.56	10409	4.3	0.71	2.17	19.6	9975	4.3	0.62	2.1	19.7	10653	2/24/83
504(HC)	1.58	1.66	10573	3.6	0.68	2.05	19.6	9897	3.5	0.58	2.0	19.7	11063	3/3/83
504(HC)(low)	b	b	7176	5.6	0.67	1.03	20.04	7222	5.6	0.67	1.03	20.1	8009	3/15/83
504(HC)(high)	1.58	1.64	9053	2.9	0.51	1.95	19.7	9053	2.9	0.55	1.87	19.7	10012	3/15/83
510[3]	1.59	1.62	10223	25.7	0.46	0.23	19.6	9811	24.0	0.43	0.23	19.7	10941	3/1/83
510[3]	1.59	1.63	10564	18.5	0.53	0.50	19.6	10183	17.7	0.50	0.50	19.6	10095	3/9/83
F-598-018(HC)	1.57	1.65	10249	4.2	0.79	2.06	19.6	9948	5.0	0.75	1.96	19.6	10058	3/2/83
F-598-018L(HC)	1.59	1.64	9504	6.8	1.09	1.20	19.7	9168	6.4	1.04	1.17	19.8	9405	3/8/83
F-598-0160 (low)	b	b	6729	25.7	0.41	0.04	20.0	6560	22.9	0.40	0.04	20.1	6819	1/11/83
F-598-0160(high)	1.58	1.55	8058	21.6	0.53	0.41	19.7	8282	19.2	0.49	0.38	19.8	8343	1/13/83

Notes:

1) Blue flame radiant heaters = R; White flame convective heaters = WC; Blue flame convective heaters = BC

2) Wickless heater

3) Blue flame radiant heater with a catalytic "deodorizer".

a) Nitric oxide values were low and oxidation rates were not constant.

b) Air exchange rate was not determined on low fuel consumption rate portion of test since no burn period was present between the low to high rate tests.

Table 1. Analysis of Emission Rates

Heater Type	Number of Heaters	Emission Rate (cc/kJ)		
		Ave	Sx	% Variation
Radiant and convective, Blue Flame				
NO_2	7	2.5×10^{-3}	0.403×10^{-3}	16.2
CO	6	0.11	0.018	16.8
CO_2	7	41.2	1.76	4.3
White Flame convective, and Wickless				
NO_2	5	3.82×10^{-3}	1.29×10^{-3}	33.9
CO	5	0.02	0.005	26.1
CO_2	5	39.1	2.3	5.9

Table 4

Kerosene Heater Emission Rates Used in Modeling
(cc/kJ)

Typical Output	Blue Flame			White Flame			Wickless		
	# in Group	CO	NO_2 ($\times 10^{-3}$)	# in Group	CO	NO_2 ($\times 10^{-3}$)	# in Group	CO	NO_2 ($\times 10^{-3}$)
6800 BTU/h	5 Maltuned:	0.079 - .145 0.24	2.0 - 2.8 2.79	1	0.01	4.5	0	-	-
10,400	2	0.09 - .244	2.1 - 3.0	2 Maltuned:	0.02 0.05	2.7 - 4.1 3.5	0	-	-
15,600	0	-	-	1 Maltuned:	0.01 0.04	2.72 6.4	1	0.02	4.4

Table 5
Kerosene Heaters: Projected Pollutant Levels Under Different Models

	Typical size: 6800 BTU/h				Typical size 10,600 BTU/h					Typical size 15,600 BTU/h		
	Low emitting Blue flame	High emitting Blue flame	Nal-tuned Blue flame	White flame	Low emitting Blue flame	High emitting Blue flame	Low emitting White flame	High emitting White flame	Nal-tuned White flame	White flame	Nal-tuned White flame	Wick-less
					CO							
Closed room	2.2-21.2	11.3-38.6	22.1-64.3	1.0-6.0	13.0-24.3	35.4-65.6	2.9-5.4	2.9-5.4	4.4-8.1	1.4-2.7	5.0-10.8	2.0-3.4
Semi-open room	6.3-8.3	11.3-15.2	18.7-25	2.3-3.2	8.6-12.5	23.4-33.9	2.0-2.8	2.0-2.8	2.9-4.2	1.1-1.8	4.5-7.0	1.3-1.5
Open house	2.9-4.7	5.3-8.7	8.8-14.4	1.1-1.8	5.1-8.2	13.9-22.7	1.2-1.8	1.2-1.8	1.7-2.7	0.8-1.4	3.4-5.5	1.7-2.7
					NO_2							
Closed room	0.12-0.39	0.16-0.54	0.16-0.54	0.26-0.87	0.22-0.41	0.31-0.59	0.20-0.53	0.43-0.80	0.26-0.50	0.15-0.53	0.37-1.26	0.26-0.87
Semi-open room	0.10-0.16	0.14-0.22	0.14-0.22	0.23-0.35	0.15-0.21	0.22-0.30	0.20-0.27	0.31-0.41	0.19-0.25	0.16-0.32	0.39-0.76	0.27-0.52
Open	0.05-0.07	0.07-0.10	0.07-0.10	0.12-0.17	0.09-0.11	0.12-0.17	0.11-0.15	0.17-0.23	0.10-0.14	0.12-0.23	0.39-0.54	0.27-0.37

Table 5a
Kerosene Heaters: Projected CO_2 Levels Under Different Models[a]

House Model	Typical Heater Size		
	6800 BTU/hr	10,400 BTU/hr	15,600 BTU/hr
	(PPM)	(PPM)	(PPM)
Closed Room	3,700-10,700	5,800-10,800	5,800-10,800
Semi-Open room	3,100-4,200	3,800-5,600	4,500-7,000
Open House	1,500-2,400	2,300-3,600	3,400-5,500

a) Since Heaters consistently produced close to 40 cc of CO_2 per kJ output these data are based on the single source strength of 40^{+} cc/kJ for all types of heaters.

Table 6a

Durations of exposure to pollutants from an 8 hour Kerosene Heater Burn and 3 hour decay period

Pollutants	Heater	Model type	Peak level	Hours of exposure in concentration range									
				0-1000	1001-2000	2001-3000	3001-4000	4001-5000	5001-6000	6001-7000	7001-8000	8001-9000	9001-10,000
CO_2	6800 BTU/h typical small blue flame	open house	1520 ppm	3.8	7.1								
	6800 BTU/h typical small blue flame	semi-open room	3010 ppm	1.6	1.0	5.7	1.9						
	6800 BTU/h typical small blue flame	closed room	6450 ppm	1.0	1.1	0.8	0.9	0.8	1.7	4.9			
	10,400 BTU typical large white flame	open house	2270 ppm	2.6	2.8	5.6							
	10,400 BTU typical large white flame	semi-open room	4490 ppm	1.1	0.8	0.7	1.4	6.2					
	10,400 BTU	closed room	9630 ppm	0.4	1.0	0.7	0.6	0.5	0.6	0.6	0.7	1.3	4.8

Table 6b

Durations of exposure to pollutants from an 8 hour Kerosene Heater Burn and 3 hour decay period

Pollutants	Heater	Model type	Peak level	Hours of exposure in concentration range			
				0-5	6-10	11-15	16-20
CO	6800 BTU/h typical small blue flame	Open house	4.6 ppm	11.2			
	6800 BTU/h typical small blue flame	Semi-open room	9.2 ppm	2.4	7.8		
	6800 BTU/h typical small blue flame	Closed room	19.7 ppm	2.0	1.3	1.5	6.4
	10,400 BTU/h typical large white flame	Open house	1.0 ppm	11.2			
	10,400 BTU/h typical large white flame	Semi-open	1.9 ppm	11.2			
	10,400 BTU/h	Closed room	4.1 ppm	11.2			

Table 6c

Durations of exposure to pollutants from an 8 hour Kerosene Heater Burn and 3 hour decay period

Pollutants	Heater	Model type	Peak level	Hours exposure in concentration range					
				0.0-0.10	0.11-0.20	0.21-0.30	0.31-0.40	0.41-0.50	0.51-0.60
NO_2	6800 BTU/h typical small blue flame	Open house	0.06 ppm	11.1					
	6800 BTU/h typical small blue flame	Semi-open	0.11	2.9	7.1				
	6800 BTU/h typical small blue flame	Closed room	0.25	2.8	1.7	6.7			
	10,400 BTU/h typical large white flame	Open house	0.13	4.5	6.9				
	10,400 BTU/h typical large white flame	Semi-open	0.29	1.8	0.8	7.6			
	10,400 BTU/h typical large white flame	Closed room	0.56	1.7	0.9	0.7	1.1	6.1	

Appendix A

Ancillary Modeling Information

The information which follows is a compilation of our treatment of various models for heat load and mass transfer. This information is presented to permit the reader to follow the steps in our model development.

A. Calculation of room size for heater operation under the closed and semi-open models:

Take the heating requirements equation from Shair (terms regrouped).

$$Q_1 = (T_1 - T_o)(q_1 \rho c_p + U_1 A_1) + (T_1 - T_2)(q_{12} \rho c_p + U_{12} A_{12})$$

Now $q_1 \rho c_p + U_1 A_1$ is the external heat loss from room per hour, per $^{\circ}F$ difference with outside temperature. If L is defined as the loss rate ($BTU/ft^2 - {}^{\circ}F - hr$), $q_1 \; c_p + U_1 A_1 = A_{floor} \; L$

In Shair's living room calculation, the ratio of internaly exposed room area to floor area is $A_{12}/A = 514/250.8 = 2.05$; this will be assumed roughly constant for calculations here. Following Shair, U_{12} is taken as 0.293 $BTU/hr\text{-}ft^2 - {}^{\circ}F$ and ρc_p as 0.0185 $BTU/ft^3 - {}^{\circ}F$. The quantity $(q_{12} \rho c_p)(T_1 - T_2)$ is now caluclated using the formula supplied to CPSC by S. Winter Associates and adopted by D. Lavery for the modeling being performed by industry. The calculation is done for a door that is open two inches, the minimum recommended by several kerosene heater manufacturers. $(q_{12} \rho c_p)(\Delta T) = Q_c = 4.6 \times W \times (h \times \Delta T)^{3/2}$ where w is the width of the opening (2 inches), h is the height of the door (6.67 ft) and ΔT is the temperature differential between the heater room and

the remainder of the house. For the door opening described $Q_c = 13.2 ($ $T)^{3/2}$ The total heat loss due to losses to the outside from the heater room, losses through the interior walls of the heater room and losses through the partially open door is:

$$Q_1 = (T_1 - T_o)\, A_{floor}\, L + 2.05\, (T_1 - T_2)\, U_{12} + 13.2\, (T_1 - T_2)^{3/2}$$

where

To is the outdoor temperature

T_1 is the heater room temperature

T_2 is the temperature of the rest of the house

A_{floor} is the area of the floor of the heater room and

U_{12} is the conductive heat transfer through interior walls.

In the case of a closed room the equation remains the same except that the last term on the right side disappears.

Values for A_{floor} are calculated for each of the heater sizes modeled; as the equation gives two solutions for room size, the smaller room is selected for modeling. In these calculations a thermal balance is assumed, that is the heater output is equated to the equilibrium heat load requirement, Q_1. This is largely justified because heaters are often operated for prolonged periods of time, e.g. 8 hours. If thermal equilibrium is not reached because of an insufficiently long burn, a greater heater output than Q_1 is allowable without increasing the heater room temperature above the 70° value that is used in calculations here. Changing outdoor temperatures could result in heat load requirements or heater run periods that are either higher or lower than those presented here.

B. Calculation of steady state pollutant levels for different room geometries and emission rates:

Shair's equation for concentration of a pollutant is applied:

$$C_1 = \frac{(q_2 + q_{12} + k A_2^*) S_1 O_1}{(q_1 + q_{12} + k A_1^*)(q_2 + q_{12} + k A_2^*) - q_{12}^2}$$

For the semi-open room calculations we calculated q_{12} using the equation of Ingersoll. The terms $k A_1^*$ and $k A_2^*$ are the rates by which pollutants are removed by chemical reactivity in areas 1 and 2 respectively. These can also be written $k A_1^* = k_o V_1$ and $k A_2^* = k_o V_2$, where k_o is the reactive decay rate as derived in the LBL studies. It is taken as 0.3 hr^{-1} for NO_2 on the basis of the CPSC/LBL unvented gas heater studies in the LBL experimental home. Also if "a" is the air change rate (external) $q_1 = a V_1$, $q_2 = a V_2$.

Rewriting the equation for C_1,

$$C_1 = \frac{(a V_2 + q_{12}/V_2 \times V_2 + k_o V_2) S_1 O_1}{(aV_1 + q_{12}/V_1 \times V_1 + k_o V_1)(aV_2 + q_{12}/V_2 \times V_2 + k_o V_2) - q12^2 \quad /V_2 \times V_2}$$

Factoring of V_1 and V_2 reduces the above equation to:

$$C_1 = \frac{S_1 Q_1}{V_1} \times \frac{(a + q_{12}/V_2 + k_o)}{(a + q_{12}/V_1 + k_o)(a + q_{12}/V_2 + k_o) - q^2 12/V_2}$$

Using S_1 in units of cc/kJ, Q_1 in kJ and V_1 in m^3; C_1 is expressed in ppm.

In both the closed room and open house models the pollutant concentrations are given simply by:

$$C_1 = \frac{S_1 Q_1}{V_1 (a + k_0)}$$

Where V_1 is respectively the volume of the room or of the house.

C. Backup Data for Model Calculations

Case	(1) Ach	(2) To	(3) Tz	(4) L	(5) BTU/h	q_{12}	(6) Semi-Open A Floor	(6) Closed A Floor	C/S(7) Non-reactive Semi-Open	C/S(7) Non-reactive Closed Room	C/S(7) Non-reactive Open House	C/S(7) Reactive (K =0.3) Semi Open	C/S(7) Reactive (K =0.3) Closed Room	C/S(7) Reactive (K =0.3) Open House
I_6	0.8	0	50°	0.49	6,800	3.3	121	147	90	272	37	76	194	27
I_{10}	0.8	0	50°	0.49	10,400	2.0	199	225	118	270	57	97	196	41
I_{15}	0.8	0	50°	0.49	15,600	1.3	311	337	148	271	85	118	197	62
II_6	0.5	0	50°	0.24	6,800	2.0	195	236	105	267	60	78	167	37
II_{10}	0.5	0	50°	0.24	10,400	1.2	320	360	139	268	91	99	168	57
II_{15}	0.5	0	50°	0.24	15,500	0.80	500	541	176	268	137	119	167	85
III_6	0.8	32	60°	0.49	6,800	1.1	259	276	78	142	37	50	104	27
III_{10}	0.8	32	60°	0.49	10,400	0.68	405	422	96	145	57	75	105	41
III_{15}	0.8	32	60°	0.49	15,600	0.45	617	633	112	144	85	85	104	62
IV_6	0.5	32	60°	0.24	6,800	0.67	422	450	88	92	60	61	58	37
IV_{10}	0.5	32	60°	0.24	10,400	0.42	659	688	8	8	91	8	8	57
IV_{15}	0.5	32	60°	0.24	15,600	8	1003	1031	8	8	137	8	8	85

1. ach: external air change rate
2. To: outdoor temperature
3. T_2: indoor temperature external to the heater room, heater room temperature 70°.
4. L: the external heat loss rate to the house in BTU/ft^2 - °F - hr.
5. BTU/h: the output of the two typical heater sizes used in models.
6. A Floor: the caluclated floor area of the heater room to maintain 70° F.
7. C/S: the predicted pollutant concentration of ppm divided by source strength in cc/kJ.
8. Room size under these conditions judged to large for common home situations and pollutant levels were not modeled.

D. Temperatures in open house models:

To calculate expected heater-room temperature in the open home model we will use Shair's equation for temperature in the two compartment model, but with the restriction that indoor mixing be high enough so that pollutant levels in both zones are approximately equal. Specifically, using Shair's nomenclature we will require that:

$$0.8 = C_2/C_1 = q_{12} / (q_2 + q_{12})$$

Applying this to the living room case (Area = 250.8 ft^2), this relation implies that: $q_{12} = 19.4\ q_1$.

We can now solve Shair's equation for living room temperature under our mild temperature assumptions ($T_o = 32°$, $T_2 = 60°$) for a large heater ($Q = 13{,}900$). These are the conditions under which temperatures are highest among all of our open house calculations.

First note that $(q_1 \rho C_p + U_1 A_1)To$ is the external heat loss from the heater room and using our notation is equal to $L\, A_{floor} T_o$.

Using Shair's values for the various parameters:

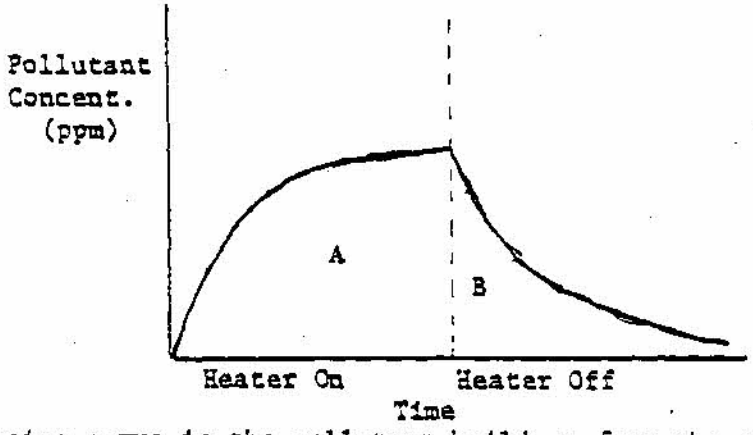
$$T_1 = \frac{Lx250.8x32 + (19.4\ q_1 x 0.0185 + 0.293x514) x\ 60 + Q_1}{Lx250.8 + (19.4\ q_1 \times 0.0185 + 0.293 \times 514)}$$

$$= \frac{8025L + 21.5\ q_1 + 9060 + 13{,}900}{250.8L + .36q_1 + 151}$$

The two heating requirement cases we have considered are a=.5 (q_1=1006), L=.24; and a=.8(q_1=1610), L=.49. Using the above formulas, the temperatures under the two cases are 81°F and 72°F. In the first instance, 81° temperature is a rather high, but not unreasonable, temperature. On a day with an outdoor temperature lower than 32° this predicted temperature would be lower.

E. Emission Rates from Kerosene Heaters

The typical pollutant level profile for a kerosene heater is shown below:



The rising curve is the pollutant build up from the combustion of kerosene. After the heater is turned off the pollutants decay by infiltrating air. In addition pollutants such as NO_2 and SO_2 also decay by non specific absorbtion or reaction. The theoretical equation describing region A is:

$$C = \frac{PaC_o + S/V}{k+a} (1-e^{-(k+a)t}) + C(o)e^{-(k+a)t}$$

1) where:

P = fraction of outdoor pollutants entering the chamber
a = air exchanges per hour
C_o = outdoor pollutant level (PPM)
s = source strength in cm^3/hour
v = volume in m^3
k = non specific decay (hr^{-1})
t = time in hours
c(o) = pollutant level in the chamber at the start of the experiment (ppm).

For the experiments performed the background C_o is the same as C(o) and by subtracting background concentration from the equation reduces it to:

2) $$C = \frac{S}{V(k+a)} (1-e^{-kt}).$$

For the decay region (B) the theoretical equation is:

3) $C(T+t)-C_o=[C(T)-C_o]e^{-(a+k)t}$

where:

$C(T+t)$ = concentration at t Hours beyond the heater shut off time T

$C(T)$ = Concentration at time of whut off of the heater

C_o = Background concentration

a = air exchanges per hour

k = reactive decay constant

Air exchange rates:

The air exchange rate in the chamber was determined by linear regression of ln C(T+t) versus t for the decay region for CO_2 and O_2 (k=0).

The slope of the line is equal to -a and the intercept is equal to ln C(T). For these procedures $C(T) >> C_o$ and C(o).

Reactive Decay Rates:

Similarly for reactive pollutants NO_x, a linear regression of ln C(T+t) versus t provides a slope equal to -(k+a) and an intercept equal to ln C(T). Knowing "a" from regression of non reactive pollutants k can be determined.

Source Strength:

Since our experiments were, with one exception, conducted to near steady state concentrations S in equation 2 was determined as follows:

At heater shut off C=C(T) at time T
therefore

$$C(T) = \frac{S}{V(k+a)} (1-e^{-(k+a)T})$$

dividing C(T) by $1-e^{-(k+a)T}$

results in the equilibrium or steady state chamber concentration (C_{ss}). That is the concentration where removal of pollutants by air exchange and reactive decay is equal to the rate of generation of pollutants from the heater. Thus:

$$C_{ss} = \frac{C(T)}{1-e^{-(k+a)t}} = \frac{S}{V(k+a)} .$$

Knowing the volume of the chamber ($24.9m^3$) and the calculated values of a, k, and C_{ss}, then $S = C_{ss} V(k+a)$.
The units of S are Cm^3/hr.

Appendix B

Survey Data on Kerosene Heater Use Patterns
with Reference to Pollutant Modeling

The National Kerosene Heater Association (NKHA) and Kerosun have both conducted telephone surveys of the home use patterns for kerosene heaters. The NKHA survey gathered responses from 300 heater users; the Kerosun survey gathered data from 349 users of the Radiant 10 heater (9,000 BTU/hr rated output) and 349 users of the OMNI 105 heater (18,500 BTU/hr rated output). Data from these surveys, as it pertains to questions relevant to the modeling of kerosene health pollutant levels in the home and assessing exposed population, is presented in the attachment.

The following inferences can be drawn from the survey data:

A. Use period

1) The predominant mode of use of the kerosene heaters is a single burn on days of use.

2) The reported daily use of heaters varies from short periods to essentially continuous operation (Kerosun results indicated roughly 11% of the users operated heaters 20 or more hours a day; NKHA data give a lower value of approximately 4%).

3) An average use time of 6 to 10 hours per day is indicated by the surveys, depending on time of year and day of the week. A value of 8 hours a day is approprate for representative modeling.

B. Character of room in which heater is used

1) Kerosene heaters are most commonly used in a room that might be called a family living area (including living rooms, dining rooms, kitchens, dens, and family rooms). Such rooms represent upwards of 80% of heater use.

2) Bedroom use occurs, but is uncommon (2 to 4 percent of respondents reported principal use in bedrooms; 3 to 8 percent reported some bedroom use). 2% or fewer respondents reported use while sleeping (Kerosun data only).

3) Heaters are used in rooms of a large variety of sizes. Rooms of moderate size (100 ft^2 to 300 ft^2 represented 52 to 69% of use situations. Use in small rooms (less than 100 ft^2) was rare (2 to 5% of respondents) and use in very large rooms (greater than 500 ft^2) was not typical (8 to 17% of respondents).

C. Ventilation

1) Typically heater use rooms had a source of external ventilation; 89 to 93% of users had open hallways or doors that were at least partially open. Roughly 20% of users had a window at least slightly open (NKHA data only).

2) The 7 to 11% of use situations with no open doors or halls are likely to be best represented by a closed room, single chamber, pollutant model (although it is possible that a larger portion of these users had open windows than the NKHA study group as a whole, the survey data do not address this possibility).

3) In the Kerosun survey 39 to 47% of respondents reported open doors but no open halls, depending on the degree that a door is open (unknown) these cases are appropriately represented by either semi-open room, two-compartment models, or open house single compartment pollutant models.

4) The 44 to 50% of use situations with open halls (Kerosun data only) are likely to be best represented by single compartment, open house pollutant models.

D. Home location and characteristics

1) Heaters see substantial use in all major climates and regions of the United States, however, use appears most concentrated in areas with moderate to cold climates. It's unknown to what extent the use patterns reported here are representative of the whole kerosene heater market.

2) The dominant proportion of reported use is in single family homes (90%, Kerosun data only). Multi-family appartment use was infrequent (1% or less, Kerosun data only). No data is available on extent of use in mobile homes.

3) The homes in the study were most often more than twenty years old, and 31 to 40% were older than 30 years. Homes less than 10 years old represented about 20% of the study populations.

4) The homes typically had storm windows and storm doors. About half of the respondents had added insulation to their homes (Kerosun data only). In the NKHA study 79% reported that their homes had insulated walls, however, as such insulation is not readily visible, respondents may have had difficulty with this question.

5) In summary, based on these two surveys, the typical house with a kerosene heater is not new, but has been fitted with storm windows and some degree of added insulation.

E. Other Data

A variety of additional interesting data was gathered in the surveys; some of this data is included in the attached tables.

Attachment: Data from the Kerosene Heater Use Surveys

The percentages of respondents giving various responses to selected questions posed in the NKHA and Kerosun surveys are tabulated below. The questions are jointly grouped under issues that they address. In many cases the response categories in the survey data have been more broadly grouped here to keep the tables brief. Additional calculations have been done in a few cases and are noted. Since "don't know" responses (usually rare) are not tabulated here, percentages do not add to 100%.

RES

INDOOR AIR QUALITY (IAQ) KEROSENE HEATER TESTING DOCUMENTATION

Engineering Sciences Document
543939

May 18, 1983

Prepared by Neil P. Zylich

The Engineering Laboratory
Directorate for Engineering Sciences
Consumer Product Safety Commission

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IAQ KEROSENE HEATER TESTING DOCUMENTATION

I. SCOPE

This IAQ Kerosene Heater Testing Documentation describes the kerosene heater test facility, the instrumentation, the data gathering system, and the procedures used to measure kerosene heater emissions at the CPSC Engineering Laboratory. The safety procedures and heater selection procedures are also described. It should be noted that the kerosene heater testing was accomplished in two phases. The first phase consisted of 20 tests on eight heaters and in the second phase 13 tests were performed on 6 heaters.

II. TEST FACILITY

A. Basic Chamber

The basic chamber used was a Hotpack, Model 10122-4, whose interior dimensions are 7 ft. high, 12 ft. deep and 10 ft. wide. Thus the test volume was approximately 840 cubic ft. The walls are three inches thick with 1/16 in. white enamel painted aluminum sheets on the inside and outside. Thermal insulation is between the aluminum sheets. The chamber was procured and installed in Bldg. 6 at the laboratory and modified for testing unvented gas space heaters. The modification related to reduction of the air changes per hour (ACH) to the lowest possible level (approximately 0.25 ACH).

B. Chamber Modifications for IAQ Testing

The chamber freon cooling systems were disabled for the kerosene heater testing since it was essential that nothing inside the chamber be below the dew point of the air in the chamber. Surfaces below the dew point condense and thus remove the reactive emissions being measured. The original chamber had egg-crate-type light diffusers and plexiglass sheets to direct the cooling air from the rear to the front of the chamber. The light diffusers and the plexiglass sheets were removed to prevent dead air space in the chamber.

II. TEST FACILITY CONT'D

C. Emission Sampling Manifolds

The chamber had in place a sampling manifold for use with the unvented gas space heater compliance test program (see Fig.1). This manifold was used for sampling CO, CO_2, and O_2. This manifold samples 4 different places in the chamber and is composed of copper tubing and a circulating air pump. Chamber air was drawn in at four sampling points, mixed in the manifold and pumped outside the chamber with a return that empties back into the chamber. Sample ports were provided on the line outside the chamber.

An additional sampling system was installed in the chamber to sample the NOx and SO_2 reactive gases (see Fig. 2). This system sampled the gases at four equally spaced points in the chamber through teflon tubing. The 4 tubes of equal length were brought outside the chamber to a teflon manifold. The gases were drawn from the manifold with a teflon pump using teflon tubing and pumped to a second teflon manifold which supplies the gases to the NOx and SO_2 instruments. A stainless steel twoway valve allowed the gases to be pumped either from the chamber or from a sample line outside the chamber (background air from the building). A flow adjustment valve downstream from the instrument manifold on the chamber return line allowed adjustment of the flow rate.

D. Air Changes per Hour Control System

A system was added to the chamber to control as closely as possible the air changes per hour during the kerosene heater testing to 1.5 (see Fig.3). Incoming air was cooled and dehumidified by an air conditioner and directed from above to the rear of the chamber. An air valve inside the rear of the chamber regulated the amount of air let into the chamber.

II. TEST FACILITY CONT'D

D. ACH Control System cont'd

A small blower was mounted at an opening on the left front door of the chamber. The exhaust from this blower was directed through a 4-inch flexible hose to an exhaust hood which conveyed the exhaust air outside (on top) of the building. A damper was used for adjustment of the air flow at the end of the flexible hose. The system was adjusted and calibrated for 1.5 ACH and slightly negative pressure during preliminary tests and was not adjusted during the actual testing.

E. Water Cooling System

A water cooling system was designed to cool the air in the test chamber during the kerosene heater testing (see Fig. 4). It was necessary to keep the temperature inside the chamber above the dew point during the testing to prevent absorption of the reactive gases (NOx and SO_2). The water cooling system had four finned heat exchangers, each having three muffin fans to circulate the air through the heat exchanger. A copper radiant cooling panel was also installed to remove the heat from radiant type heaters efficiently. The heat exchangers were connected to water supply manifolds inside the chamber with equal length 3/4-inch vinyl rubber hoses to provide equal flow in the four heat exchangers. Control valves outside the chamber provided control of the total flow to the four heat exchangers and the radiant panel. A circulating pump pumped the water through the cooling system from the mixing tank (a 55 gallon drum).

Cool tap water was brought into the system through the mixing tank to cool the air in the test chamber at the start of each test. However, the kerosene heater puts large amounts of water into the air as it burns (approximately 1 pound of water for each pound of fuel burned). Thus, the dew point was continuously rising during the test. To prevent condensation, the cooling water temperature was kept 2°C above the dew point by

II. TEST FACILITY CONT'D

E. Water Cooling System cont'd

recirculating the water through the system while adding a small amount of cool tap water. Precise temperature control is achieved with this cooling system.

F. Dew Point Monitor

A General Eastern Model 1100 DP Optical Dewpoint Hygrometer with Model 111D Sensor (0.2°C accuracy) was used to measure the dew-point in the test chamber. This dewpoint monitor uses the principle of optically detecting condensation (or dew point) on a thermo-electrically cooled mirror (see Fig. 5). The detection mirror is illuminated with a high intensity light-emitting-diode (LED). A photodetector monitors the specular (direct) component of the light from the mirror. A separate LED and photo detector combination are used to compensate for any thermally-induced changes in the optical components. The combination of the photo detectors in a bridge circuit, and an amplifier which controls the thermoelectric cooler form a system which maintains a thin layer of dew on the mirror. A precision thermometer is embedded in the mirror and measures the dew point temperature. Digital electronics read the temperature with a 3 1/2 digit readout which has a resolution of 0.1°C. The range of the dew point monitor is -40°C to +80°C. The sensor was mounted on the chamber wall, about 3 feet from the floor in the middle of the left wall of the chamber. The dew point hygrometer has National Bureau of Standards traceable certification of accuracy.

II. TEST FACILITY CONT'D

G. Temperature Measurement

The temperatures measured during the kerosene heater testing employed the use of Type J iron-constantan thermocouples The thermocouple output was read by a Molytek analog plotter and a Doric digital recorder (both to be described later).

The air temperature inside the test chamber was measured at four sampling line inlets. These 4 inlet temperatures being spaced around the chamber at different levels give an indication of the air mixing in the chamber, i.e., uniform temperatures indicate good mixing. The number and positions of the water cooling muffin fans were adjusted to provide a maximum temperature difference of 2°C among the four inlet temperatures. Two additional muffin fans were added to the front and rear of the chamber to provide sufficient air mixing, bringing the total muffin fans used to 14.

The temperature of the incoming water inside the chamber was measured for comparison to the dew point. The right front corner, right side wall (middle), and the floor (at the wall) temperatures were also monitored to make sure they were above the dew point during the kerosene heater test.

A thermocouple was placed on the top of the heater during each test to monitor the temperature at one of the heaters hottest areas. This temperature was compared to non-chamber testing levels to determine that the cooling air inside the chamber did not affect normal operation of the heater.

The temperature of the room where the instruments were housed was monitored with a thermocouple to insure that it is 85°F or slightly above. This keeps the instruments at a relatively constant temperature and prevents condensation of any gases in the sampling tubing or within the instruments themselves.

II. TEST FACILITY CONT'D

H. Explosive Vapor Detectors

The test chamber was equipped with two Scentry Gas Vapor Detectors, Models CH-405 and CH-415 manufactured by AQUA Meter Instrument Corporation. One sensor was mounted near the ceiling in the center of the test chamber and the other on the floor near the center of the room. The electronics and controls were in the instrumentation room. If, for any reason, an explosive mixture was present in the test chamber a warning buzzer would sound and alert the operators. The detectors had three levels of conditions. A green light indicated safe conditions; a yellow light indicated dangerous and a red light indicated explosive conditions in the test chamber.

I. Building G Considerations

The test chamber was located in Bldg. G. Since the incoming air to the test chamber came from inside the building the temperature of the building was kept at or near 65°F whenever possible. This provided some cooling air and the lower temperature allowed the building to dissipate some of the heat in the chamber during the testing. This was needed when testing very large heaters (over 18,000 BTU/hr) and on days when the humidity was high.

The temperature controlled instrumentation room was constructed of 2 X 4's, pressboard and plastic sheets to keep that area of the building at a constant temperature. The temperature was held at 85°F to prevent condensation in the sample lines and the emissions measurement instrumentation.

A "Princo" Standard Mercurial Barometer, U.S. Signal Corps type was used in Bldg. G to determine the barometric pressure at the beginning of each kerosene heater test. The barometer was manufactured by Princo Industries, 1020 Industrial Highway, Southampton, Pennsylvania 18966.

III. EMISSIONS INSTRUMENTATION, OPERATION AND CALIBRATION

A. Carbon Monoxide (CO) Measurement

The instrument used to measure carbon monoxide was a Lira Model 303 Infrared Analyzer manufactured by Mine Safety Appliances Company (MSA), Pittsburgh, PA 15208. The instrument uses the principle of infrared absorption of carbon monoxide to determine the amount of CO in air (oxygen and nitrogen do not absorb infrared energy). (See Fig. 6).

Two similar nichrome filaments are used as sources of infrared radiation. Using an optical bench, beams from these filaments travel through parallel optical paths. One beam traverses the sample cell; the other beam the comparison cell. The emergent radiation is directed to a single detector cell. As the gas in the detector absorbs radiation, its temperature and pressure increase. An expansion of the detector gas causes the membrane of a condenser microphone to move. This movement is converted to an electrical signal and amplified to produce an output signal.

The instrument was housed in an MSA portable cabinet which has provisions for continuously sampling via a pumping system (see Fig.7). The assembly also houses the carbon dioxide measuring instrument and a solenoid and piping arrangement to introduce zero and span calibration gases into the instruments. Flow control is via flowmeter needle valves located on the side of the cabinet. A four position mode switch controls the solenoid system and when in the off position provides power to a temperature control thermostat-heater assembly located in the optical bench of each instrument, which maintains the infra-red source cells and detectors at a constant temperature.

The range of the instrument used in the kerosene heater testing is 0-1000 ppm carbon monoxide. NBS Standard Reference Material Calibration gases of 97, 243.5 and 764 ppm were used at the beginning of testing,

III. EMISSIONS INSTRUMENTATION OPERATION AND CALIBRATION CONT'D

A. Carbon Monoxide (CO) Measurements cont'd

however, it was found that the levels generated by the kerosene heaters during the testing resulted in readings below 97 ppm. Additional calibration gas of 24.32 ppm was obtained for more precise calibration. The steps of the calibration procedure are described in Section D later since the CO, CO_2 and O_2 instruments were calibrated at the same time.

B. Carbon Dioxide (CO_2) Measurement

The carbon dioxide measuring instrument was similar to that used to measure carbon monoxide. It is also a Lira Model 303 Infrared Analyzer manufactured by Mine Safety Appliances Company (MSA), Pittsburgh, PA. 15208. The instrument uses the principle of infrared absorption of carbon dioxide to determine the amount of CO_2 in oxygen and nitrogen.

The instrument is housed in the MSA portable cabinet with similar controls to the CO instrument.

The range of the instrument is 0-12% carbon dioxide. NBS Calibration gases with concentrations of 1.4519, 2.872 and 9.02% CO_2 were used at the beginning of testing. After the first few tests it was found that the levels generated by the kerosene heaters were below 1.4519% CO_2. The steps of the calibration procedure are described in Section D below.

C. Oxygen (O_2) Measurement

The instrument used to measure the oxygen level in the kerosene heater test chamber was a Model 802 Magnetic Oxygen Analyzer manufactured by Mine Safety Appliance Company (MSA), Pittsburgh, PA. 15208. The instrument uses the principle that a paramagnetic wind is generated when a gas mixture containing oxygen is locally heated to produce a susceptability gradient in a non-homogeneous magnetic field (see Fig. 8).

The sensor in this paramagnetic wind type oxygen analyzer uses multiple electrically heated filaments. These filaments are sensitive to oxygen in the paramagnetic wind flowing around a circular cell. Four pairs of filaments are located circumferentially in each quadrant of a cylindrical housing. One element of

III. EMISSIONS INSTRUMENTATION OPERATION AND CALIBRATION CONT'D

C. Oxygen (O_2) Measurement cont'd

each pair is located in a magnetic zone of high field intensity and magnetic field gradient. Oxygen lowers the temperature in the wind producing filaments, and increases the temperature in the adjacent filament due to conductive heat transfer. A measuring bridge circuit is formed by the filaments and produces an output which is proportional to the temperature unbalancing thermomagnetic wind.

The instrument sampling line input is connected to the same manifold in the MSA portable cabinet assembly as the CO and CO_2 instruments. A flowmeter is used to control and indicate the gas flow to the oxygen measuring instrument. The instrument was left on continuously because normal warm-up time is 12 hours.

The range of the oxygen instrument was set at 0% to 25%. Room air was used to provide a calibration at 20.93% O_2. NBS Standard Reference Material Calibration gas of nitrogen with 9.56% O_2 was also used to check the linearity of the instrument. The steps of the calibration procedure are described in Section D below.

D. Calibration of CO, CO_2, and O_2 Instruments

Before and after each kerosene heater test the CO, CO2, and O2 instruments were calibrated in accordance with the following procedure:

1. Turn on MSA portable cabinet sampling pump which draws air from the chamber or from a background measuring sample line. The background sample line was located external to the chamber.

2. Admit background sample air from inside the building into the O_2 instrument.

3. Allow the O_2 instrument output reading on the "Doric" digital recorder to stabilize (two consecutive readings at the same level, readings are 1 minute apart). Then mark digital recorder output to indicate that stabilized reading has been achieved and also record time of reading on CO, CO_2, and O_2 time sheet.

4. Turn off MSA sampling pump and allow zero gas (ultra-pure nitrogen) in to the CO, CO_2 and O_2 instruments.

III. EMISSIONS INSTRUMENTATION OPERATION AND CALIBRATION CONT'D

D. Calibration of CO, CO_2, and O_2 Instruments cont'd

5. Allow the CO, CO_2, and O_2 output readings to stabilize and mark recorder output when stabilization is achieved. Record time of stabilization on time sheet.

6. Admit the 24.32 ppm CO calibration gas into the CO instrument and allow the output readings to stabilize. Mark recorder output and record stabilization time. Repeat for 97, 243.5 and 764 ppm CO calibration gases. The 24.32 ppm calibration gas was used late in the test program while the 243.5 and 764 ppm test gases were only used early in the test program.

7. Admit the 1.4519% CO_2 calibration gas into the CO_2 instrument. Allow readings to stabilize and mark recorder output. Record the stabilization time on the time sheet. Repeat for 2.872 and 9.02% CO_2 calibration gases. The 2.872 and 9.02% CO_2 calibration gases were not used after the early part of the test program.

8. Turn on the MSA cabinet assembly sampling pump to measure the background levels of CO, CO_2, and O_2. Mark recorder output and record time of stabilization.

9. The above Steps #2 through #7 were repeated after the kerosene heater test was over. However, Step #8 (the background sample) was done at the conclusion of the test prior to repeating Step #1.

E. Oxides of Nitrogen (NOx) Measurement

The oxides of nitrogen were measured with a Series 1600 Oxides of Nitrogen Analyzer manufactured by the Columbia Scientific Industries Corp., 11950 Jollyville Rd., Austin, Texas 78766. The instrument utilizes the photometric detection of chemiluminescent light resulting from the reaction of nitric oxide with ozone for measurement of nitric oxide (See Fig.9). The instrument includes an NOx to NO converter to change nitrogen dioxide into nitric oxide for subsequent measurement via the chemiluminescent detection method.

III. EMISSIONS INSTRUMENTATION OPERATION AND CALIBRATION CONT'D

E. Oxides of Nitrogen (NOx) Measurements cont'd

The chemiluminescent method of detection is based on the principle that nitric oxide (NO) reacts with ozone (O_3) to produce nitrogen dioxide (NO_2*) in an electronically excited state and oxygen (O_2).

The transition of electronically excited (NO_2*) to nitrogen dioxide (NO_2) is accompanied by photon emmission (hv). The reaction equations are:

$$NO + O_3 \rightarrow NO_2^* + O_2$$

$$NO_2^* \rightarrow NO_2 + hv$$

The intensity of this photon emission is proportional to the mass flow rate of NO into the reaction chamber where it is mixed with O_3. The photon emission is converted into an electrical output by a photomultiplier tube and associated electronics

The incoming sampled gas goes to a motorized 3-way valve. This motorized valve alternately allows the air sample to go to a NO_2 --> NO chemical converter or to bypass the converter and go directly to the reaction chamber. Thus, the input to the reaction chamber is either NO + NO_2 (converted to NO) which is defined as NOx or only NO (bypassed converter). The position of the 3-way valve is detected electronically and an electronic subtraction circuit provides a measurement of NO_2 as the difference between NOx and NO. The instrument has three electrical outputs NO, NO_2 and NOx.

The instrument was connected to the teflon sampling manifold for reactive gases. Either air from the chamber, Bldg. G background or calibration gases were directed to the instrument sample port through a "tee" and two on/off valves. A "tee" at the instrument allowed air flow past the sample port to a flowmeter then to the exhaust. Outside air was brought to the ozone generator through an air dryer.

The ranges of the NOx instrument are 0-1, 0-2, 0-5, 0-10, 0-20, and 0-50 ppm NO, NO_2, and NOx. The instrument was calibrated at 4.72, 9.48 and 19.33 ppm with NBS NO calibration gases. It was found early in the test program that very few heaters exceeded 4.72 ppm NO generation, therefore the 9.48 and 19.33 ppm calibration gases were not used after that time. The steps of the

III. EMISSIONS INSTRUMENTATION OPERATION AND CALIBRATION CONT'D

E. Oxides of Nitrogen (NOx) Measurement cont'd

calibration procedure performed before and after each kerosene heater test are as follows:

1. Turn on Ozone generator and let warm up for 15 minutes before proceeding to the next step.

2. Admit zero gas (pure nitrogen) to NOx instrument and determine zero readings on "Doric" digital output. The instrument range should be set on 0-1 ppm. Allow output to stabilize for two consecutive readings at the same level and then mark digital recorder output to indicate that stabilized readings have been achieved.

3. Repeat Step 2 for instrument range settings of 0-2, 0-5, 0-10, and 0-20 ppm NO (it was found that after testing a few heaters it was not necessary to do the 0-10 and 0-20 ppm calibrations since the heaters did not generate more than 5 ppm NO, NO_2 or NOx).

4. Admit 4.72 ppm NO calibration gas to the NOx instrument with the instrument range set at 0-5 ppm and allow output readings to stabilize. Then mark digital recorder output when stabilization is achieved.

5. Repeat Step 4 for NO calibration gases of 9.48 and 19.33 ppm and instrument range settings of 10 and 20 ppm (this step was not performed after the early part of the test program because it was found that the heater did no generate higher than 5 ppm NO, NO_2, or NOx).

6. Admit background sample air to the NOx instrument to determine the background levels of NO, NO_2 and NOx. Mark "Doric" digital output when stabilization is achieved.

7. The above Steps #1 through #5 were repeated after the kerosene heater test was completed. The background sample air (Step #6) was repeated immediately after the test prior to repeating Step #1.

III. EMMISSIONS INSTRUMENTATION OPERATION AND CALIBRATION CONT'D

F. Sulfur Dioxide (SO_2) Measurement

Sulfur dioxide was measured with a Model SA700 Fluorescent Sulfur Dioxide Analyzer manufactured by Columbia Scientific Industries Corp., 11950 Jollyville Rd., Austin, Texas 78759. The instrument uses the fluorescent technique which involves the excitation of SO_2 molecules by an ultraviolet source and the detection of the resultant fluorescence as the molecule returns to its original state (see Fig. 10). An SO_2 calibration source employing an NBS traceable certification permeation tube to provide gas samples was used to calibrate the SO_2 analyzer. The calibration source used was a Model CS10-ST TEMPERFLOW* manufactured by Columbia Scientific Industries.

The SO_2 analyzer is based on the principle that when a molecule of sulfur dioxide is irradiated with particular wave lengths of ultraviolet (UV) light, the molecule will absorb the light energy. Within nanoseconds, the excited molecule releases this energy in the form of light energy of longer wave lengths. An ultraviolet lamp is operated in the continuous wave mode to provide the excitation light. The excitation energy is selected by an interference filter. A UV detector controls the lamp output to a constant intensity. The sampled air is drawn through a membrane dryer to remove water and a hydrocarbon scrubber to remove aromatic hydrocarbons prior to passing into the reactor cell. The UV light enters the reactor cell in a concentrated beam and is absorbed by the sulfur dioxide. A photomultiplier tube detects the fluorescing light from the sulfur dioxide. The current output of the photomultiplier tube is converted to a voltage via an electrometer amplifier, which provides the signal outputs for the instrument.

The instrument was connected to the teflon reactive gas sampling manifold. Air from either the background (Bldg. G), the chamber or calibration gas was directed to the instrument. The ranges of the SO_2 instrument are 0-250, 0-500, 0-1000, 0-5000 and 0-10,000 ppb. The calibration source provided a 200 ppb calibration gas. During the test program the instrument was operated on the 0-250 ppb range for all tests except one. The steps of the calibration procedure performed before and after each kerosene heater test are as follows:

*Trademark

III. EMISSIONS INSTRUMENTATION OPERATION AND CALIBRATION CONT'D

F. Sulfur Dioxide (SO_2) Measurement cont'd

1. Check SO_2 calibration gas generator to see that it is operating at proper temperature and flow rate.

2. Check SO_2 instrument for proper condition of UV lamp, temperature control ovens and rotometer for air flow.

3. Run instrument system tests #1, #2 and #3 on range function selector switch. This checks the amplifier, the UV lamp current and the photocell output.

4. Set the range of the instrument to 0-250 ppb.

5. Set air system to bring in background air and adjust for zero positive flow if necessary.

6. Set instrument to generate "zero air" and allow output reading to stabilize. Adjust zero after stabilization if necessary. Mark digital recorder output when stabilization is achieved.

7. Admit 200 ppb SO_2 calibration gas into the instrument and place in the span mode. Allow output readings to stabilize and mark recorder output.

8. Repeat Steps #6 and #7.

9. Admit background sample air to the SO_2 instrument to determine the background level of SO_2. Mark recorder output when stabilization is achieved.

10. Steps #1 through #8 were repeated after the test of the kerosene heater was completed. Step #9 to measure the background level was repeated immediately after the test.

III. EMISSIONS INSTRUMENTATION OPERATION AND CALIBRATION CONT'D

G. Formaldehyde (HCHO) Measurement

The measurement of the amount of formaldehyde in the air during the kerosene heater testing was performed by a TGM 555 Air Monitor manufactured by CEA Instruments, Inc., 15 Charles St., Westwood, N.J. 07675. The instrument uses an automated wet-chemical colorimetric analyses technique to measure the formaldehyde in the sampled air (see Fig. 11).

The TGM 555 Air Monitor measures the concentration of formaldehyde in the air by monitoring the amount of color change produced by the instruments reagent system. The air to be analyzed is drawn into the instrument via an internal pump through a glass gas/liquid contact absorber coil. Reagent #1 (Sodium Sulfite in water) and reagent #2 (Mercuric Chloride and Sodium Chloride in water) absorbs the formaldehyde as the air passes through the coil. The absorbed reagents continue along the system where a third reagent (pararosaniline chloride in hydrochloric acid) is added. The reagents then pass through the mixing coils into the sample cell. The dual beam colorimeter measures electronically the formation of the color from the addition of the reagents and determines the color difference from the unreacted reagent that is pumped through the reference cell. The electrical signal, generated by a matched set of photodetectors in the colorimeter, is amplified and fed to a digital display.

The formaldehyde instrument was calibrated with a Model SC-100 Calibrator manufactured by CEA Instruments, Inc., 15 Charles St., Westwood, N.J. 07675. The calibration source uses an NBS traceable certification permeation tube to supply formaldehyde gas samples at known levels.

The range of the formaldehyde instrument is adjustable from 0-0.15 up to 0-10 ppm full scale. The instrument was setup for 0-1 ppm (0-1 Volt output) range when used to measure the kerosene heater output. The calibration procedure for the formaldehyde instrument is as follows:

III. EMISSIONS INSTRUMENTATION OPERATION AND CALIBRATION CONT'D

G. Formaldehyde (HCHO) Measurement cont'd

Calibration Procedure Performed Weekly:

1. Flush instrument thoroughly with distilled water for 30 minutes.
2. Flush with 1N nitric acid for one hour.
3. Flush with distilled water for one hour.
4. Put fresh reagents in instrument and admit zero air into the instrument through the zero air filter and allow to stabilize 30 minutes.
5. After a stable reading is obtained, set the electrical zero of the instrument.
6. Admit zero air from the CEA Model SC-100 calibrator into the sample intake of the instrument and allow to stabilize (this is double check of the instrument zero)
7. Admit span gas of about 0.4 ppm formaldehyde from the CEA calibrator into the sample intake of the instrument and allow to stabilize.
8. Adjust instrument electrical span reading to agree with calibrator output (about .4 ppm).
9. Observe instrument output reading for at least 10 minutes and readjust the electrical span if necessary.
10. Admit zero air from calibrator to the instrument and allow to stabilize.

Calibration Procedure Performed On Days of Test

1. Put fresh reagents in instrument.
2. Admit zero air from the CEA Calibrator and check zero reading. Instrument is now ready for measurement of kerosene heater output.
3. After completion of test remove reagents and flush system with distilled water.

IV. DATA COLLECTION SYSTEM

A. General

The data collection system monitored the temperatures and dew point inside the chamber and recorded the electrical output of the kerosene heater emission measuring instrumentation during calibration and testing (See Fig. 12). The system has the capability of 32 channels, however only 23 channels were used (see table 1 for the assignment of channels).

B. Multipoint Digital Plotter

Real time plotting of the digital data was performed by a Molytek Model 2701 Strip Chart Recorder manufactured by Molytek, Inc., 2419 Smallman Street, Pittsburgh, PA. 15222. The programable Molytek uses digital components to plot 32 channels in less than 10 seconds. The plotter was used to plot about 15 channels in about 6 seconds (a rate of 6 sec/point) during the kerosene heater testing. It was valuable in determining when a parameter has stabilized and tracking the dew point temperature versus the chamber internal temperatures. A built-in precision reference source automatically checks both zero and full scale every two seconds and makes adjustments if needed. Standard thermocouples were directly connected to the Molytek which has internal linearization and dynamic cold junction compensation. Left and right margins are programable and the instrument has five linear ranges for 1 millivolt to 10 volts. All strip charts were retained for inspection if required.

C. Digital Data Logger

The electrical signal level in volts (or millivolts) of the outputs of each emission measuring instrument was measured and recorded once a minute on paper tape by a programable Digitrend 235A Data Logger manufactured by Doric Scientific, 3883 Ruffin Rd., San Diego, CA. 92123. The data logger also linerizes thermocouple inputs. The Digitrend 235A uses an 8-bit NMOS microcomputer chip to provide monitoring, display and logging capabilities.

IV. DATA COLLECTION SYSTEM CONT'D

C. Digital Data Logger cont'd

All 23 of the channels used by the data collection system during calibration and test of the kerosene heaters were logged and converted into ASC-II code and transmitted to a microcomputer through an RS-232C communications port. All paper tapes generated are retained for analysis if needed.

D. Data Storage Microcomputer

An IBM personal computer is used to record the 23 channels of calibration and test data collected each minute by the data logger. The microcomputer stores the data on a floppy disk. After completion of the test and post calibration, a copy was made of the data on a second disk to insure preservation of the original test data. This microcomputer is then used to recall the data from the disk and transmit it through an RS-232C communications port to the CPSC main-frame computer at Parklawn via a telephone modem. The data were stored in files at the main-frame for later processing by the Health Sciences Staff. Log books were kept at the Engineering Laboratory which recorded for each test run, the following information:

1. The disk on which the data are stored at the laboratory.
2. The file name under which the data are stored on the disk.
3. The file name under which the data are stored at the Parklawn Main-frame Computer.
4. Miscellaneous information such as the start and stop times of each test.

V. TEST PROCEDURES

A. Fuel, Mixing and Analysis

Sixty gallons of prepackaged 1K kerosene was purchased. The fuel came in 5 gallon cans. Forty-five gallons of this 1-K kerosene was mixed together in a clean, dry, and empty new 55-gallon drum for use during the IAQ Kerosene Heater Testing Program.

A sample of the prepackaged 1K was sent to the U.S. Testing Co., Inc., 1415 Park Ave., Hoboken, N.J. 07030, for analysis. A sample of locally obtained kerosene (from a bulk tank) was also sent for comparison.

V. TEST PROCEDURES CONT"D

A. Fuel, Mixing and Analysis Cont'd

The samples were given the following tests according to ASTM Procedures:

1.	Distillation Range	ASTM D-86
2.	Sulfur Content	ASTM D-1266
3.	Flash Point	ASTM D-56

The results of the tests are attached. The only significant difference in the two kerosene samples was the sulfur content of 0.01% for pre-packaged 1K kerosene and 0.03% for the local bulk kerosene.

B. Chamber Calibration

Several mixing tests were performed on the chamber prior to the start of Phase I testing. The most significant test was conducted using a small kerosene heater (less than 10,000 BTU/hr.). The heater was allowed to stabilize (run for 2 hours) in the chamber and then the teflon sample lines were connected to the CO_2 instrument one at a time and the resulting levels noted. This was repeated again and the full manifold was connected to the CO_2 instrument for comparison. All readings were within 3% of the average.

The throughput of the sample lines were measured using CO for the non-reactive gas sampling manifold and NO for the reactive gas manifold. This test was performed prior to the start of Phase I and after Phase I. Three of the four sample lines were blocked and the normal NBS certified calibration gas was admitted to the fourth line. The gas measuring instrument should then read the same level as when the calibration gas was admitted directly into the instrument. The results showed that all lines tested were within 5% of the direct calibration level.

Prior to Phase I of the IAQ Kerosene Heater testing, the chamber was tested with gas inputs of carbon dioxide and nitrogen dioxide. The gases were put into the kerosene heater test chamber at known, constant emission rates for two hours to simulate a test run. The CO_2 and NO_2 instrumentation was operated as it would in a normal test run including the one hour decay period. From this test the chamber calibration with respect to CO_2 and NO_2 was determined.

Another chamber calibration was performed between the Phase I and Phase II testing. Sulfur dioxide and carbon dioxide were put into the test chamber at calibrated emission rates to simulate a heater test. The SO_2 and CO_2 instruments were operated using the normal test procedures.

V. TEST PROCEDURES CONT'D

C. Log Books and Log Sheets

A log book was maintained for each kerosene heater tested which contained a record of the date, the test used for, the hours operated and the total hours of operation. Each IAQ kerosene heater test had an emissions test data sheet on which was recorded the conditions underwhich the test was run. Also a check list was filled out prior to each test run. During the test a log sheet for CO, CO_2, and 0_2 times of pretest calibration, test run start and stop and post test calibration was kept by the instrument operator. Samples of these three log sheets are attached.

D. Heater Burn In and Pre-Burn

Prior to the start of the IAQ Kerosene Heater testing a pilot program was conducted to determine how many hours of operation were required before the CO, CO_2, and 0_2 emission rates and fuel consumption rate stabilized. Two heater's emissions were evaluated at the beginning of the burn-in test and every 5 hours of operation thereafter until a total of at least 30 hours of operation had been completed. The results of the heater burn-in study show that the CO, CO_2, and 0_2 emissions and fuel consumption rate all stabilized after 20 hours of operation. Subsequently, all kerosene heaters tested in the IAQ program were operated a minimum of 20 hours before the emissions tests were run.

Prior to the start of each test all kerosene heaters were allowed to warm-up a minimum of 15 minutes to allow the heaters to thermally stabilize.

E. Fuel Consumption

The fuel consumption of the kerosene heaters was measured during the testing in two ways. First, all heaters were weighed before and after the heater test (after heater had been shut-off). The time the heater was weighed before the test (while burning) was recorded as well as the time that the heater was shut-off. Secondly, the fuel consumption rate was monitored by a load cell in the chamber during the test. The load cell supported the heater during the test and generated an electrical signal through an Instron Electronic System which was recorded by the data collection system. Early in the

V. TEST PROCEDURES CONT'D

E. Fuel Consumption Cont'd

IAQ Kerosene Heater Test Program efforts to monitor the fuel consumption with a different system were unsuccessful, however in Phase II good results were obtained with the Instron system.

F. Pre-test Calibration

Prior to each test run the emissions measuring instrumentation was calibrated using the steps outlined in Section III above. There were four instrumentation operators and a test director present during this portion of the test. One operator for CO, CO_2, and O_2 instruments and one operator for each of the other three instruments, i.e., NOx, SO_2 and HCHO. The test director and all instrument operators were from ESEL except the SO_2-instrument operator who was from the Directorate of Health Sciences. Once every minute all 23 channels of data were recorded by the data collection system during the pre-test calibration and the system was on for the remainder of the test. This portion of the test usually required about 1 hour to complete.

G. Test Run

The final preparation for the test run began with the placing of the warmed-up kerosene heater in the test chamber on the load cell. The water-cooling system was turned on prior to putting the heater in the chamber to provide cooling water to precool the chamber to 65°F. The ACH air conditioner, air removal blower and exhaust hood blower were all on at this time to provide 1.5 ACH. Also on were the 14 air-mixing and cooling muffin fans. The heater was instrumented with a thermocouple on one of its hottest areas and a remote shut-off mechanism that was operated from outside the chamber door. For some tests the heater was also instrumented with a remote means for burner adjustment which changed the heater output from outside the chamber. The above preparation was accomplished in 2 minutes or less.

The actual test run began when the door of the chamber was closed and this was the time noted as the start of the test run. Normally, the heater is run for 2 hours minimum since it was found in preliminary tests that at 1.5 ACH the chamber gas levels stabilize after 1 hour and

V. TEST PROCEDURES CONT'D

G. Test Run Cont'd

45 minutes of operation. However, if the average temperature of the air inside the chamber exceeded 85°F the heater was shutoff. The test director, the instrumentation operators and a water-cooling system operator were present during this part of the test.

After build-up stabilization was achieved (2 hours) the heater was remotely turned off and the gas levels in the chamber were allowed to decay for 1 hour. During this time all chamber conditions were held constant, i.e., the water-cooling, the ACH air removal blower and the ACH exhaust hood blower were left on. The end of the decay phase of the test completed the test run.

H. Post Test Calibration

After each test run the emissions measuring instrumentation was calibrated again using the steps in Section III above. This portion of the test required about 1 hour to complete. The heater was weighed during this portion of the test.

VI. SAFETY PROCEDURES

A. General

The testing of kerosene heaters presents several potential hazards in the storage and handling of the kerosene and the kerosene heaters. The two main areas of concern are the protection of personnel and of the equipment.

B. Responsibility

The Laboratory Director has the overall responsibility for personnel and equipment safety for the Engineering Laboratory. The Laboratory Safety Officer has the responsiility to the Laboratory Director for reviewing and inspecting the overall laboratory procedures with respect to safety. During the IAQ Kerosene Heater tests, the Test Director has the responsibility for insuring the safety of the test, including enforcement of safety procedures, instruction of personnel, and assignment and delegation of safety tasks.

VI. SAFETY PROCEDURES CONT'D

C. Procedure

The hazards associated with IAQ Kerosene Heater testing are fire, explosion and thermal burns. Personnel involved with the testing were made aware of the basic laboratory safety procedures and, made aware of the following specified hazards presented by the testing of kerosene heaters.

D. Fire Extinguishers

Two Class B type fire extinguishers were kept in the general vicinity of the conditioning room. They were kept a minimum of 10 feet apart so a single fire would not likely block access to both extinguishers. At least one extinguisher was kept outside the test chamber door.

E. No Smoking

Smoking in the areas of the conditioning room and outside the chamber in Bldg. G was strictly prohibited. This precaution was necessary because of the flammable liquids present and because smoking produces formaldehyde and other gases which could effect the test results.

F. Fuel Storage

All bulk storage of kerosene was kept outside of the building on the north side out of the direct rays of the sun.

G. Fueling of heaters

All heaters were fueled outside of the building. Spillage of fuel is difficult to fully remove and constitutes a potential fire hazard from ignition sources that are present such as kerosene heaters and matches.

H. Operation of Heaters

Heaters were generally operated inside the chamber chamber and at one other location (conditioning area) which was determined by the test operator. No combustibles of any type were allowed within 3 feet (measured in any direction) of an operating kerosene heater.

VI. SAFETY PROCEDURES CONT'D

I. Pressurized Cylinders

1. Explosion of the cylinders and accidental knocking off of the valve, causing the cylinders to become a projectile, are real possibilities. Cylinders were stored in an upright position and were securely fastened so they could not be inadvertently knocked over.

2. Ignition of the contents of the cylinders was also a consideration. Flammable gases were stored outside of the building.

3. Toxicity of the contents of the cylinder was also taken into account. Even if not in itself toxic, inert gases such as nitrogen can displace available oxygen with resultant injury. Many gases are odorless and tasteless and the person breathing them is unaware that available oxygen has been cut off until they fall unconscious.

VII. HEATER INFORMATION

A. Heater Selection and Characteristics

The kerosene heaters used in the IAQ Kerosene Heater Test Program were selected by the Division of Special Studies of the Directorate of Economic Analysis. An effort was made to get a cross-section of types of heaters available to the consumer. For Phase I of the test program 8 heaters were purchased from retail outlets. The heaters tested were 3 blue flame radiant heaters, 3 blue flame convective heaters, 1 white flame convective heater (all wick type) and 1 wickless convective heater. The sizes ranged from 8,700 to 19,500 BTU/hr. rated output. Twenty tests were performed on the 8 heaters.

In Phase II of the test program, 6 heaters were tested. Four not used in Phase I, and 2 that had been used in Phase I. Three blue flame radiant heaters and 3 white flame convective heaters, all wick type were evaluated in the 13 tests performed in Phase II. The rated output of the 6 heaters ranged from 9,600 to 22,600 BTU/hr.

VII. HEATER INFORMATION CONT'D

B. Heater Number Assignment

All heaters tested in the IAQ Kerosene Heater test program were assigned a number prior to the testing. In some cases, the heater was assigned a sample number by the local resident investigator when collected. In other cases the Engineering Laboratory assigned a sample number upon receipt of the kerosene heater. Each heater was referred to and identified by the sample number throughout the test program, including identifying the assigned logbook.

C. Test Order

The order in which the heaters were tested was randomized to make the test results as accurate as possible. The random order was determined by Health Sciences prior to the start of testing.

DESTROY

LIST OF FIGURES

Fig.	1	CO, CO_2 and O_2 Emission Sampling Manifold
	2	NO_x and SO_2 Emission Sampling Manifold
	3	Air Changes Per Hour (ACH) Control System
	4	Water Cooling System
	5	Dew Point Hygrometer
	6	CO, CO_2 Infrared Type (Lira 303) Instrument
	7	CO, CO_2 and O_2 Gas Control System
	8	O_2 Paramagnetic Wind Type Instrument
	9	NO_x Chemiluminescent Type Instrument
	10	SO_2 Fluorescent Type Instrument
	11	HCHO Wet-Chemical Clorimetric Type Instrument
	12	Data Collection System

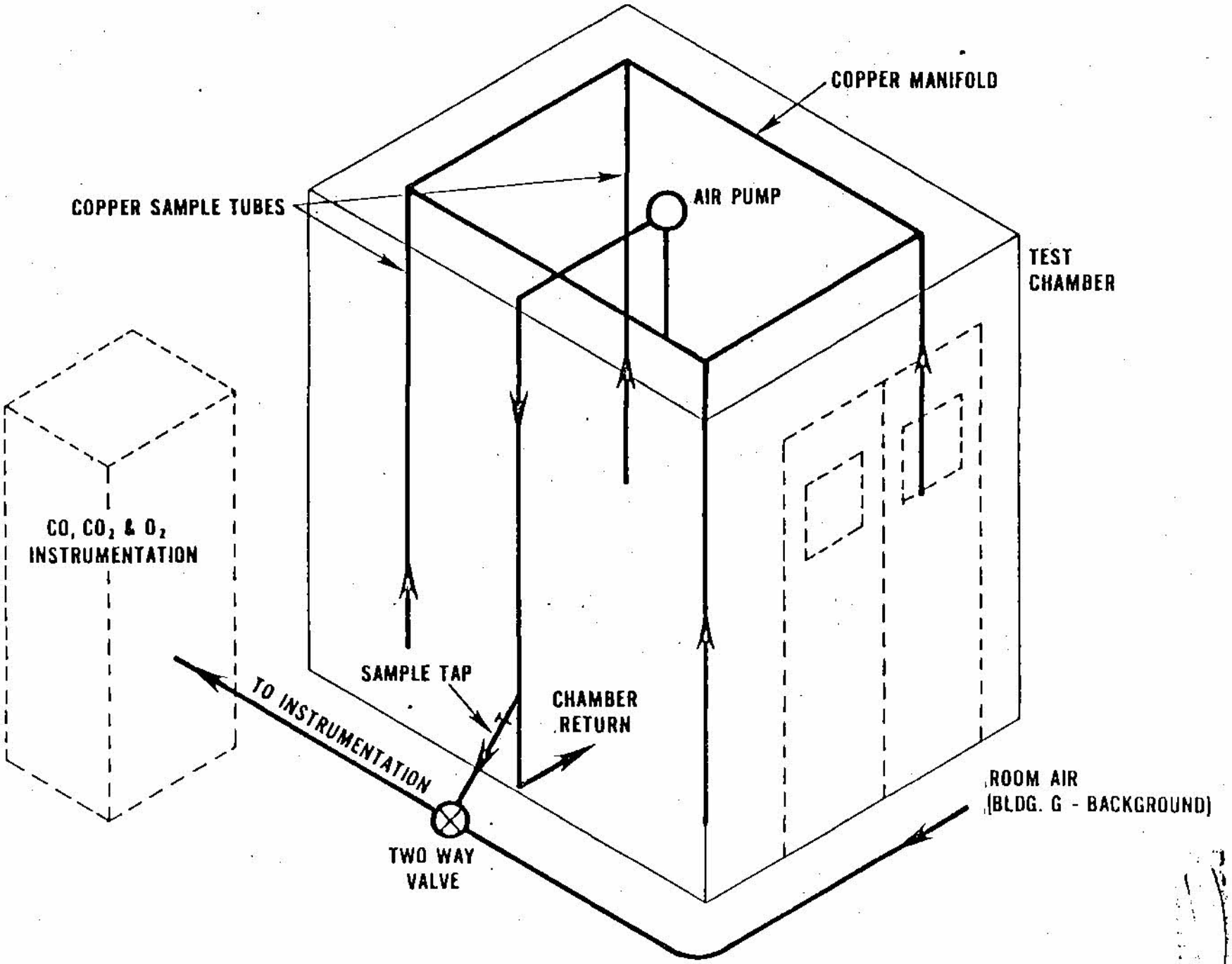


FIGURE 1 - CO, CO_2 & O_2 EMISSION SAMPLING MANIFOLD

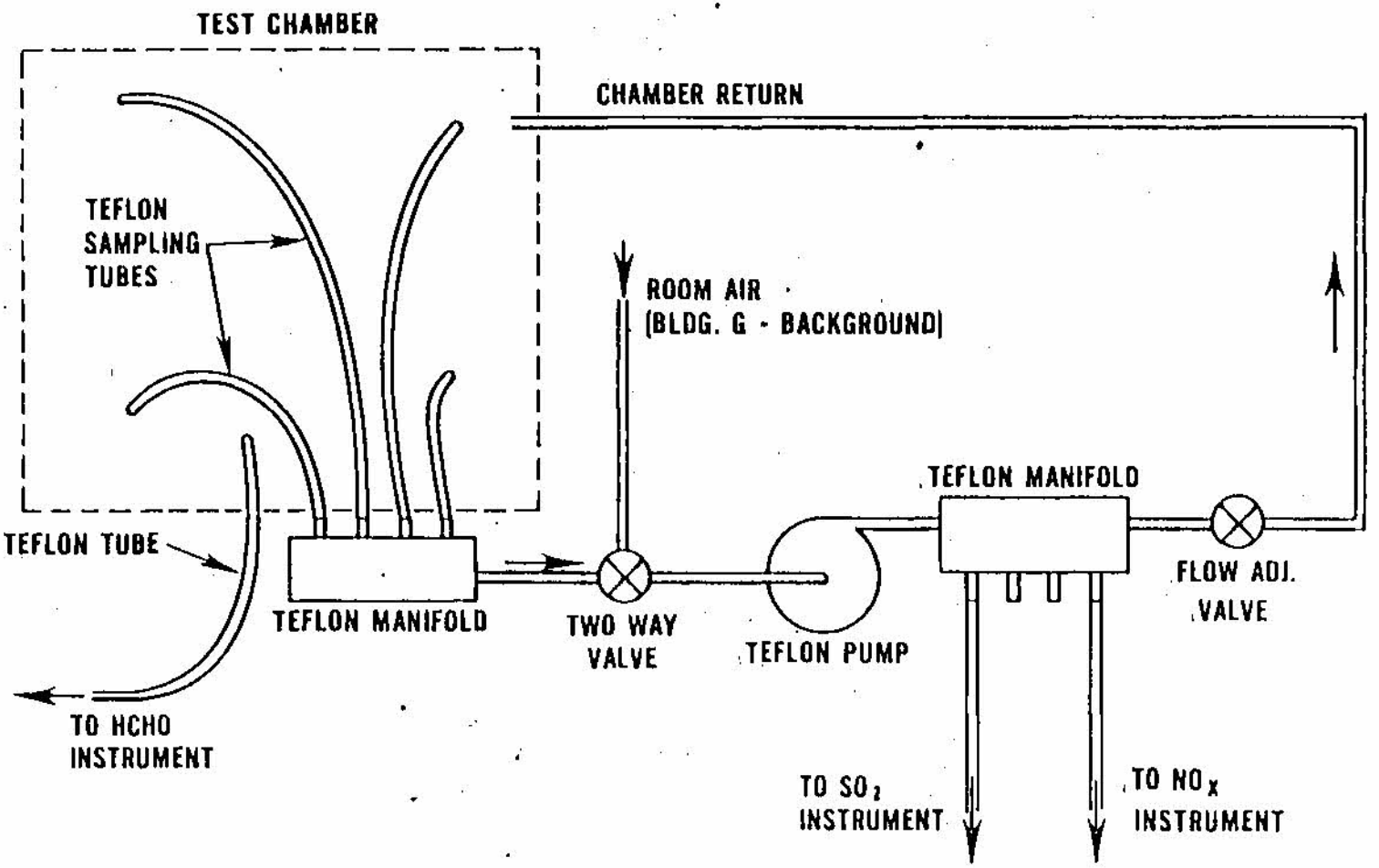


FIGURE 2 - NO_x & SO_2 EMISSION SAMPLING MANIFOLDS

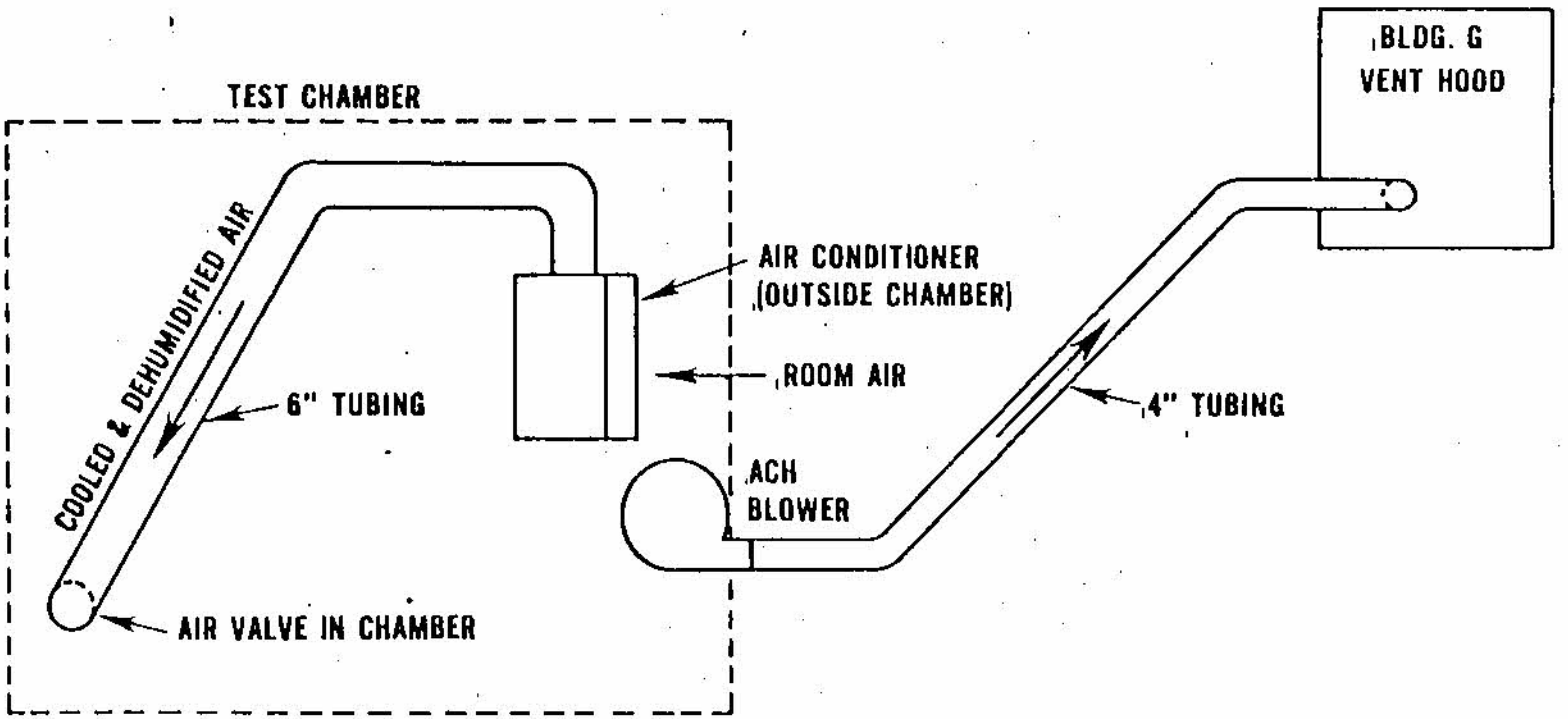


FIGURE 3 - AIR CHANGES PER HOUR (ACH) CONTROL SYSTEM

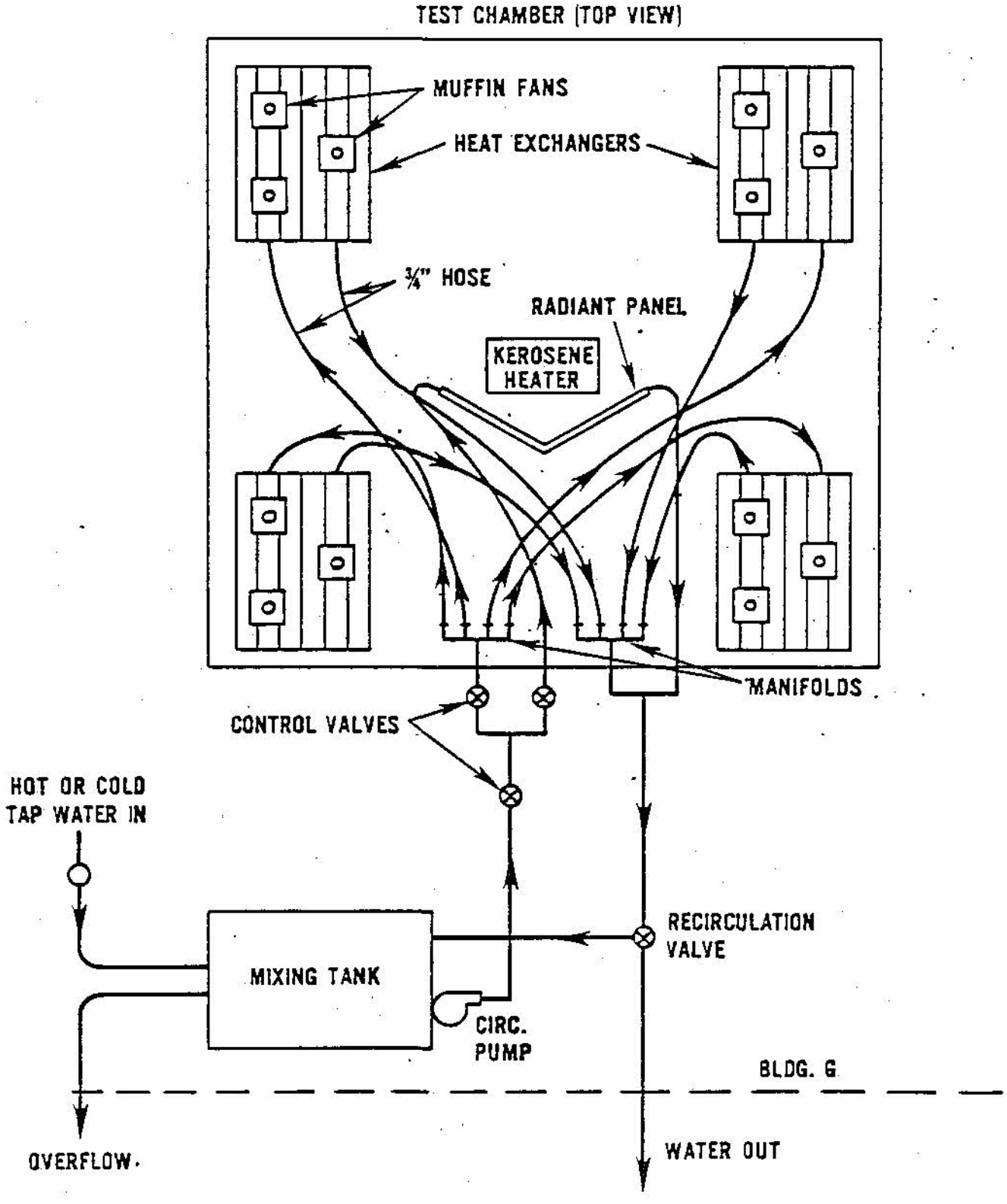


FIGURE 4 - WATER COOLING SYSTEM

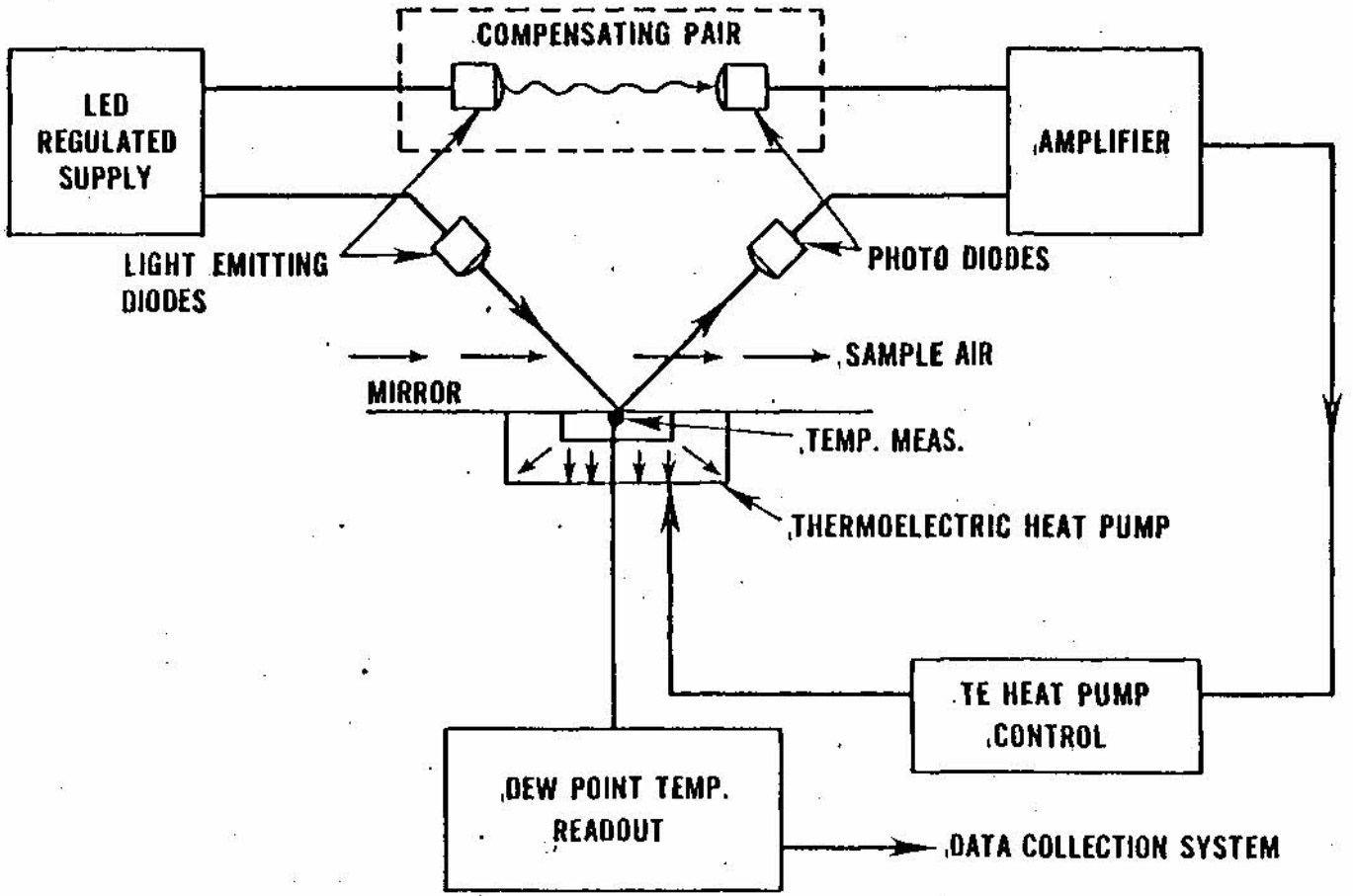


FIGURE 5 - DEW POINT HYGROMETER

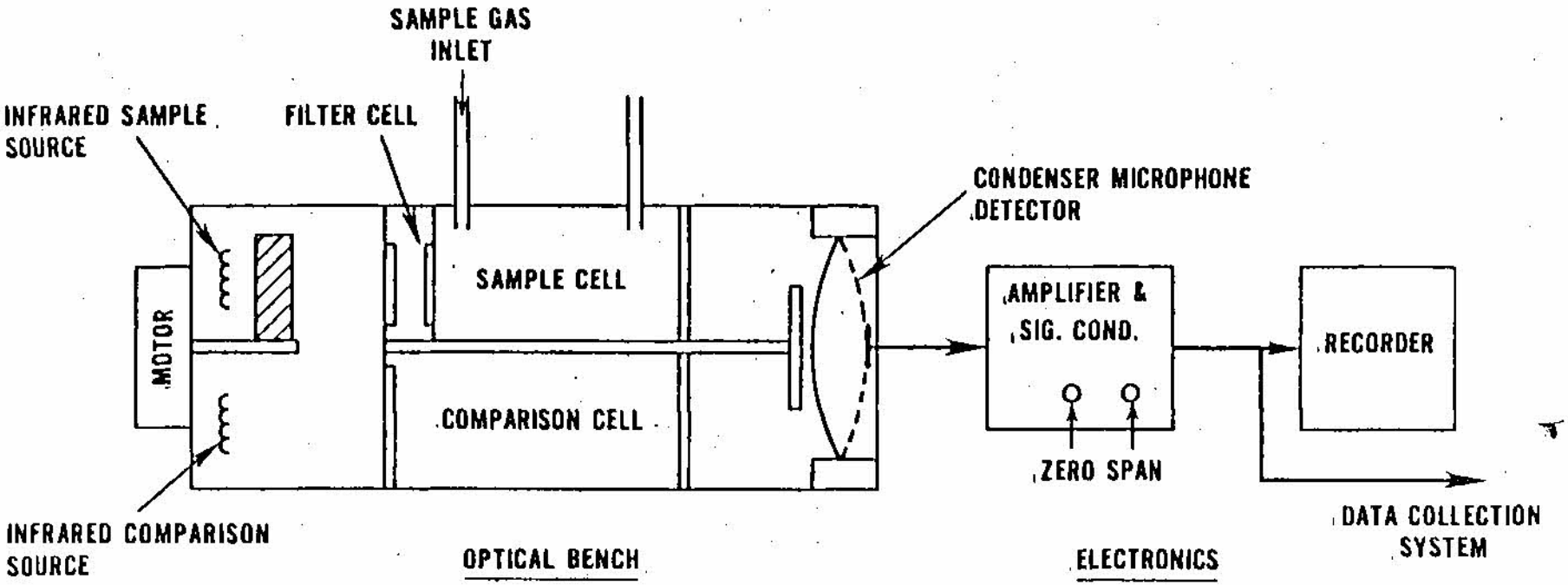


FIGURE 6 - CO & CO_2 INFRARED TYPE (LIRA 303) INSTRUMENT

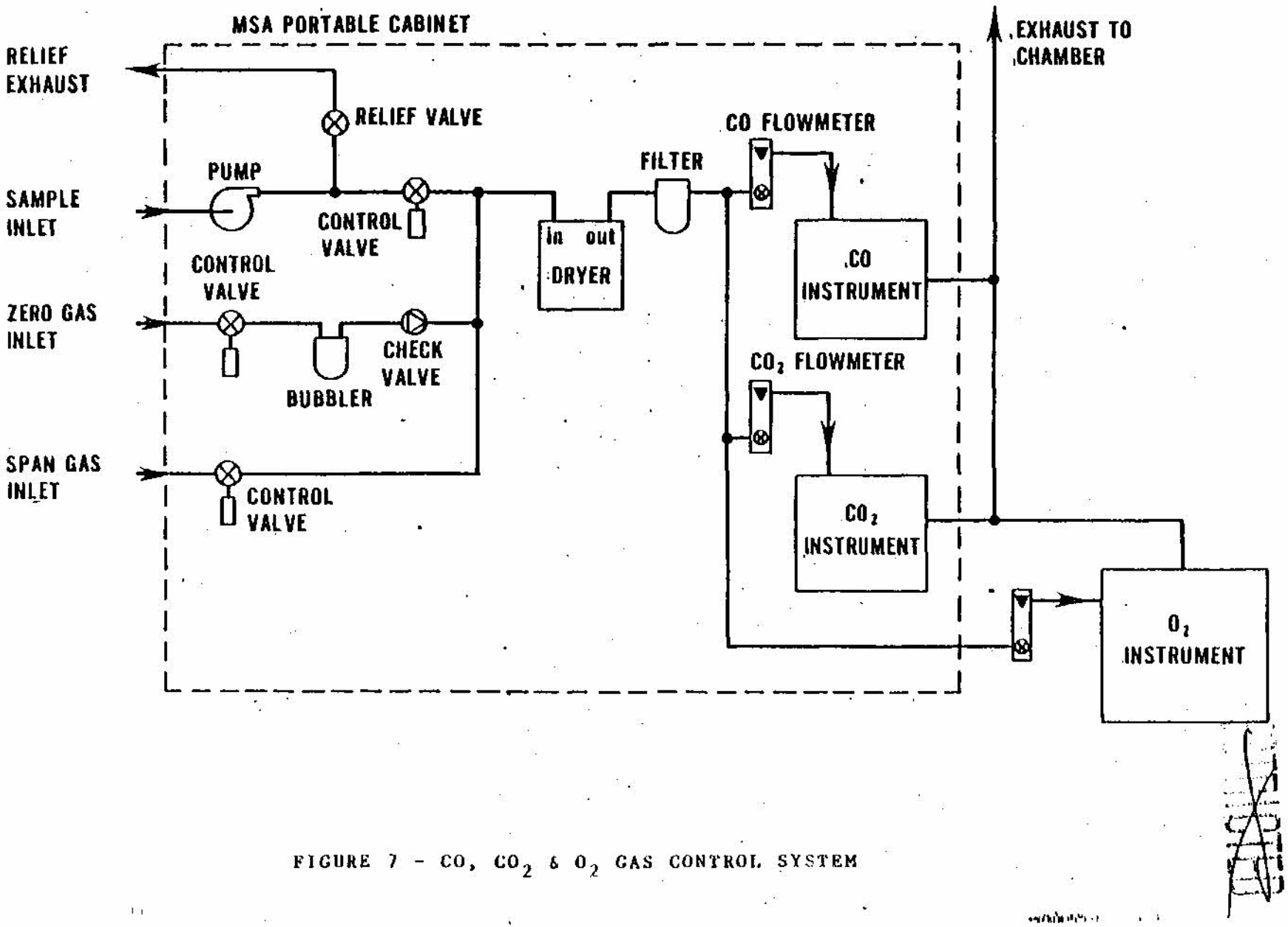


FIGURE 7 - CO, CO_2 & O_2 GAS CONTROL SYSTEM

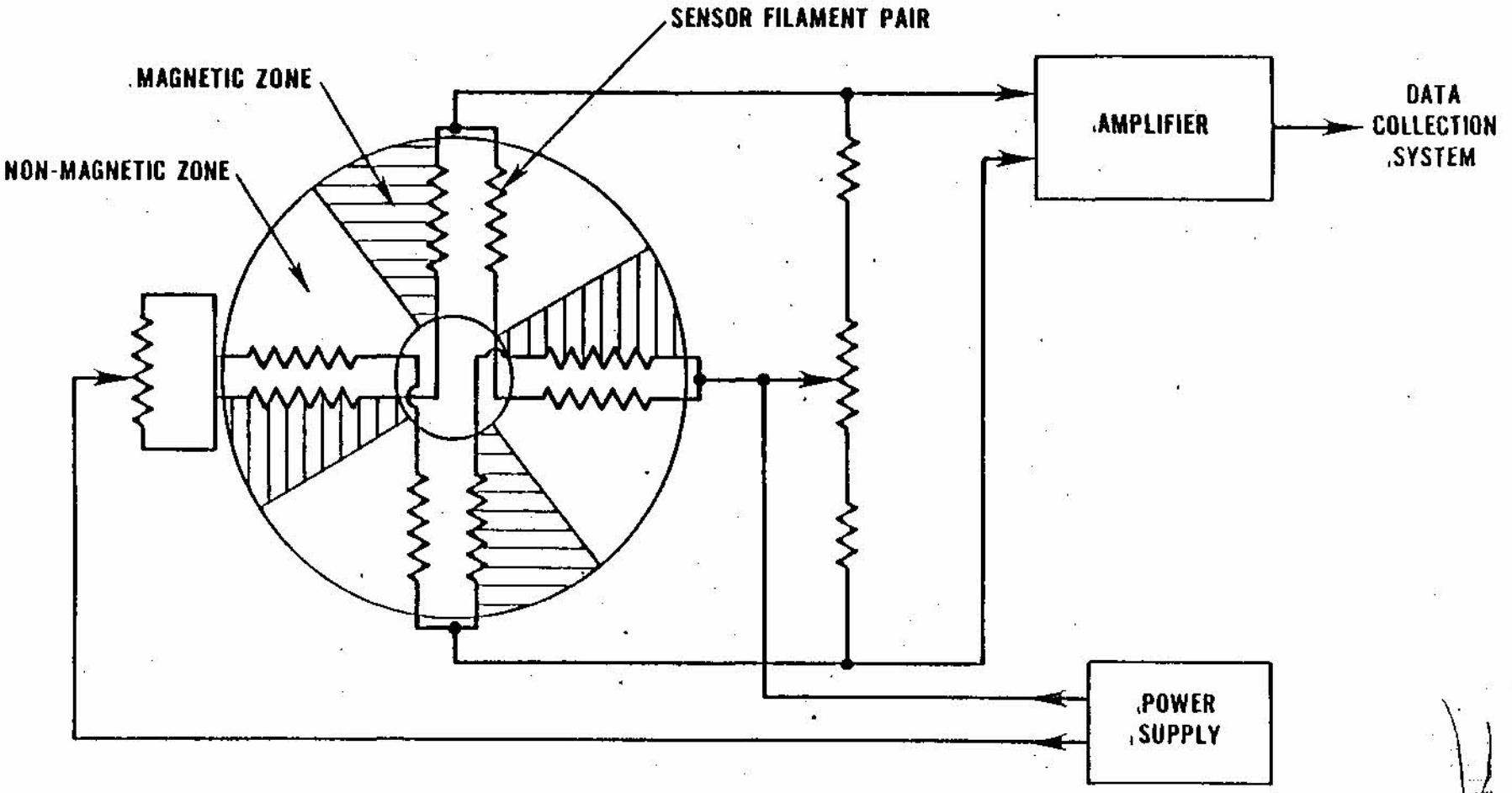


FIGURE 8-O_2 PARAMAGNETIC WIND TYPE INSTRUMENT

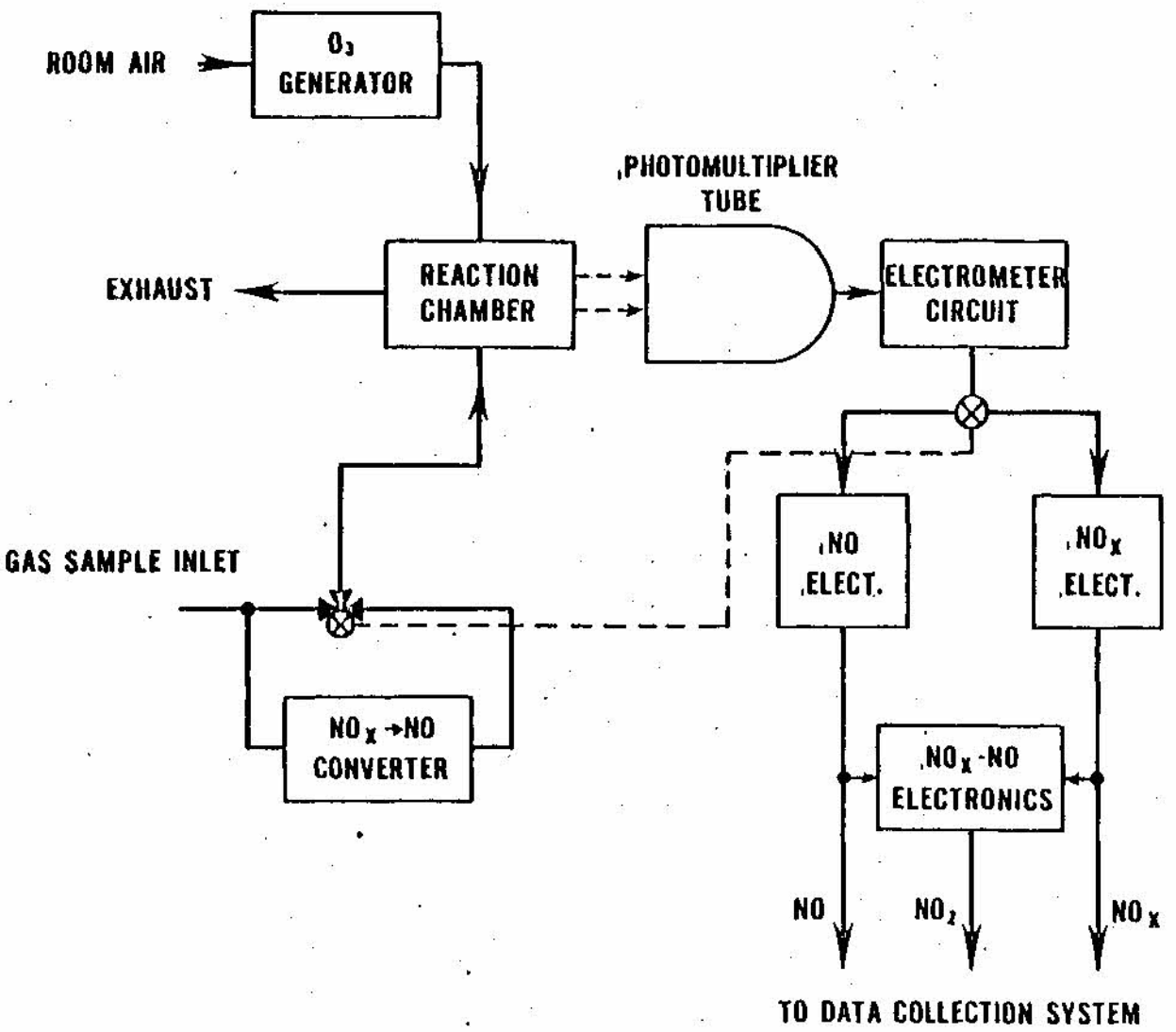


FIGURE 9 - NO_x CHEMILUMINESCENT TYPE INSTRUMENT

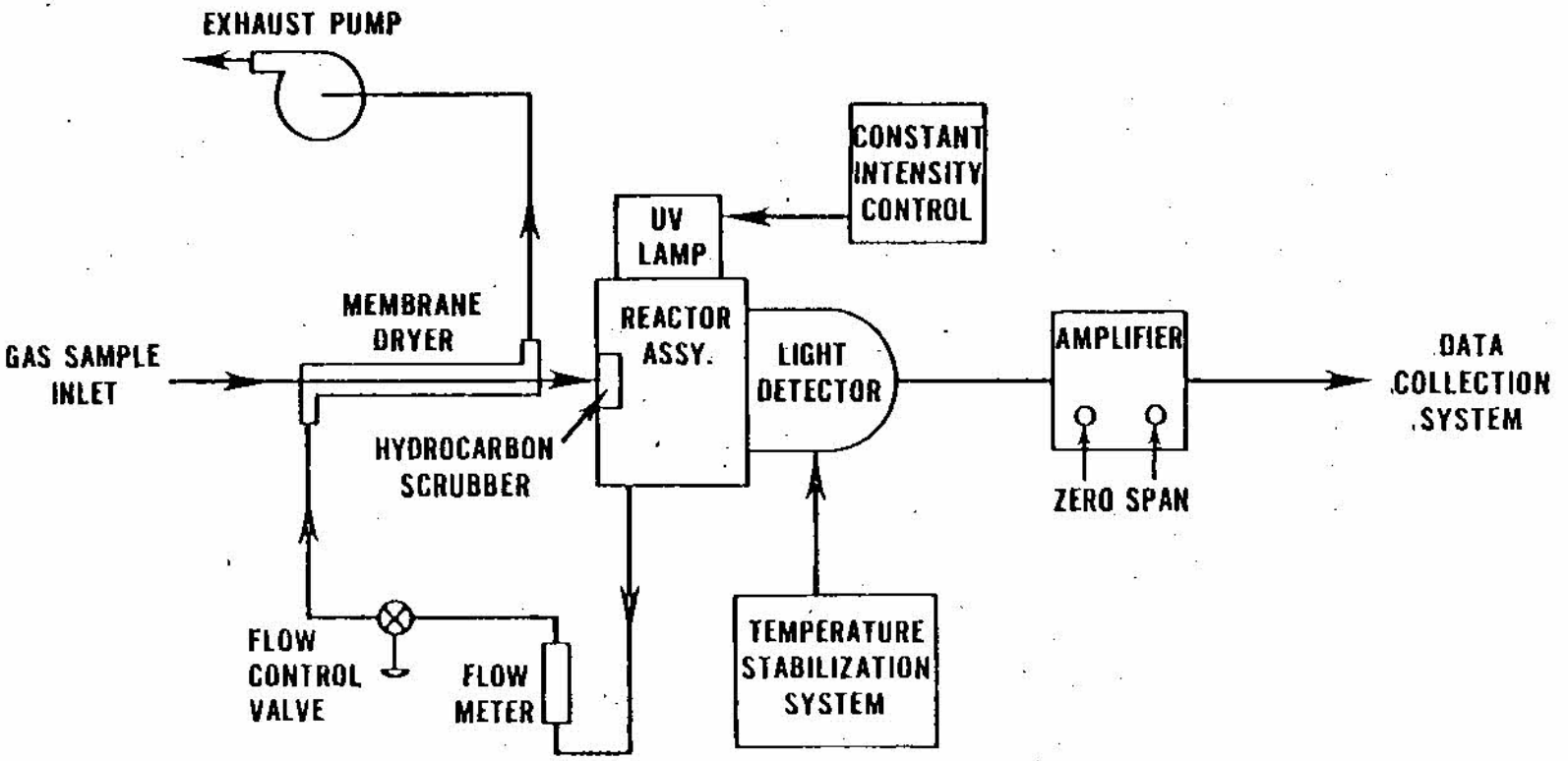
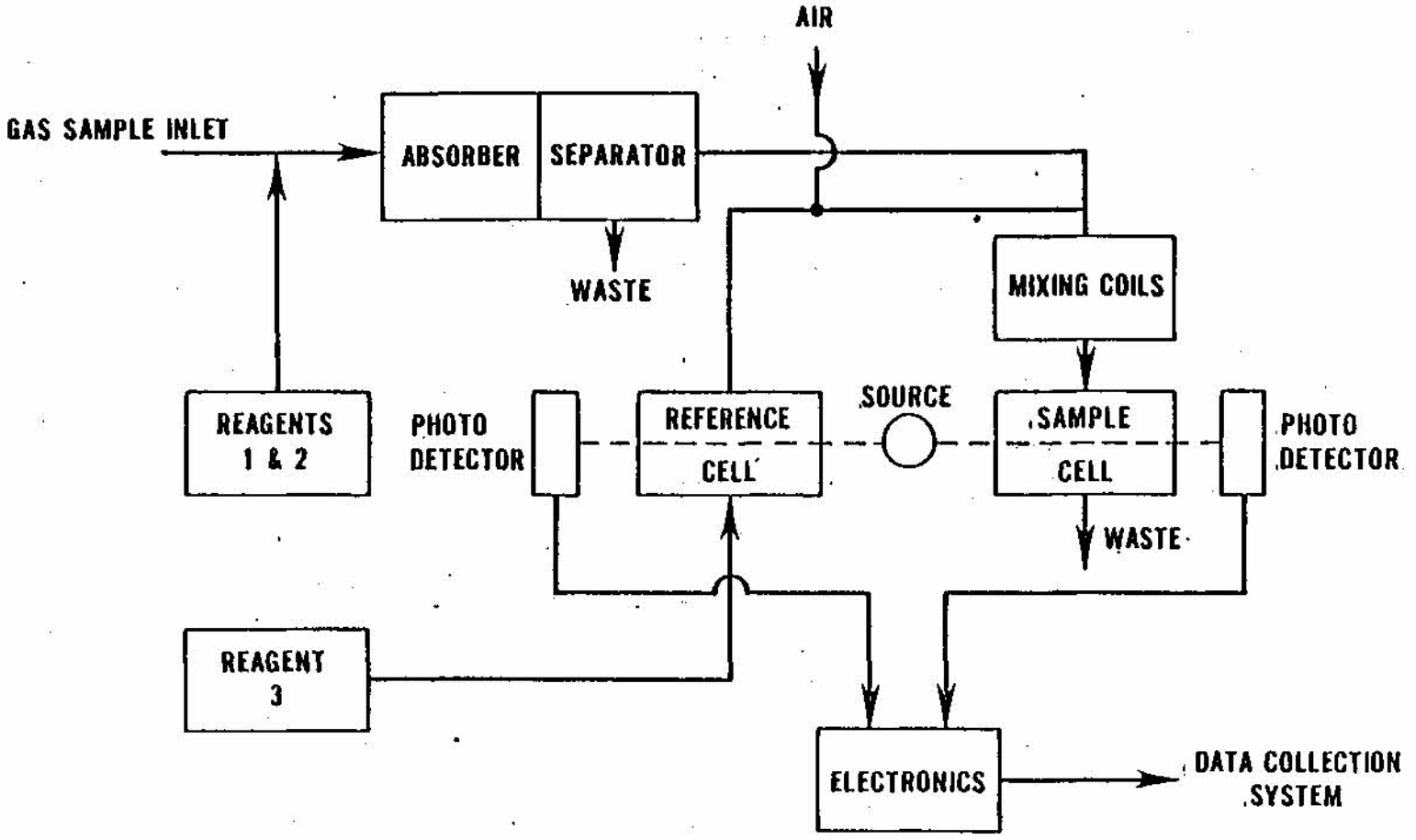


FIGURE 10 - SO_2 FLUORESCENT TYPE INSTRUMENT



REAGENT 1: SODIUM SULFITE IN WATER
REAGENT 2: MERCURIC CHLORIDE + SODIUM CHLORIDE IN WATER
REAGENT 3: PARAROSANILINE CHLORIDE + HYDROCHLORIC ACID IN WATER

FIGURE 11 - HCHO WET-CHEMICAL COLOROMETRIC TYPE INSTRUMENT

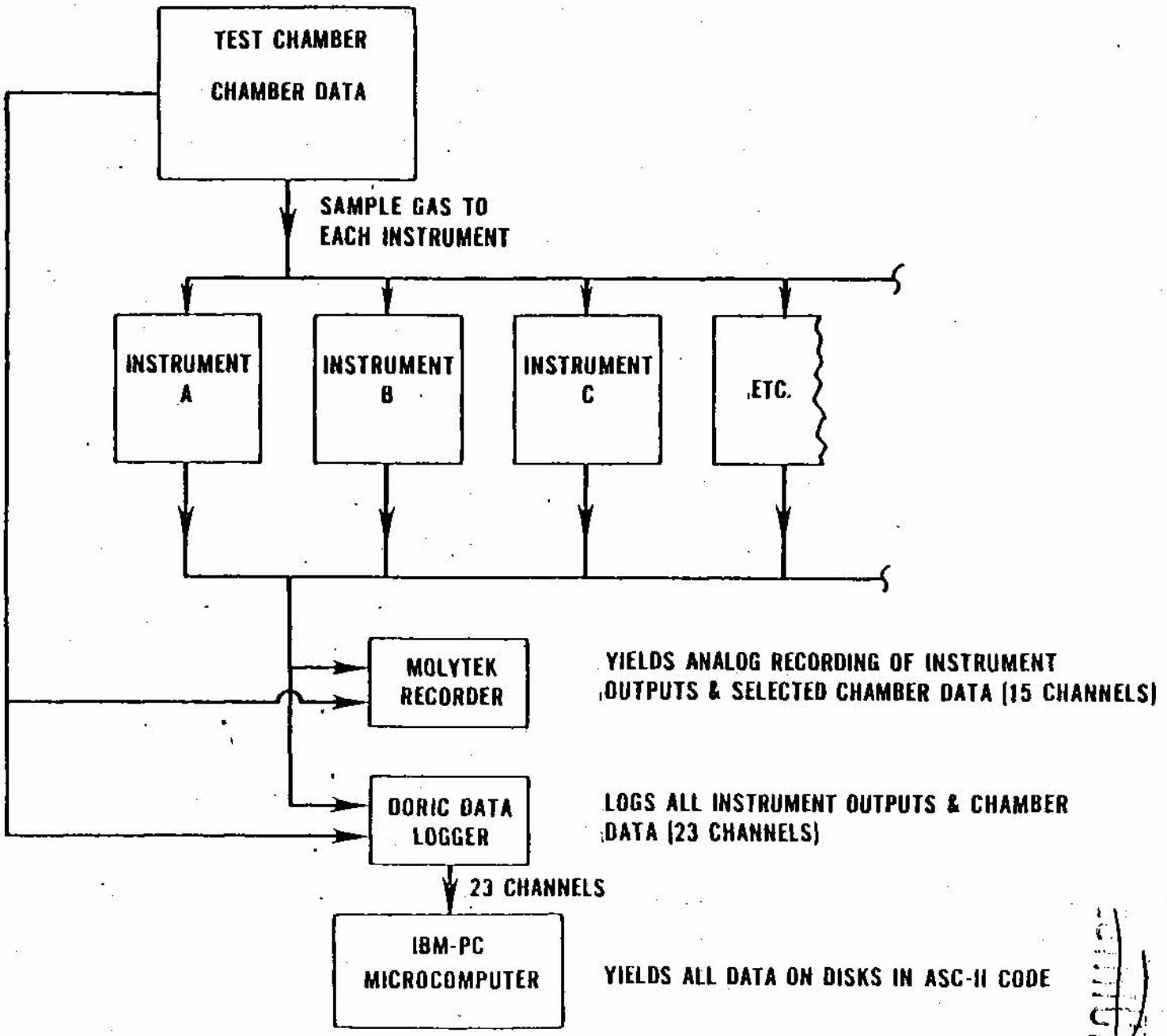


FIGURE 12 - DATA COLLECTION SYSTEM

TABLE I

KEROSENE HEATER EMISSIONS TEST DATA COLLECTION SYSTEM ASSIGNMENT OF CHANNELS FOR MOLYTEK, DORIC AND IBM-PC

Channel No.	Device	Channel No.	Device
1	CO Instrument	17	Calib. Voltage
2	CO_2 Instrument	18	Dew Point
3	O_2 Instrument	19	Spare
4	NO }	20	Spare
5	NOx } Instrument	21	Temp.- Radiant Cooling Panel
6	NO_2 }	22	Temp.- Water and Radiant Cooling Panel
7	Spare	23	Temp.- Chamber Floor - R. Side
8	Spare	24	Temp.- Water to Conv. Cooler
9	Spare	25	Temp. - Top of Heater
10	SO_2	26	Temp. - Instrument Room
11	SO_2 (Inst. Ck. Only)	27	Temp. - Inlet #1 (L. Front)
12	SO_2 (Inst. Ck. Only)	28	Temp. - Inlet #2 (L. Rear)
13	HCHO	29	Temp. - Inlet #3 (R. Rear)
14	Spare	30	Temp. - Inlet #4 (R. Front)
15	Spare	31	Spare
16	Load Cell	32	Spare

KEROSENE HEATER EMISSIONS TEST CHECK LIST

SAMPLE NO. _____ TEST # _____ DATE _____

_____ Water Cooling System On

_____ Air Conditioner On

_____ ACH Fan On

_____ Air Circulation Fans On

_____ CO, CO_2 and O_2 Pump On

_____ Carbon Monoxide Analyzer On and Calibrated

_____ Carbon Dioxide Analyzer On and Calibrated

_____ Carbon Monoxide and Carbon Dioxide Recorder On

_____ Oxygen Analyzer On and Calibrated

_____ Oxides of Nitrogen Pump On

_____ Oxides of Nitrogen Analyzer On and Calibrated

_____ Molytek Recorder Turned On

_____ Molytek Clock On and Synchronized

Channels On _____

_____ Doric Recorder Turned On

_____ Doric Clock Synchronized

Channels On _____

_____ Dew Point Monitor On

_____ Heater Fueled

_____ Heater Preburn Completed

_____ Load Cell Zero

_____ Shut-Off Mechanism Set Up

KEROSENE HEATER EMISSIONS TEST DATA SHEET

SAMPLE NO._____ TEST #_____ DATE__/__/__

Air Changes Per Hour - Fan Setting_____

Air Conditioner Setting_____

Chamber_____ Air Changes Per Hour

Barometer Reading_____

Heater Fuel_____

Heater Setting_____

Heater Operated_____ hours

Preburn Started_____

Heater Weight Before Test_____

Heater Put Into Chamber_____

Heater Weight After Test_____

Heater Shut-Off_____

Building G Room Temperature_____

Load Cell Range_____

KEROSENE HEATER EMISSIONS TEST CO, CO_2, and O_2 TIME SHEET

PRE TEST CALIBRATION	TIME
O_2 - 20.93%	
CO - ZERO CO_2 - ZERO O_2 - ZERO	
CO - 24.32 PPM	
CO - 97.0 PPM	
CO - 243.5 PPM	
CO_2 - 1.4519%	
CO_2 - 2.872%	
CO - 764 PPM CO_2 - 9.02%	
Background	
Test Start	
Heater Shut-Off	
Test Completed	

POST TEST CALIBRATION	
Background	
O_2 - 20.93%	
CO - ZERO CO_2 - ZERO O_2 - ZERO	
CO - 24.32	
CO - 97.0	
CO - 243.5	
CO_2 - 1.4519	
CO_2 - 2.872	
CO - 764 PPM	
CO_2 - 9.02%	

UNITED STATES
CONSUMER PRODUCT SAFETY COMMISSION
WASHINGTON, D.C. 20207

The Chairman

Honorable Lee M. Thomas
Administrator
U.S. Environmental Protection Agency
401 M Street S.W.
Washington, DC 20460

Dear Mr. Thomas:

I am requesting the assistance of the Environmental Protection Agency's Clean Air Science Advisory Committee (CASAC) in our review of the potential health hazards associated with exposure to 0.1 to 1.0 plus ppm nitrogen dioxide generated by the unvented combustion sources used in the home.

The Commission has been concerned about consumer exposure to nitrogen dioxide associated with the use of gas cooking stoves and a variety of combustion home heaters. Various studies, including several conducted for the CPSC, have shown that the levels of nitrogen dioxide exposure associated with the use of these appliances significantly exceed the ambient air standard as well as the short term standard previously recommended by the EPA staff.

Various gas stove and combustion heater industry representatives have indicated a willingness to modify their product in order to reduce consumer exposure to nitrogen dioxide but there remains some disagreement as to what the target level should be. In an effort to expedite this process, I believe that the CASAC, since it has recently reviewed the data on NO_2, could give the Commission guidance on questions such as:

- -the levels of nitrogen dioxide for which there are data indicating adverse health effects;
- -the identity of subsets of the population more sensitive to nitrogen dioxide than others; and
- -whether exposure to nitrogen dioxide leads to irreversible lung damage.

Please advise me as to the feasibility of obtaining such assistance, and, if it is feasible, the process for obtaining CASAC review. Thank you for your assistance in this matter.

Sincerely yours,

Terrence Scanlon

Terrence Scanlon
Chairman

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

MAY 1 1985

THE ADMINISTRATOR

Honorable Terrence Scanlon
Chairman
U. S. Consumer Product Safety
Commission
Washington, D. C. 20207

Dear Mr. Scanlon:

Thank you for your March 29 letter in which you request the assistance of EPA's Clean Air Scientific Advisory Committee (CASAC) in evaluating consumer exposures to nitrogen dioxide associated with the use of gas cooking stoves and a variety of combustion home heaters. The Committee, which has reviewed the scientific basis of EPA's National Ambient Air Quality Standard for Nitrogen Dioxide, is well qualified to address the issues identified in your letter, and I support your request to solicit its scientific advice. I know that the CASAC Chairman, Dr. Morton Lippmann of New York University, would also be willing to assemble his panel to undertake this review.

I suggest that your staff coordinate the preparation for the CASAC meeting with Dr. Terry F. Yosie, Director of EPA's Science Advisory Board, (382-4126) and Mr. Bruce Jordan of EPA's Office of Air Quality Planning and Standards (919) 541-5655. Two specific requests that I have of the Commission staff is to work closely with Dr. Yosie and Mr. Jordan in preparing the scientific materials to submit to CASAC, and to provide budgetary support to defray the cost of the meeting. I estimate that the costs of the review will approximate $15,000 - $20,000.

Thank you for your interest in working with the Agency on this important public health issue.

Sincerely,

Lee M. Thomas
Administrator

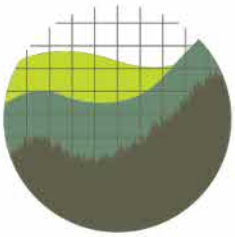
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CONSUMER PRODUCT
SAFETY COMMISSION

ADM-CPSC-GEN.-COMMITTEE ON INDOOR
AIR QUALITY

Memorandum from HS to the Commission re: Committee on Indoor Air Quality -
6/28/84

RESTRICTED

U.S. CONSUMER PRODUCT SAFETY COMMISSION



Institute *for*
Policy Integrity
NEW YORK UNIVERSITY SCHOOL OF LAW



Abioye Mosheim
Chief FOIA Officer, Assistant General Counsel
U.S. Consumer Product Safety Commission
4330 East West Highway
Bethesda, MD 20814
Via foia.gov

July 6, 2020

Re: Freedom of Information Act Request

This is a request under the Freedom of Information Act. We request copies of the following records:

- All research, data, and correspondence between the Consumer Product Safety Commission (CPSC) and the Environmental Protection Agency (EPA) that informed the May 1986 publication entitled *Review Of The Consumer Product Safety Commission's Health Effects And Exposure Assessment Documents On Nitrogen Dioxide*, available on https://nepis.epa.gov/
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- Any record of communication between CPSC and gas stove manufacturers, gas companies, or related companies or trade associations regarding the health effects of indoor air pollution from gas stoves/ranges/ovens
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- Laboratory or field test reports of gas stove/range/oven emissions, including source-strength emissions

If access to any of the above information is delayed due to the impacts of COVID-19, please prioritize the documents that are readily accessible and send archived records as they become available.

In order to help to determine our status to assess fees, we are affiliated with the Institute for Policy Integrity at NYU School of Law, an educational institution, and Rocky Mountain Institute, a non-partisan, non-profit organization that provides information that is in the public interest. This request is made for a scholarly purpose and not for a commercial use.

Accordingly, we request a waiver of all fees for this request. Disclosure of the requested information is in the public interest as it is likely to contribute to public understanding of the health and environmental impacts of gas cooking stoves.

If you have any questions regarding this request, please contact us at jack.lienke@nyu.edu or dgrab@rmi.org.

Thank you for your consideration of this request.

Sincerely,

Jack Lienke
Regulatory Policy Director
Institute for Policy Integrity at NYU School of Law
139 MacDougal Street, Third Floor
New York, NY 10012
(212) 998-6222

Denise Grab
Building Electrification Program Manager
Rocky Mountain Institute
1111 Broadway
Oakland, CA 94607
(510) 501-6380

FOIA Request 142481

The following list contains the entire submission submitted July 08, 2020 01:10:03pm ET, and is formatted for ease of viewing and printing.

Contact information

First name	Jack
Last name	Lienke
Mailing Address	139 MacDougal Street, Third Floor
City	New York
State/Province	NY
Postal Code	10012
Country	United States
Phone	2129986222
Company/Organization	Institute for Policy Integrity at NYU School of Law
Email	jack.lienke@nyu.edu

Request

Request ID	142481
Confirmation ID	141956
Request description	We request copies of the following records: • All research, data, and correspondence between the Consumer Product Safety Commission (CPSC) and the Environmental Protection Agency (EPA) that informed the May 1986 publication entitled Review Of The Consumer Product Safety Commission's Health Effects And Exposure Assessment Documents On Nitrogen Dioxide, available on https://nepis.epa.gov/ • All research, data, and correspondence between CPSC and EPA that informed the April 1995 publication entitled The Inside Story: A Guide to Indoor Air Quality, located at https://www.cpsc.gov/Safety-Education/Safety-Guides/Home/The-Inside-Story-A-Guide-to-Indoor-Air-Quality • Any record of communication between CPSC and gas stove manufacturers, gas companies, or related companies or trade associations regarding the health effects of indoor air pollution from gas stoves/ranges/ovens • Any record of communication between CPSC and gas stove manufacturers, gas companies, or related companies or trade associations regarding potential regulatory or research initiatives related to gas stoves/ranges/ovens • Any record of communication between CPSC and health and medical researchers, practitioners, doctors, medical associations, or related groups regarding health risks related to gas stoves/ranges/ovens • Any documents containing information on the health effects of indoor air pollution from gas stoves/ranges/ovens • Any documents containing information on potential regulatory or research initiatives related to the health effects of indoor air pollution from gas stoves/ranges/ovens • Laboratory or field test reports of gas stove/range/oven emissions, including source-strength emissions

Supporting documentation

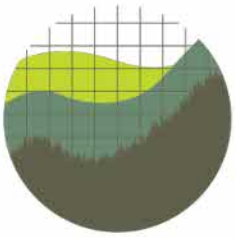
Additional Information	CPSC FOIA Request_2020.07.06.pdf

Fees

Request category ID	educational
Fee waiver	yes
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Willing to pay	$25.00

Expedited processing

Expedited Processing	no



Institute *for*
Policy Integrity
NEW YORK UNIVERSITY SCHOOL OF LAW



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