HP 99-1

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CPSC/OFFICE OF
THE SECRETARY

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CPSA 6 (b)(1) Cleared

___ No Mfrs/PrvtLblrs or
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X Excepted by Pet Predz
___ Firms Notified,
Comments Processed.

November 19, 1998

Office of the Secretary
Consumer Product Safety Commission
4330 East West Highway
Bethesda, MD 20899

Please accept the attached petition to initiate a Commission rulemaking to ban polyvinyl chloride (PVC) from all toys and products intended for children five years of age and under and to issue a national advisory on the health risks that have been associated with PVC toys and products.

Attachments to this request include:

"A Select Annotated Bibliography on the Toxicity of Diisononyl Phthalate (DINP) and its Migration from Children's Products," Barbara Bass, National Environmental Trust, October 1998.

"Determination of the Composition and Quantity of Phthalate Ester Additives in PVC Children's Toys," Stringer et al., Greenpeace Research Laboratories, September 1997.

"Lead and Cadmium in Vinyl Children's Products, " Joseph Di Gangi, Greenpeace.

"Report on DINP Phthalate: Summary of the Published Literature and Quantities Found in Common Toys," Thomas Natan, National Environmental Trust, November 1998.

"Toxic Chemicals in Vinyl Children's Toys," Greenpeace, November 1998.

Petition

To the Consumer Product Safety Commission Concerning PVC in Children's Toys

Whereas soft plastic vinyl—polyvinyl chloride (PVC)—requires the addition of chemical softeners and hard metal stabilizers that have been linked to potentially serious health effects;

Whereas independent health studies have consistently found associations between DINP phthalate softeners in soft plastic vinyl (PVC) and liver and kidney damage;

Whereas a preliminary Consumer Product Safety Commission (CPSC) hazard assessment stated that DINP phthalate exposure was associated with "toxic effects in the liver, kidney, and other organs of mice and rats;"

Whereas CPSC's hazard assessment of DINP phthalate stated, "It is conceivable that one or more existing types of DINP for which data are unavailable could also be more toxic and/or carcinogenic;"

Whereas DINP phthalate is found in virtually every soft plastic vinyl (PVC) toy often at levels of 30 percent or more by weight;

Whereas four out of six studies reviewed by the European Union found levels of phthalate leaching that translated to daily exposure levels higher than the CPSC's "acceptable daily intake level;"

Whereas existing exposure studies likely understate the extent of exposure given that children mouth, bite, and swallow plastic much more aggressively than study simulations;

Whereas eight foreign countries have taken official action on phthalates and PVC including two bans (Austria, Denmark), one pending ban (Sweden), four requests for voluntary action (Belgium, Germany, Italy, Netherlands), and one national health advisory (Canada);

Whereas the attorneys general of 11 states are conducting a joint investigation of lead and cadmium levels in soft plastic vinyl (PVC) toys;

Whereas lead may still be found in soft vinyl (PVC) toys after the CPSC urged its removal;

Whereas some U.S. toy manufacturers and retailers have not adequately addressed the problem by acting only on toys intended for the mouth;

Whereas infants and toddlers put all toys in their mouths;

Whereas alternatives to soft plastic vinyl (PVC) are commercially available and affordable;

Whereas an April 21, 1997, Presidential Executive Order states: "Each Federal agency shall ensure that its policies, programs, activities, and standards address disproportionate risks to children that result from environmental health risks or safety risks;"

The petitioners listed below request information about the progress and disposition of this petition at the Commission's earliest opportunity. The Commission may notify petitioners through the following contact person: Jeffrey Becker Wise, Policy Director, National Environmental Trust, 1200 18th Street, N.W., Suite 500, Washington, DC, 20036 (202-887-8800).

Sincerely,

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The undersigned call on the Consumer Product Safety Commission to:

I. Institute an immediate ban on polyvinyl chloride (PVC) in all toys and other products intended for children five years of age and under;

II. Issue a national advisory on the health risks that have been associated with soft plastic vinyl (PVC) toys to inform parents and consumers about the risks associated with PVC toys currently in stores and homes.

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A SELECT ANNOTATED BIBLIOGRAPHY ON THE TOXICITY OF DIISONONYL PHTHALATE (DINP) AND ITS MIGRATION FROM CHILDREN'S PRODUCTS

October 1998

Prepared for
The National Environmental Trust
Washington, DC
by
Barbara F. Bass, Ph.D.

The scope of this bibliography was limited due to the degree of availability of various reports, articles, and studies, as well as the time constraints of the project. There are a number of studies on the toxicity of diisononyl phthalate as well as its migration from children's products that are not in the peer reviewed literature. Some of these have been submitted to the U.S. Environmental Protection Agency (EPA) under the Toxic Substances Control Act, others are studies that have been performed by government agencies in other countries. Access to some of these reports has, therefore, been limited (e.g., most of the studies submitted to EPA are available from the Agency, but only on microfiche.)

The summaries of the articles and reports reviewed for this bibliography are based directly on the authors' statements and data presented in the reports. They do not reflect the opinions of the preparer of this bibliography.

I. Articles and Reports on the Toxicity of Diisononyl Phthalate (DINP)

A. Articles and Reports Reviewed in the Annotated Bibliography

1. Barber, E.D., Astill, B.D., Moran, E.J., Schneider, B.F., Gray, T.J.B., Lake, B.G., and Evans, J.G. (1987). Peroxisome Induction Studies on Seven Phthalate Esters. *Toxicology and Industrial Health*, 3:7-24.

Seven phthalate esters, including DINP, were fed to groups of Fischer 344 rats for 21 days at levels of either 0.3% or 0.6%, and 1.2% and 2.5%. The following observations were made regarding DINP:

- dose-dependent body weight reduction in males and females;
- similar level of peroxisome proliferation in DINP rats fed 2.5% DINP in their diet as seen in rats fed 2.5% di(2-ethylhexyl)phthalate (DEHP) in their diets;
- dose-related increase in relative liver weights and palmitoyl-CoA oxidation;
- slight decrease in serum triglyceride levels in males and a slight increase in females;
- serum cholesterol levels similar in their resistance to change as the triglyceride levels; and
- dose-related increase in lauric acid 12-hydroxylase levels.

The authors concluded that DINP showed similar activity for many of the parameters as DEHP and di(isodecyl)phthalate (DIDP). The authors state that relative liver weight and cyanide-insensitive palmitoyl-CoA oxidation were the most sensitive and responsive parameters in their study and that the results in the study are in keeping with the phthalate esters as a class being weak peroxisome proliferators.

2. Bird M.G., Kapp, R.W., Keller, C.A., Lington, A.W. (1986). A Thirteen Week Feeding Study on Diisononyl Phthalate (DINP) in Rats. *The Toxicologist*, 6:302. (Note: This is an abstract.)

Groups of both male and female F-344 rats were fed DINP in their diets at levels of 0, 1,000, 3,000, 6,000, 10,000, and 20,000 ppm for 13 weeks. The authors observed the following:

- no deaths before the end of the study;
- in the high dose group a significant depression in food intake and body weight gain;
- dose-related decrease in clinical chemistry values for triglycerides;
- a dose-related elevation in albumin globulin ratios;
- no significant hematologic changes;
- increases in absolute and relative liver and kidney weights of various treated groups (≥ 3,000 ppm) compared to controls;
- kidney lesions (nephrotic and regenerative changes) in various males groups (≥ 3,000 ppm);

- dose-related diffuse cytoplasmic eosinophilic hypertrophy of liver hepatocytes in all treatment groups; and
- dose-related proliferation of peroxisomes and smooth endoplasmic reticulum in the liver as evidenced by electron microscopy in some groups (≥ 6,000 ppm).

The authors state in summary that high doses of DINP produced liver and kidney effects in rats with a no observed effect level at 1,000 ppm.

3. Brodell, R.T., and Torrence, B.P. (1992). Sqwish Ball Dermatitis. *Journal of the American Academy of Dermatology*, 26:641-642.

This is a case report in which it was reported that a 10 year old girl had itching and burning hands for 12 hours after she had cut open a Sqwish Ball the day before and her hands had come in contact with the material inside. The material was very sticky and efforts to remove it with soap and water caused spreading of it on her hands. Soft Scrub was then used to help remove the material. The next day tiny erythematous papules and papulovesicles were observed on the back of her hands and her palms, and slight scaling was also noted.

The manufacturer stated that there were 20 reports of instances in which consumers had opened the toys with subsequent problems being reported. Five of these involved reports of dermatitis. DINP is one of the ingredients of the material inside Sqwish Balls. The toy manufacturer believed this to be the cause of the dermatitis resulting from contact with the toy.

The other possible irritant to which the patient was exposed was Soft Scrub cleanser, whose main ingredient, according to the manufacturer, is calcium carbonate. The manufacturer of Soft Scrub stated that there had been few reports of dermatitis resulting from the use of the cleanser.

The authors state that the eruption was that of an irritant as opposed to an allergic contact dermatitis. The irritation may have been caused by the material inside the Sqwish Ball, which contains DINP, or from attempts to remove the material, or both.

4. Butala J.H., Moore, M.R., Cifone, M.A., Bankston, J.R., and Astill, B. (1997). Oncogenicity Study of Di(isononyl)phthalate in Mice. *The Toxicologist*, 36:173. (Note: This is an abstract.)

Male and female B6C3F1 mice were fed DINP (CAS # 68515-48-0) in their diets at levels of 0, 500, 1,500, 4,000, and 8,000 ppm for up to 104 weeks. Additional mice received 8,000 ppm for 78 weeks followed by 26 weeks of control diet (recovery). Cell proliferation and peroxisome proliferation were determined after weeks 78 and 104. The authors made the following observations:

- reduced survival in both sexes in the 4,000 and 8,000 ppm groups in a dose-related way which reversed, but did not reach control levels, in the recovery groups;
- at week 105, male body weights reduced by 11.1% and 18.9% in the two highest dose groups and 5.7% in recovery group as compared to controls;
- at week 105, female body weights reduced by not more than 9.8% in any dose group

and a 5.7% reduction in the recovery group as compared to controls;

- in male mice, increased hepatocellular carcinoma in treated vs control animals:

  controls - 23%
  1,500 ppm group - 30%
  4,000 ppm group - 47%
  8,000 ppm group - 44%
  recovery group - 38%;

- in female mice, increased hepatocellular carcinoma in treated vs control animals:

  controls - 4%
  1,500 ppm group - 17%
  4,000 ppm group - 18%
  8,000 ppm group - 46%
  recovery group - 36%;

- no test related tumors of any other type;
- non-neoplastic liver changes included: increased pigment, cytoplasmic eosinophilia and hepatocellular enlargement in both sexes in the high dose groups, but not recovery groups;
- in high dose female mice, treatment related chronic progressive nephropathy;
- no evidence of effects on spermatogenic activity as evidenced in sections of testes with epididymides; and
- at weeks 78 and 104 a statistically significant increase in liver weight, hepatocellular protein content, and palmitoyl CoA oxidase activity, but not cellular labeling or DNA content.

5. Butala J.H., Moore, M.R., Cifone, M.A., Bankston, J.R., and Astill, B. (1996). Oncogenicity Study of Di(isononyl) Phthalate in Rats. *The Toxicologist*, 30:202. (Note: This is an abstract.)

Male and female F344 rats received DINP in their diet for up to 104 weeks at levels of 0, 0.05, 0.15, 0.6, and 1.2%. Additional animals received 1.2% for 78 weeks followed by 26 weeks of the control diet (recovery group). After weeks 1, 2, 13, 79, and 104, cell proliferation was determined. The authors observed the following:

- NOEL in both sexes for systemic and carcinogenic effects was 0.15%;
- reduced survival in the high dose males;
- reduced body weight in high dose group of both sexes;
- absolute and relative increases in liver weights in both sexes of the 0.6 and 1.2% groups, but no pathology in the livers of the 0.6% group;
- hepatocellular carcinoma in the 1.2% groups after week 79;
- livers in the recovery rats were comparable to controls at termination;
- absolute and relative increases in kidney weight in females of the 0.6 and 1.2% group;
- tubule cell carcinoma in high dose male group;
- reversible kidney enlargement in the recovery group, but irreversible kidney carcinoma; and
- mononuclear cell leukemia in both sexes in the 0.6, 1.2%, and recovery groups.

6. Harris, C.A., Henttu, P., Parker, M.G., Sumpter, J.P. (1997). The Estrogenic Activity of Phthalate Esters *In Vitro*. *Environmental Health Perspectives*, 105:802-811.

Thirty-five phthalate esters were tested for estrogenic activity in a recombinant yeast assay. A human estrogen receptor has been inserted into the yeast genome. The authors state that this assay is highly specific for estrogens. Those phthalates showing activity in the yeast assay were then tested on estrogen-responsive breast cells. The following observations regarding DINP were made by the authors:

- DINP exhibited slightly unreproducible behavior in the yeast screen: in three separate assays it caused estrogenic activity, but in three others it appeared completely inactive;
- no differences were observed in the yeast assay when a commercial preparation of DINP obtained from EXXON Chemical Ltd., Fareham, United Kingdom, was tested as compared to the analytical standard;
- DINP produced cell proliferation of ZR-75 human breast cancer cells at a level significantly greater than the control; and
- DINP showed relatively little activity when tested using MCF-7 human breast cancer cells, consistent with the results of the yeast assay.

The reason for differences seen in the two human breast cancer cell lines is unclear, but the ZR-75 line is considered to be more estrogen specific. Based on these studies the authors concluded that DINP was extremely weakly estrogenic *in vitro*.

7. Hellwig, J., Freudenberger, H., and Jackh, R. (1997). Differential Prenatal Toxicity of Branched Phthalate Esters in Rats. *Food and Chemical Toxicology*, 35:501-512.

Several different phthalate esters were tested for prenatal toxicity in Wistar rats of outbred strain Chbb/THOM. The phthalates esters were administered by gavage at 0, 40, 200, and 1,000 mg/kg/day from gestation day 6 to 15. Of the compounds tested, three were types of DINP: DINP 1 (CAS No. 68515-48-0), DINP 2 (CAS No. 28553-12-0), and DINP 3 (CAS No. 28553-12-0, but resulting from a different production line). DINP 2 and DINP 3 were produced at BASF Aktiengelsellschaft. At least 95% of the main alcohol moieties in DINP 2, which was derived from n-butene, were alkyl-substituted octanol or heptanol. In DINP 3 the main alcohol moieties were synthesized from codimerbutene (n-; iso-butene) yielding at least 60% alkyl-substituted hexanols. DINP 1 was of commercial origin and consisted of isomers whose alcohol moieties were mainly approximately equivalent amounts of 3,4-, 4,6-, 3,6-, 3,5-, 4,5-, and 5,6-dimethylheptanol-1. The authors observed the following in rats treated with DINP 1, DINP 2, and DINP 3.

- DINP 1 - these effects were seen only at the highest dose
  - less food consumption in dams;
  - one animal with vaginal hemorrhage and urine-smeared fur;
  - statistically significant increase in relative kidney weights;
  - slight increase in relative liver weights; and

- increased incidence of fetal skeletal variations consisting mainly of rudimentary cervical and/or accessory 14th rib(s).

The authors state that no treatment related effects were seen at levels of 40 and 200 mg/kg/day.

- DINP 2 -
  - one rat in highest dose group with vaginal hemorrhage during treatment;
  - statistically significant increase in relative liver weights in the 40 mg/kg/day group only;
  - statistically significant decrease in the number of implantation sites per dam in the 40 mg/kg/day group only; and
  - statistically significant decrease in the number or live fetuses per dam in the 40 mg/kg/day group only; and
  - increased incidence of skeletal variation (accessory 14th rib) in highest dose group.

The authors state that the multiple malformations seen in one fetus of the highest dose group were considered to be spontaneous in nature. The malformations included globular-shaped heart, unilobar lung, hydrocephaly, dilatation of the aortic arch, and anasarca. No other developmental toxicity was exhibited in the highest dose or other groups.

- DINP 3 - these effects were seen only at the highest dose
  - significantly reduced food consumption during several days of treatment in the dams at the highest dose;
  - reduction in mean body weight gain (30.8 mg/kg/day *vs.* 49.6 mg/kg/day) from gestation days 6-15 at the highest dose ;
  - significantly lower mean body weights at days 13, 15, and 17 post-coitum;
  - significantly increased relative liver weights;
  - significant increase in number of affected fetuses per litter;
  - slightly increased rates of certain fetal skeletal retardations (unossified or incompletely ossified sternebrae) and soft tissue (hydroureter) and skeletal (rudimetary cervical and/or accessory 14th rib(s)) variations.

- The authors state that the occurrence of some soft tissue and skeletal malformations were assumed to be substance induced at the 1000 mg/kg/day dose, a dose at which overt maternal toxicity also occurred. No treatment-related effects were seen in the dams or fetuses of the 40 or 200 mg/kg/day groups.

8. Lin, L.I-K., The Use of Multivariate Analysis to Compare Peroxisome Induction Data on Phthalate Esters in Rats. (1987). *Toxicology and Industrial Health*, 3:25-48.

The author statistically analyzed data from the study described by Barber et al. (1987). (See above, I.A.1.) DINP ranked third in potency for inducing peroxisome proliferation with DEHP and DIDP ranking first and second, respectively.

9. Lington, A.W., Bird, M.G., Plutnick, R.T., Stubblefield, W.A., Scala, R.A. (1997). Chronic Toxicity and Carcinogenic Evaluation of Diisononyl Phthalate in Rats. *Fundamental and Applied Toxicology*, 36:79-89.

Fischer 344 rats of both sexes were fed DINP (CAS No. 68151-48-0) in their diet at concentrations of 0, 0.03, 0.3, and 0.6 wt% for up to two years. Based on body weights and food consumption, the mean daily dose of DINP was estimated to be: for male rats - 15, 152, and 307 mg/kg/day corresponding to 0.03, 0.3, and 0.6 wt%, resrectively, and for female rats - 18, 184, and 375 mg/kg/day, respectively. Animals were sacrificed at 6, 12, 18, and 24 months. In a previous study (Bird *et al.*, 1986, see above I.A.2.) where Fischer 344 rats were fed DINP for 13 weeks, significant changes, including peroxisome proliferation, were observed at the higher doses (1.0 and 2.0%) and less so at lower doses, with peroxisome proliferation only weakly observed at the mid-level dose of 0.6% DINP. Hence, 0.6% DINP was chosen as the highest level in this study because of concern that higher dose levels might impact the survival of the animals. The authors stated that this study had three main objectives:

1. assess the chronic toxicity and oncogenecity of DINP at a dose close to the maximum tolerated dose of 0.6 wt%;
2. assess whether DINP could cause hepatic peroxisome proliferation under chronic exposure conditions; and
3. to ascertain a no-observed-effect level (NOEL) for DINP under chronic exposure conditions.

The following observations were made:

- for mid and high dose groups -
  - slight decreases in food consumption;
  - decreases in body weight, which were statistically significant in males;
  - an increase in mortality with both sexes, but whether the increases were statistically significant or not depended on the statistical method used;
  - statistically significant dose-related increases in relative liver and kidney weights in both sexes;
  - effects on urinalysis, hematologic, and clinical chemistry parameters;
  - liver lesions were observed in both sexes (centrilobular to midzonal hepatocellular enlargement);
  - kidney lesions were observed (increase in tubular cell pigment in the tubular epithelium) in high dose male rats;
  - statistically significant increase in mononuclear cell leukemia (MNCL) and changes known to be associated with MNCL;
- no treatment related effects on the testes;
- no peroxisome induction was observed in the liver of treated versus control rats based on electron microscopy.
- a clear no-observed-effect level for all biological endpoints at 0.03 wt% of DINP, which corresponds to approximately 17 mg/kg/day.

The authors state that the literature suggests that an increase in the incidence of MNCL in aging Fischer 344 rats is a common finding and, while the increases observed in this study were statistically significant as compared with controls, the highest incidence (64%) observed in this study was below the highest historical incidence (72%) reported for male

rats. The authors also believe that an increase in MNCL in rats is not relevant to humans based on the fact that prevailing data indicate that the MNCL tumor type seen in rats is not seen in humans and that MNCL appears to arise from the spleen in rats whereas it arises from effects on bone marrow in humans.

10. Myers, B.A. (1991a). A Subchronic (4-Week) Dietary Oral Toxicity Study of Di(isononyl)phthalate in B6C3F1 Mice. *Hazelton Washington, Inc.*, U.S. Environmental Protection Agency Document I.D. 86-910000793, Microfiche No. OTS0529425.

DINP (CAS No. 28553-12-0) was fed in the diet to B6C3F1 mice of both sexes for at least four weeks at dose levels of 0, 3,000, 6,000 12,500, or 25,000 ppm. The author observed the following:

- all animals survived to the end of the study;
- no treatment-related clinical signs;
- body weights were significantly lower than controls in the highest dose group of both sexes from Weeks 3-5;
- increase in serum alanine aminotransferase activity in the highest dose group of both sexes;
- increase in blood urea nitrogen level of the highest dose male group;
- significant dose-related decreases in absolute kidney and testes/epididymous weights in males dosed at 6,000, 12,500, or 25,000 ppm;
- significantly increased liver/gallbladder weight parameters were observed in all groups of treated animals except those treated at the lowest dose of 3,000 ppm;
- dose-response related hepatocellular enlargement in livers of females treated at 6,000 ppm or higher, and all treated male groups;
- focal coagulative necrosis and/or separate chronic inflammatory foci in livers of the highest dose group of males and at a lower incidence in females at the two highest dose levels;
- tubular nephrosis in kidneys of all males and females at the highest dose;
- lesions of the reticuloendothelial system in the highest dose group of both sexes including atrophy of the spleen and lymphoid depletion in the thymus;
- lesions of the epididymous (increased cellular debris) in all males at the highest dose; and
- lesions of the reproductive tract (absence of corpora lutea in the ovaries and atrophic uteri) in all females at the highest dose.

The author states that in this study, the target tissues were liver, kidney, and testes, and that the results also suggest that effects on the ovaries, uterus, and the reticuloendothelial system may be treatment-related as well, although not as obviously so. The ovarian and uterine effects have been observed in female mice that have been food-restricted to a similar degree as that which occurred in the highest dose group of females, in which these symptoms were observed.

11. Shellenberger, T.E., Kowalski, J.J., Unwin, S., Grandjean, C., Carter, J., and Hodgson, J.R. (1983). Comparative 28-Day Oral Toxicity of Selected Phthalate Esters. *The Toxicologist*, 3:157. (Note: This is an abstract.)

Fischer 344 rats were fed di-(2-ethylhexyl)-phthalate (DEHP), DINP, or Di-(C_{7-11})phthalate ($D_{7-11}P$) in their diets for 28 days at levels of 0, 0.2, 0.67, or 2.0%. The authors observed the following in the case of DINP treated rats:

- significant dose-related increases in absolute and relative liver weights of both sexes in the mid and high level groups;
- no histopathological changes in the livers upon microscopic examination;
- significant reduction in serum triglyceride levels in males in the mid and high dose groups;
- significant increase in hepatic catalase activity in both sexes; and
- increased carnitine acetyltransferase (CAT) activities in all treatment groups.

The authors stated that the relative increases in CAT activities were greater than with catalase suggesting that the former may be a more sensitive indicator of peroxisome proliferation in the liver.

12. Zeiger, E., Haworth, S., Mortelmans, K., and Speck, W. (1985). Mutagenicity Testing of Di(2-ethylhexyl)phthalate and Related Chemicals in *Salmonella*. *Environmental Mutagenesis* 7:213-232.

Di(2-ethylhexyl)phthalate and 33 other related compounds, including DINP (CAS No. 28553-12-0), were tested for mutagenicity using several *Salmonella* strains without metabolic activation and in the presence of rat and hamster liver S-9 metabolic activation systems. Each chemical was tested at five dose levels. No mutagenicity was observed in *Salmonella* with or without metabolic activation.

B. Articles on DINP Not Reviewed in the "Annotated Bibliography", but Cited in the Consumer Product Safety Commission (CPSC) Report[1]

(A brief summary of CPSC's description of the results in each report is included.)

1. David, R.M. (1997). Understanding Phthalate Esters and Endocrine Disruption. Seminar presented by Raymond M. David, Chemical Manufacturers Association, Phthalate Esters Panel, October 29, 1997.

Reproductive toxicity of DINP was studied in male rodents. No effects at doses up to 930 mg/kg/day, the highest dose used, were reported.

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[1] Consumer Product Safety Commission. (1998). Preliminary Hazard Assessment of Diisononyl Phthalate (DINP) in Children's Products, Memorandum from Michael A. Babich to Ronald L. Medford, March 10, 1998.

2. Experimental Pathology Laboratories, Inc. (1982). One-week Prechronic Oral Feeding Study in F-344 Rats. Pathology Report. July 1982. EPA Document No. 87-8210916.

Fischer rats were fed 2% DINP for one week. No adverse effects were reported.

3. Lington, A.W., Bird, M.G., Plutnick, R.T., Stubblefield, W.A., Scala, R.A. (1993). Short-term Feeding Studies Assessing the Testicular Effects of Nine Plasticizers in the F344 Rat. *Pharmacology and Toxicology*, 73:132.

Male Fischer rats were dosed with DINP for three weeks in order to study testicular effects of the compound. No atrophy or histopathological changes were observed at 2.5% DINP (about 2,500 mg/kg/day), the highest dose used.

4. Myers, B.A. (1991b). A Subchronic (13-Week) Dietary Oral Toxicity Study of Di(isononyl)phthalate in Fischer 344 Rats. *Hazelton Washington, Inc.*, U.S. Environmental Protection Agency Document I.D. 89-920000224.

Rats were dosed with DINP for 13 weeks. The following effects were observed:

- increase in relative liver and kidney weights at 2,500 ppm in males and 5,000 ppm in females; and
- histopathological effects in the liver, kidney, and glandular stomach at 5,000 ppm in males and 10,000 ppm in females.

The 2,500 ppm lowest-observe-effect level (LOEL) approximates 140 mg/kg/day. (It is unclear whether this is CPSC's calculation or the author's.)

5. U.S. Environmental Protection Agency. (1986). A Chronic Toxicity Carcinogenicity Feeding Study in Rats with [DINP-4]. *Bio/dynamics, Inc.* Project number 81-2572. June 20, 1986. EPA Document No. 86-870000362.

Sprague-Dawley rats were dosed with DINP for two years. According to CPSC, this version of DINP was never produced commercially. The following was observed:

- a statistically significant increase in hepatocellular carcinoma (HCC) in females at the two highest doses, 331 and 672 mg/kg/day;
- increase in HCC in males at the two highest doses, 271 and 553 mg/kg/day, but the increase not statistically significant although in excess of historical controls;
- liver necrosis in male rats at the dose level of 30 mg/kg/day.

LOEL was established at 30 mg/kg/day. No NOEL was established.

6. U.S. Environmental Protection Agency. (1992). Supplement: 13-week Subchronic Dietary Oral Toxicity Study with Di(isononyl)phthalate in Mice with Cover Letter Dated 070692 and Attachments. July 1992. EPA Document No. 89-920000303. (It should be noted that CPSC discusses both this study and the Myers (1991a, see above I.A.9.) 4 week study in mice in such a manner that it is difficult to separate

completely what was observed in each.)

B6C3F1 mice were fed DINP in their diet for 13 weeks. The following were observed:

- statistically significant differences in relative organ weights and/or histopathological changes in liver, kidney and other sites of treated animals;
- reduced relative weights of the testes, although histologically normal;
- no adverse effects at 4,000 ppm.

II. Articles and Reports on Leaching of DINP from Children's Products

A. Articles and Reports Reviewed in the Annotated Bibliography and/or Cited by CPSC[2]

1. Denmark

a. Vikelsoe, K., Jensen, G.H., Johansen, E., Carlsen, I., and Rastogi, S.C. (1997). Migration of Phthalates from Teething Rings. Environmental Chemistry Department, Danish Environmental Testing Agency, April 15, 1997.

Eleven teething rings were tested for migration of various phthalates. Approximately one gram of each sample was shaken in synthetic saliva, pH 5.0, at 37°C for 20 hours. Regarding DINP, it was released from three of the rings in amounts of 89, 10,356, and 24,691 ug/g of plastic. The authors state that the phthalates released under these particular experimental conditions should be regarded as minimum amounts which may be released during actual chewing conditions.

b. Vikelsoe, K., Jensen, G.H., Johansen, E., Carlsen, I., and Carlsen, L. (1997). Migration of Phthalates from Teething Rings. Environmental Chemistry Department, Danish Environmental Testing Agency, July 2, 1997.

(Below is a summary of this study which was cited in the CPSC report, but not reviewed for this Annotated Bibliography.)

DINP was released from one teether at a rate of 558,233 ug/dm^2/day, which is equivalent to 232.6 ug/cm^2/hour. The release rate of DINP from two other teethers was less than 0.01 ug/cm^2/hour.

2. Netherlands

a. Barnard, H. (1997). Letter from Herbert Barnard, Counselor for Health and

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[2] Consumer Product Safety Commission. (1998). Preliminary Hazard Assessment of Diisononyl Phthalate (DINP) in Children's Products, Memorandum from Michael A. Babich to Ronald L. Medford, March 10, 1998.

Welfare, Royal Netherlands Embassy, Washington, DC, to Michael A. Babich, Directorate for Epidemiology and Health Sciences, U.S. Consumer Product Safety Commission. October, 16, 1997.

In the CPSC report, this cite was given referring to a study by the Netherlands Health Protection Inspectorate. The release of phthalates was tested using artificial saliva in an ultrasound bath. Six teethers and toys were tested. Results showed that the items contained between 43 to 49% DINP and that DINP was released at a rate from 7.9 to 31.4 $ug/cm^2/h$.

b. RIVM, National Institute of Public Health and the Environment. (1998). Phthalate Release from Soft PVC Baby Toys. Report from the Dutch Consensus Group, RIVM Report 613320 002, September 1998.

The results of several studies are included in this report, including a study designed to measure the release rates of DINP into saliva from PVC samples. Adult human volunteers were used. Three different samples were employed, each 10 cm in total area: Specimen #1 - disc of a standard PVC sample containing 38.5% DINP; Specimen #2 - finger of a commercially available teething ring; and Specimen #3 - punched flat part of the teething ring (i.e., Specimen #2). A control specimen was also employed in order to obtain "blank" saliva.

The authors state the following:

- differences between the way in which adults and children bite and suck was not considered to impact the results in a significant way;
- differences in composition of saliva between volunteers did not have an impact on the release rate;
- differences in saliva between children and adults might be more significant, but given that there was no quantitative information on this, such differences were ignored;
- it was not technically feasible to measure the amount of DINP adsorbed to the surface of the oral cavity and only the content of the saliva was used to measure DINP release rates, which could potentially yield an underestimation of the amount released and absorbed; and
- young children do not swallow all the saliva they produce.

The following release rates (mean and range) of DINP were determined:

Specimen #1 - mean = 1.38 ug/minute, range 0.3-8.3;
Specimen #2 - mean = 2.44 ug/minute, range 0.9-8.9;
Specimen #3 - mean = 1.63 ug/minute, range 0.9-5.7.

The authors state that while releases from Specimens #1 and #3 differed little, the higher values of Specimen #2 might be due to the different shape of the specimen (finger vs. disk) which might result in a different interaction in the mouth during sucking and biting.

3. United Kingdom

Pindar, A., Barwick, V., and Cody, M. (1993). The Leaching of Phthalate Ester Plasticizers from Teethers and Toys. Consumer Hazards Group, Laboratory of the

Government Chemist, UK, Presented at the Conference of International Product Safety Research, Amsterdam.

A dynamic versus static leaching test was developed to assess leaching of phthalates from children's products. A portion of each toy was placed in a glass jar with artificial saliva (pH 4.5-5), pellets were added, and the jar was sealed. The jar was then rotated in a "head-over-heels" manner for six hours. All portions underwent a repeat extraction. Other toy portions underwent a static leaching test at the same time, letting the jar stand for 6 hours. Also tested was the total content of the plasticizers being studied in the children's products.

It was observed that polyvinyl chloride products in this study usually contained 40 to 50% phthalates.

The following was observed regarding DINP:

- in the dynamic leaching test, DINP release rates ranged from 3.89 to 4.51 mg/dm^2 at the 95% confidence limit, with a mean of 4.2 mg/dm^2;
- the amount of DINP released during the second extraction was not significantly different from the amount released during the first extraction; and
- DINP was detected in some of tests, but levels were close to detection limit.

The authors state that agitation increases considerably the amount of phthalate ester released and that given that the amount of phthalate released does not decrease on subsequent extractions, phthalate is likely to be available for ingestion for the lifetime of the toy.

4. Greenpeace

Greenpeace. (1997). Excerpt from "Children's Toys Made of Plasticized PVC".

Over 20 samples of children's toys were analyzed for leaching of phthalate esters. The samples were submerged in deionised water with a temperature of 40°C for one hour and 24 hours. Several items were found to leach DINP at levels of 0.74 to 14 mg/dm^2.

5. European Union

EU Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE). (1998). Phthalate Migration from Soft PVC Toys and Child-care Articles. Opinion Expressed at the CSTEE Third Plenary Meeting in Brussels, 24 April 1998.

See "Section 3.2.4 Leached Amounts" for a listing of additional studies reporting leaching of DINP from toys.

III. Reports by the Consumer Product Safety Commission (CPSC) and the European Union's Committee on Toxicity, Ecotoxicity and the Environment (CSTEE)

A. Consumer Product Safety Commission (CPSC)

Consumer Product Safety Commission. (1998). Preliminary Hazard Assessment of Diisononyl Phthalate (DINP) in Children's Products, Memorandum from Michael A. Babich to Ronald L. Medford, March 10, 1998.

This report reviewed some of the literature on the toxicity of DINP as well as potential exposure of children to DINP via leaching of DINP from toys. The report makes clear that DINP is actually a mixture of diisononyl phthalate isomers and that the particular mixture can vary between manufacturers, processes, and batches.

CPSC derived an Acceptable Daily Intake (ADI) based on non-carcinogenic effects of DINP reported by Lington et al. (1997) and Myers (1991b). Based upon a NOEL of 17 mg/kg/day derived from Lington et al. and a LOEL of 140 mg/kg/day from Myers, CPSC derived an ADI of approximately 0.15 mg/kg/day. Based upon CPSC's review of some of the leaching studies of DINP by the Netherlands and Denmark, the highest exposures calculated would exceed this ADI.

CPSC states that the hepatocellular carcinoma reported as a result of exposure to DINP (U.S. EPA, 1986) was not used to derive a cancer potency for DINP because the particular type of DINP used in this study was never produced commercially. The mononuclear cell leukemia observed in the study by Lington et al. (1997) was not used either because of the high background incidence of this lesion in control animals and because the relevance of this lesion to humans is unknown.

B. EU Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE).

EU Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE). (1998). Phthalate Migration from Soft PVC Toys and Child-care Articles. Opinion Expressed at the CSTEE Third Plenary Meeting in Brussels, 24 April 1998.

This report covers the migration of numerous phthalates from children's toys, including DINP. Leaching rates of DINP as reported by various investigators are presented (see Section 3.2.4) as are studies relevant to DINP's toxicity (see Section 4.2.1). The CSTEE derived a no-observed-adverse-effect level (NOAEL) for DINP of 15 mg/kg/day based upon the increased liver and kidney weights observed in the study by Lington et al. (1997). The CSTEE recommended that a Tolerable Daily Intake (TDI) be derived based upon a margin of safety of 100 for NOAELs, thus yielding a TDI of 0.15 mg/kg/day for DINP, the same as that derived by CPSC.

According to CTSEE's calculations, using maximum leaching rates from the reports reviewed and based on certain intake assumptions, the margin of safety for DINP in toys was 8.8 versus the recommended 100.

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Report on DINP Phthalate:

Summary of the Published Scientific Literature and Quantities Found in Common Toys

November 1998

Prepared by Thomas Natan, Ph.D.
Director of Research
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I. The Health Effects of DINP Phthalate

Summary

Studies consistently indicate that diisononylphthalate—DINP phthalate—demonstrates chronic toxicity. The most consistent and well documented toxicity effects appear to be liver and kidney damage. The Consumer Product Safety Commission (CPSC) has based its "acceptable daily intake levels" for the chemical on studies looking at liver and kidney toxicity.

DINP is carcinogenic at higher dose levels, but more studies are necessary to determine potential human cancer risk. Until then, the CPSC has listed DINP as a "possible" human carcinogen. Studies have found birth defects and effects on the reproductive system at higher doses as well as potential interference with the hormone system.

Introduction

Phthalates are a class of chemicals used in polyvinyl chloride (PVC) plastic to make the plastic more flexible and provide greater chemical stability. DINP is the most commonly used phthalate in soft plastic vinyl children's toys. As with all phthalate plasticizers, DINP leaches from plastic products, providing the potential for human exposure, especially for infants and young children who put plastic toys in their mouths.

DINP has been the focus of two major reviews: one by the U.S. Consumer Product Safety Commission (CPSC), which examined toxicity and leaching of DINP from children's products, and one by the European Union Scientific Committee on Toxicity, Ecotoxicity, and the Environment (CSTEE), which focused on toxicity and leaching from PVC of a number of phthalates, including DINP. Using the CPSC and CSTEE studies as a starting point, many of the most relevant studies cited in those reports were reviewed. The summaries of the reports reviewed are based directly on the authors' statements and data.

Any attempt to provide a complete characterization of the toxicity of DINP is complicated by the fact that DINP is not a single substance but consists of many compounds in a mixture. The mixtures vary not only from manufacturer to manufacturer but even between batches made by a single manufacturer. Although these differences may not affect the chemical's performance in plastic, they will likely impact the toxicity of a particular mixture. As stated in the CPSC report, it is entirely possible that there are mixtures available that have greater toxicity than those used in the studies (see Addendum I).

Findings

Chronic and Sub-Chronic Toxicity

The most consistent toxicity findings appear to be kidney and liver damage. This included lesions on both organs and increases in absolute and relative kidney and liver weights. These effects were observed to various extents with different DINP mixtures and at dose levels lower than those at which most carcinogenic effects were observed.

In a study by Lington, *et. al.* (I.A.9), the maximum dose was set at a level below which liver cancer was observed, but liver and kidney damage were still found. Other studies also tested doses below this level and found similar organ damage. The CPSC used the "no observed effects level" of 17 mg/kg/day established by Lington as well as the "lowest observed effects level" of 140 mg/kg/day established by Myers *et. al.* (I.A.10) as the basis for its "acceptable daily intake" of 0.15 mg/kg/day. This is the amount that the CPSC believes can be safely ingested by humans without showing toxic effects.

Carcinogenicity

DINP appears to be carcinogenic in rats and mice at the higher doses examined in the studies, which found development of liver cancer. But the relevance of these findings to humans is not yet clear. Mononuclear cell leukemia was observed in Lington's study (I.A.9) at doses lower than those at which liver cancer was observed. Mononuclear cell leukemia occurs relatively frequently among older rodents, and the relevance of these findings to humans is uncertain. Based on these studies, the CPSC considers DINP a "possible" human carcinogen for those mixtures it determined to be commercially available. As noted above, it is impossible to know whether the full range of commercially available DINP has been covered in these studies.

Developmental Toxicity and Estrogenic Activity

With one study on each of these effects, neither developmental toxicity nor estrogenic activity of DINP has been well studied. Hellwig (I.A.7) studied rats exposed to three DINP mixtures and found some developmental toxicity (birth defects) at a level more than 60 times higher than Lington's "no observed effects level" of 17 mg/kg/day. Toxicity occurred in the mother at this level, making it difficult to determine if DINP was acting on the mother or directly on the fetus. Toxic effects on the mother could cause fetal toxicity apart from direct effects of the chemical on the fetus itself. Likewise, Harris (I.A.6) tested a number of phthalate esters for estrogenic activity and concluded that DINP was extremely weakly estrogenic *in vitro*.

Reproductive Toxicity

Myers *et. al.* (I.A.10) found male and female reproductive toxicity at higher dose levels, including lesions of the epididymous in male mice and of the reproductive tract in females. However, the dose levels were significantly higher than the "lowest observed effects level" of 140 mg/kg/day established by Myers in the same study. There was also a decrease in testicular weight at dose levels equal to the maximum dose in the Lington study (I.A.9).

II. The Extent to which Children May be Exposed

There have been at least six studies looking at the rate at which DINP leaches out of soft plastic vinyl. **The attached table demonstrates that four of those six studies found daily intake levels higher than what the U.S. Consumer Product Safety Commission and the European Union's "Toxicity Committee" identify as acceptable. The CPSC has also reported this finding.**

There is a wide variation in the results of the studies. One study from the United Kingdom (II.A.3) suggests why: dynamic leaching tests (using gumming and chewing motions) produced a greater leaching rate than static tests. (The study also found that the amount of phthalate leaching does not decrease in subsequent extractions, making it likely that these substances will be available for leaching over the lifetime of the toy.)

Every study reported its results in different units, making direct comparison difficult. NET normalized leaching rates to a single exposure scenario and found several reported rates that translate to potential exposure exceeding the CPSC's and the European Union's "acceptable daily intake" (ADI) of 0.15 mg/kg/day.

Conversion of Various DINP Leaching Rates to Daily Exposure Rate

Study	Leaching Rate Found	Units Leaching Rate was Reported In	Average Daily Intake- (mg/kg/6hours)	Potential Exposure
Netherlands, Barnard, 1997	9 - 35	ug/cm²/hour	0.07 - 0.26	**Over 0.15 ADI**
Denmark, Artsana Corporation, 1997	0.09 - 0.29	mg/kg/6 hours	0.09 - 0.29	**Over 0.15 ADI**
United Kingdom, Pindar, et al., 1993	3.89 - 4.51	mg/dm²/6 hours	0.05 - 0.06	Under 0.15 ADI
Spain, Ministry of Health, 1997	n.a. - 0.02	mg/cm²/hour	n.a. - 0.16	**Over 0.15 ADI**
Greenpeace, 1997	0.85 - 14	mg/dm²/hour	0.06 - 1.05	**Over 0.15 ADI**
Germany, European Chemical Laboratory Assoc., 1998	<0.05 - 0.25	mg/dm²/24 hours	<0.00015 - 0.00078	Under 0.15 ADI

Assumptions made for calculating average daily dose: 1) Toy area of 10cm² 2) Child sucks on toy for 6 hours per day, 3) Child weighs 8kg.

III. Toy Content Analysis

Several European soft vinyl toys have already been analyzed for their content of the phthalate DINP. Two major studies out of the Netherlands (Barnard 1997, RIVM 1998) found DINP levels in several teethers and toys of between 38 and 49 percent by weight. Such high levels of DINP are due to the fact these levels are needed to achieve the softness and pliability that toy manufacturers require.

NET conducted one of the first broad analyses of DINP levels in U.S. toys. NET analyzed DINP levels in 33 soft plastic vinyl toys purchased in the U.S. The toys were analyzed by STAT Analysis Corporation, a private, independent, EPA-certified laboratory located in Chicago, Illinois.

NET tested toys or parts of toys that are capable of being put in a child's mouth. STAT's testing methodology included complete dissolution of the toy or toy part and analysis of that solution by Gas Chromatograph/Mass Spectroscopy—a standard method of chemical identity and content analysis.

NET tested two groups of toys:

1) Toys manufactured by large, well known toy companies and available for purchase nationwide at large retailers such as Wal-Mart and Toys R Us.

2) Toys manufactured by small, less well known manufacturers and purchased in discount and "dollar" stores in select metropolitan areas.

NET found comparable levels of DINP in both sets of toys ranging from 17 to more than 40 percent by weight. Results are summarized in the table on the next page.

Toy Content Analysis

Manufacturer	Store/City	Toy	Part Tested	% DINP by Weight
Major Brands				
Playskool	Wal-Mart	Snuzzles Very Soft Baby Doll	Face	39.7
Mattel	Wal-Mart	Rugrats Tommy Pickles Ice Cream Face Doll	Arms	30.2
Mattel	Wal-Mart	Cabbage Patch Kids Starr Rosie Doll	Shoes	34.1
Tyco	Wal-Mart	Sesame Street Big Bird Softy Sounds Phone	Bird	26.3
Mattel	Wal-Mart	Shopping Time Barbie Wal-Mart Edition	Face	35.9
Kenner/Hasbro	Wal-Mart	Star Wars Princess Leia Action Doll	Dress	25.8
Playskool	Toys R Us	Teletubbies Laa-Laa Squeezie	Whole	16.9
Mega Blocks	Toys R Us	Baby Soft Blocks Squeezie	Bear	32.7
Warner Brothers	Toys R Us	Little Squeakers Baby Bugs	Whole	23.5
First Years	Toys R Us	Sesame Street Cool Teether Ring	Whole	30.0
Funomenon	Toys R Us	Sesame Street Talking Cup Lid	Hat Brim	24.2
Hasbro	Toys R Us	My Little Pony Sky Skimmer	Leg	21.8
McDonaldland	Toys R Us	Happy Meal Hamburger Buns	Whole	26.8
Mattel/Disney	Kay Bee	Daisy Duck Squeezie	Whole	28.2
Mattel	McDonald's	Barbie Happy Meal Toy	Arms	22.8
Dollar Store Brands				
Baby King	Baton Rouge	Squeak Book	Whole	26.2
Children Toy	Philadelphia	Squeeze Bear	Whole	36.9
Nursery Needs	New York City/99 Cent-Inwood	Blue Hand Teether	Whole	40.5
Grow Time	Chicago	Squeeze Toy	Whole	37.1
Baby First Steps	Chicago, Detroit	Squeeze Helicopters	Whole	34.0
Safety Toys	San Francisco	Squirting Fish	Whole	21.9
Baby King	Philadelphia, Baton Rouge	Squeeze Bears	Whole	31.6
Baby Buzz	Detroit/99 Cent-Grand River	Teething Gloves (yellow)	Whole	17.3
Safety 1st	Albuquerque/Family Dollar	Teethers	Whole	32.7
Baby King	Baton Rouge	Boat, Key Ring Teethers	Soft Area	29.2
Crib Mates	Baton Rouge	Squeeze Toys (animals, numbers)	Whole	40.9
Baby's Best	Detroit/Dollar Super Mart	Soft Teether Ring (on string)	Whole	17.4
Baby World	Baton Rouge	Baby Teething Beads	Whole	49.3
Tutti Fruiti	New York City/99 Cent-Inwood	Elephant Squeeze Toy	Whole	34.1
Baby's 'n Things	Miami	Baby's Sailor Duck	Whole	38.1
Honey Baby	Miami	Turtle Toy	Whole	44.7
	Miami	L'il Stone Ager Dinosaur	Whole	43.1
Kids 2 Grow	Miami	Round Soother	Whole	26.5

Data Source: STAT Analysis Corporation

Data summarized by National Environmental Trust, November 1998

Toxic Chemicals in Vinyl Children's Toys

Warning: Children at Risk
Toxic Chemicals Found in Vinyl Children's Products

By: Joseph Di Gangi, PhD
Greenpeace USA
November 1998

A 1998 Greenpeace investigation revealed the presence of a toxic chemical in popular vinyl children's products. The products were purchased in September from national chain stores and included items designed for infants and toddlers such as First Years teethers, Gerber nipples, a Hasbro Teletubbies doll, and a Sesame Street bib.

The chemical is DINP (diisononyl phthalate), a member of a family of compounds called phthalates. Manufacturers warn laboratory scientists about DINP as follows: "harmful by inhalation, in contact with skin and if swallowed," "possible risk of irreversible effects," and "may cause cancer."[1] In contrast, toy makers label vinyl children's toys containing large amounts of DINP as "non-toxic."

Table 1 shows the latest Greenpeace test results on an assortment of popular vinyl children's toys sold in the United States. The vinyl products contained between 3 to 41 percent DINP by weight. Recently, NBC in Chicago (WMAQ) also found DINP in a variety of popular vinyl teethers and toys.[2] A 1997 Greenpeace study of vinyl toys from 17 countries found that most of them contained 10-40 percent DINP by weight.[3]

Since vinyl is a hard, brittle plastic, chemical softeners like DINP are required to make it pliable. DINP is not chemically attached to the plastic, and government studies show that DINP and other phthalates are easily released from vinyl toys into saliva.[4, 5] The toy industry and phthalate manufacturers admit that vinyl leaches phthalates.[6, 7]

DINP is not acutely toxic. However, animal studies indicate that longer exposures to DINP damage the liver and kidneys and affect reproductive organs.[8, 9, 10, 11] In addition, DINP weakly mimics estrogen in laboratory tests, indicating possible endocrine disruption.[12]

DEHP is another member of the phthalate family that was widely used in toys until government agencies found in the early eighties that it was carcinogenic.[13] Instead of recalling vinyl toys, the Consumer Product Safety Commission (CPSC) quietly entered into a voluntary agreement with the toy industry to limit DEHP content to 3 percent.[14] This agreement caused toy makers to shift from DEHP to DINP as the chemical softener in vinyl toys. However, DINP was not tested by the CPSC for safety.

In March 1998, the CPSC issued a preliminary report on DINP.[15] The report used data from DINP manufacturers (including Exxon and BASF) to conclude that DINP was not carcinogenic or a reproductive toxicant. However, the agency admitted that "...DINP

(various types) may be considered 'probably toxic to humans,' based on sufficient evidence in animals."

In addition, the CPSC revealed that there are up to 100 isomers or chemical variations of DINP and that only five have been studied. One type of DINP was carcinogenic, but the CPSC claimed that it was never commercialized. However, the agency did warn that "It is conceivable that one or more existing types of DINP for which data are unavailable could also be more toxic and/or carcinogenic."

Four European countries are planning or have already implemented bans on DINP and/or vinyl toys (Sweden, Denmark, Austria, and Norway). The CPSC has not "regulated" phthalates in children's products since the 1986 agreement with the toy industry.

The current regulatory system does not protect children from exposure to toxic chemicals. Each toxic chemical added to vinyl is "innocent until proven guilty." It is time to implement a materials-based regulatory policy that declares vinyl an inappropriate material for children's products since it requires so many toxic additives to make it useful. Such a policy would have prevented the switch from DEHP to DINP and would prevent the substitution of another potentially toxic softener to replace DINP. A materials-based regulatory policy would be proactive instead of reactive, precautionary instead of reckless, and efficient instead of disorganized. The wide availability of safer, alternative materials makes eliminating vinyl children's products a realistic goal and an ethical obligation.

Recommendations

1. Parents should not purchase vinyl or PVC products to which children might be exposed. Vinyl children's products should be returned to the manufacturer or retailer.

2. Retailers, distributors, and manufacturers should remove vinyl children's products from the market.

3. The Consumer Product Safety Commission should regulate vinyl as a material. Vinyl children's products containing phthalates, lead, cadmium, or other untested or toxic additives should be recalled by the agency.

4. Retailers and trademark licensers should not market vinyl products to which children might be exposed. This should be made explicit in future sales and licensing agreements.

5. The plastics industry and toy manufacturers, through trade associations such as the Society of Plastics Industry and the Toy Manufacturers of America, should adopt an industry-wide standard against manufacturing children's products with vinyl.

Table 1. DINP in Vinyl Children's Products

Item	Store	Phthalate	Amount (%)
Baby Looney Tunes Cooling Ring Teether	Target, Mountainview mfr: Gerber	DINP	34
Cool Ring Teether by The First Years	Toys R Us, Santa Monica mfr: Kiddie Products	DINP	32
Big Bird Soft Sports Car SS Soft Squeeze VEH/24	Mattel Toy Club, El Segundo mfr: Tyco/Mattel	DINP	31
Disney Touch N Surp/4, Baby Minnie doll	Mattel Toy Club, El Segundo mfr: Mattel	DINP	22
Gerber Nipples; Bright Clear & Soft	Target, Mountainview mfr: Gerber	DINP	3.2
Hair Raising Angelica, Rugrats Doll AST/6	Mattel Toy Club, El Segundo mfr: Mattel	DINP	31
Mickey Mouse Vinyl Coverall; 100% PVC	Toys R Us, Santa Monica mfr: Disney Babies, Evenflo Co.	DINP	26
Munchkin Spoon White Hot	Target, Mountainview mfr: Munchkin Inc.	DINP	7.5
Pooh Baby 1st Blocks	Mattel Toy Club, El Segundo mfr: Arco Toys/Mattel	DINP	32
Sesame Street Tie Neck Vinyl Bib, Big Bird	Target, Mountainview mfr: Playskool/Hasbro	DINP	27
Squeeze'Ems Elmo SS Squeeze Toy AST/24	Mattel Toy Club, El Segundo mfr: Tyco/Mattel	DINP	35
Teletubbies Plush Po; red	Toys R Us, Santa Monica mfr: Playskool/Hasbro	DINP	41
Three Little Froggies; soft vinyl toys; non-toxic	Nana, Santa Monica mfr: Knobler Int.	DINP	34

Abbreviations: mfr = manufacturer. All products were purchased in California in 1998. Determination by Stat Analysis, Chicago, IL and First Environmental, Naperville, IL. Amounts are expressed as percent by weight. Results shown are the averages of duplicates. The average coefficient of variation was 9%.

Materials and Methods

Sample preparation was conducted by Stat Analysis, Chicago, Illinois AIHA, NIST /NVLAP accredited. Samples were prepared according to ASTMD3421, which was modified to avoid using chlorinated solvents.[16] Briefly, samples were frozen in liquid nitrogen and grated to a particle size of approximately 3 mm^3.

DINP content was determined using GC/MS by First Environmental, Naperville, Illinois IEPA and IDPH certified. Analysis was performed essentially according to EPA Method 8270C. Testing was conducted in duplicate using a single blind design to prevent possible bias introduced by product recognition.

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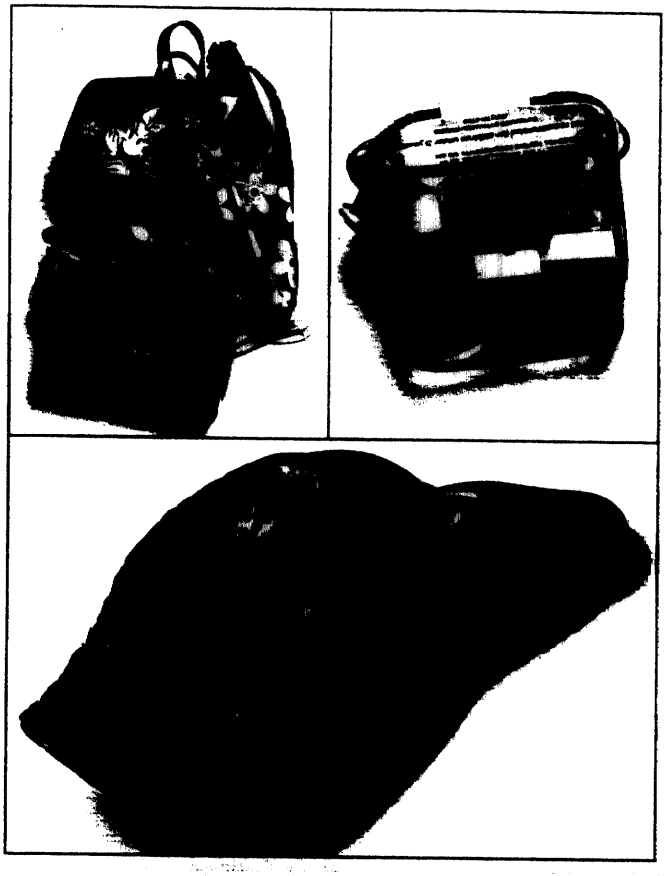
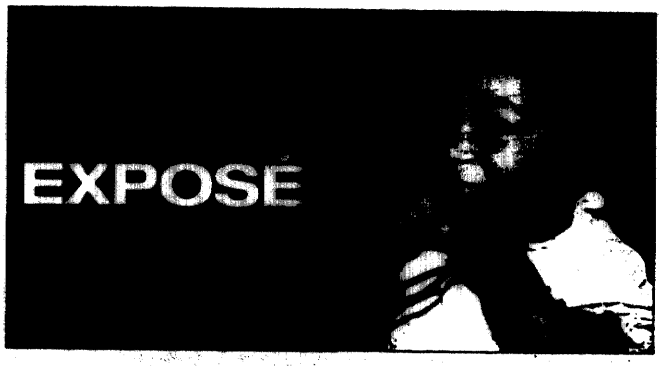


LEAD AND CADMIUM IN VINYL CHILDREN'S PRODUCTS

by Joseph Di Gangi, PhD



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Lead and Cadmium in Vinyl Consumer Products

A Greenpeace Exposé

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Summary

A Greenpeace investigation revealed the presence of hazardous levels of lead and cadmium in a variety of vinyl consumer products, including items specifically designed and marketed for children. The study was spurred by the discovery of hazardous lead levels in vinyl blinds and associated lead poisoning of children in 1996. Since no government agency appeared to be investigating whether other vinyl consumer products might also pose a similar health risk, Greenpeace initiated a nationwide study of vinyl products.

The Greenpeace investigation discovered:

- Lead is present in vinyl products throughout the US. A survey of 10 major US cities showed the presence of many lead-containing products. They were also found in Montreal, Quebec, Canada.
- Lead- and cadmium-containing vinyl products are readily available from some of the nation's leading retailers, including Kmart, Wal-Mart, Target, and Toys R Us.
- Children are a marketing target. Products featured Barbie, Minnie and Mickey Mouse, 101 Dalmations, Michael Jordan, Bugs Bunny, and various other Looney Tunes characters.
- Lead-containing vinyl products are common. The investigation began by testing vinyl children's products purchased in Chicago. Twenty-one percent of the vinyl consumer products examined contained greater than 100 parts-per-million lead (28 out of 131). Since all of the items were purchased at national chain stores, this high percentage may reflect the US frequency for lead-containing vinyl products.
- The lead levels are hazardous. Eighteen percent of the products examined violated the limit advocated by the Consumer Product Safety Commission staff for vinyl miniblinds.
- Cadmium is an added hazard. In every case in which it was measured, all of the lead-containing products also contained varying levels of cadmium, a known carcinogen and renal toxin which is not regulated in children's products by the Consumer Product Safety Commission.
- Lead- and cadmium-containing vinyl products are widely available in California, despite its stringent regulation of carcinogens and reproductive toxins. A representative sample of items grossly exceeded limits for exposure to lead and cadmium set by the Safe Drinking Water and Toxic Enforcement Act of 1986, also known as Proposition 65.
- Both lead and cadmium are readily available for ingestion by children. Lead and cadmium were released to the surfaces of products as they aged. Lead also became available under conditions that mimic accidental swallowing.

The study began in Chicago with a mix of 131 vinyl products (also known as PVC or polyvinyl chloride). The items were bought at national chain stores and included vinyl clothing, backpacks, cables, toys, and household items. Lead testing by an independent

laboratory showed that about one-fifth of the products (28) contained from 100 to over 22,000 parts-per-million (ppm) lead.

Lead and cadmium in vinyl consumer products

Item	Store	Lead (ppm)	Cadmium (ppm)
backpack; Minnie's Spring Fever	Disney	163	224.5
backpack; 101 Dalmations	Kmart	104	321.0
backpack; Barbie	Kmart	372	75.7
barbell; 2lb vinyl cover	Target	7,050	12.4
breast milk cooler; Medela	Target	375	29.3
cable; Sega Controller	Toys R Us	4,100	17.4
cable; Gemini 3' video coaxial	Kmart	7,505	10.3
cable; Gemini mod. Phone cord	Kmart	865	15.3
cable; Philco in-ear headphones	Kmart	3,770	11.0
cable; Philco headphones	Kmart	3,490	52.4
cable; AT&T 25' phone line cord	Kmart	213	6.5
cable; Gemini computer printer	Kmart	5,765	18.1
key ring; Disney Minnie Totes	Target	1,430	6.1
pencil case; Fun d Mentals	Kmart	197	25.6
placemat; Warner Bros. Space J.	Kmart	178	7.9
placemat; Barth & Dreyfuss	Kmart	398	6.2
placemat; Gloria Vanderbilt	Wal-Mart	505	12.0
purse; Pacific Kids	Wal-Mart	349	104.8
rain hat; Warner Bros. Tweety	Warner Bros.	4,060	35.4
rain coat; Columbia Youth Parka	Uncle Dan's	22,550	47.9
shower curtain; Springs Bath	Wal-Mart	864	105.5
tent pole; Barbie Slumber	Toys R Us	6,105	14.6
totebag; Tweety	Wal-Mart	459	228.5
toy; Looney Tunes hackey sack	Toys R Us	1,610	nd
toy; Kentucky Fried Chicken	Toys R Us	207	344.0
toy; Toteables cosmetics pouch	Target	392	152.0
toy; Dimples doll stroller	Toys R Us	7,115	22.6
umbrella; Looney Tunes	Toys R Us	817	27.0

Abbreviations: ppm, parts-per-million by weight in product, nd, not determined. All products shown were purchased in Chicago, Illinois. A representative sample of these products contained lead in cities throughout the US. See Results and Discussion for data and information regarding other cities. The Consumer Product Safety Commission staff-recommended limit for lead in vinyl is 200 ppm. Cadmium is not regulated by the Consumer Product Safety Commission. Proposition 65 regulates cadmium at one-tenth the level set for lead. Results shown are averages of duplicates. The average coefficient of variation was 9% and 22% for lead and cadmium respectively.

Eighteen percent of the products exceeded the maximum lead limit in vinyl formally recommended by the staff of the Consumer Product Safety Commission for vinyl

miniblinds (200 ppm). More than half of these lead-containing vinyl products contained over 600 ppm lead and therefore would have been illegal and recalled if they had been made out of a regulated material like painted wood. In addition, all of the lead-containing products also contained varying levels of cadmium, a known carcinogen and kidney toxin which is not regulated in children's products.

The investigation widened to include California since it has the most stringent regulation of lead and cadmium in the US. A group of the items found to contain lead in Chicago was also purchased in Los Angeles and San Francisco and tested for both toxic metals. All seventeen products contained both lead and cadmium at levels previously seen in Chicago. Despite evidence that both metals are available for ingestion, none of the items carried labels warning California parents or children about the presence of either lead or cadmium as required by Proposition 65.

The study expanded to include 10 major US cities and Montreal. A representative sample of 19 lead-containing items from Chicago was sought in each locale. Not all of the products were found in every city, but all of the items which were purchased and tested contained lead. Seventeen of the 19 items in the sample exceeded the Consumer Product Safety Commission staff-recommended limit in the nationwide survey. The results indicate that vinyl products containing lead are readily available throughout the US and in at least one major Canadian city.

In order to determine whether the vinyl items might release toxic lead or cadmium during foreseeable use over the lifetime of these products, Greenpeace tested the possible availability of the metals. Two studies were conducted by independent laboratories using Consumer Product Safety Commission protocols.

Three commonly chewed products were examined to determine if they might release lead to a child's body. The data demonstrated that swallowing less than one-hundredth of an ounce of either a Gemini phone cord, Disney key ring, or Kentucky Fried Chicken toy would exceed the exposure limit used by the Consumer Product Safety Commission. Even swallowing less than one-thousandth of an ounce would release more than four times the amount of lead legally permitted under California's Proposition 65. Most importantly, it may not be necessary to actually swallow an item to be poisoned by it since simply chewing and sucking on plastic cables is a known source of lead poisoning.

Seven products were tested to determine whether lead or cadmium could be released to the surface during aging. Lead- or cadmium-contaminated dust is especially hazardous since it can easily enter the body by licking, chewing, inhalation, and hand-to-mouth behavior. A Minnie Mouse backpack, 101 Dalmations backpack, Barbie backpack, Tweety totebag, Columbia rain coat, Barbie tent pole, and Warner Bros. rain hat all released lead during four weeks of laboratory aging. Cadmium was released by five of the six products that were examined. All levels of lead and cadmium on the product surfaces exceeded the Proposition 65 limits for both metals. Lead was also detected on the

surfaces of all seven products right out of the package. The likely use of several vinyl products by a child would further increase the amount of lead and cadmium exposure.

The health hazard posed by lead in vinyl products is not just theoretical. In 1996, unexplainable high blood lead levels in children from Arizona, North Carolina, and Virginia provoked an investigation by the Departments of Health. Unexpectedly, vinyl miniblinds were identified as the common source of lead dust. An investigation by the Consumer Product Safety Commission found that new vinyl miniblinds contained from 7,700 to 12,300 ppm lead. In contrast, painted toys are recalled by the agency if they contain greater than 600 ppm lead. The Consumer Product Safety Commission estimated that 25 million vinyl miniblinds containing lead are imported into the US each year. An experimental study conducted by the agency demonstrated that light degraded the vinyl blinds and caused the formation of toxic lead dust which was apparently ingested by the children.

The Consumer Product Safety Commission issued a warning to the public and also entered into a voluntary agreement with the Window Covering Safety Council, the trade association of vinyl blind manufacturers. The agency sought reductions in lead content in vinyl miniblinds to the lowest level possible. The agency staff recommended a limit of 200 ppm lead in vinyl due to the cumulative health hazards posed by lead poisoning. Retailers were instructed to place a warning label on brands known to contain lead. Several months later, Greenpeace found unlabeled, lead-containing miniblinds featured in K-Mart's 1996 Labor Day Sale. The blinds were deeply discounted and present in Kmart stores across the US.

In contrast to the federal advisory to retailers, the State of California filed suit against companies that made or sold lead-containing blinds. Twelve companies including Wal-Mart, Kmart, J. C. Penney, and Montgomery Ward & Co. were named as defendants for violating Proposition 65.

The initial discovery of lead in vinyl blinds was surprising to the public, yet the industry has been using lead stabilizers and pigments in vinyl since the 1950s. Vinyl contains chlorine and therefore requires the addition of metals that act as stabilizers to retard degradation.

Lead causes irreversible nervous system damage, decreased intelligence, behavioral abnormalities, and learning disabilities. Lead also interrupts normal kidney function and blood formation. Cadmium can cause kidney damage and lung cancer. Both metals are developmental and reproductive toxins and cause damage at extremely low doses. Children are especially susceptible to these metals and no children's product should contain them. Neither metal is regulated in vinyl consumer products.

Findings

- Lead and cadmium are widely present in vinyl consumer products including those designed for children.
- The amount of lead present in many vinyl consumer products exceeds current Consumer Product Safety Commission regulations as well as the formal staff-recommended limit for lead in vinyl.
- Lead and cadmium in vinyl consumer products represent a health hazard since both are inevitably released as toxic dust when the product deteriorates.
- The amount of lead and cadmium released by a representative sample of vinyl consumer products violates California's Safe Drinking Water and Toxic Enforcement Act of 1986.

Factors

- Lead is a neurotoxin. Cadmium is a renal toxin and carcinogen. Both metals are highly toxic and especially damaging to children.
- Lead and cadmium are widely present in vinyl consumer products as stabilizers, components of pigments, or as contaminants.
- Lead is not regulated in vinyl consumer products by the Consumer Product Safety Commission. An agency staff-recommended limit was not adopted.
- Cadmium is not regulated in consumer products by the Consumer Product Safety Commission.
- There are alternative materials for all consumer uses of vinyl.

Recommendations

1. Parents should not purchase vinyl or PVC products to which children might be exposed including but not limited to rain coats, umbrellas, clothing, backpacks, ponchos, school supplies, purses, sports equipment, and toys. Vinyl children's products should be returned to the manufacturer or retailer.

2. Retailers, distributors, and manufacturers should remove vinyl children's products containing lead and/or cadmium from the market.

3. The Consumer Product Safety Commission should prohibit the use of lead and cadmium in consumer products such as vinyl. Vinyl children's products containing lead or cadmium should be recalled by the agency. A recall level should be set low enough to prohibit the entry of lead- and/or cadmium-containing products like the vinyl backpacks shown in Table 1, into the market.

4. Vinyl products sold in California should be tested for lead and cadmium and labeled by retailers and manufacturers if they are found to contain either metal. The State of California should pursue legal action against manufacturers and retailers selling lead- and cadmium-containing consumer products that violate the Safe Drinking Water and Toxic Enforcement Act of 1986 (also known as Proposition 65).

5. Retailers and trademark licensers should not market vinyl products to which children might be exposed. This should be made explicit in future sales and licensing agreements.

6. The plastics industry and toy manufacturers, through trade associations such as the Society of Plastics Industry and the Toy Manufacturers of America, should adopt an industry-wide standard against manufacturing children's products with vinyl.

Introduction

In 1996, the unexplained lead poisoning of children in three states provoked an investigation that identified vinyl blinds as the common source of lead dust.[1] The blinds were not suspected to be a source of lead even though vinyl has contained lead stabilizers since the 1950s. Vinyl requires the addition of metal stabilizers because it contains chlorine (vinyl is also known as polyvinyl chloride or PVC). Without a stabilizer, the chlorine can degrade the product by forming hydrochloric acid. Lead effectively stabilizes bound chlorine and binds any free chlorine that might be formed during processing or degradation.[2] Lead is also used in various pigments that color plastic.

There are three important aspects of the lead poisoning associated with vinyl blinds. First, lead poisoning is one of the most serious preventable public health hazards in the US.[3] Lead decreases intelligence and damages the nervous system at extremely low doses.[4, 5] Its effects are cumulative and irreversible.[6] Second, lead has a long history of serving the obligatory role of stabilizer in vinyl products. Finally, lead is released from vinyl during product degradation. The Consumer Product Safety Commission experimentally demonstrated that light and heat can cause degradation of vinyl and liberation of lead dust.[7] Unfortunately for children, vinyl miniblinds release lead during normal product use.[8]

Given the serious health effects of lead and its inevitable release from vinyl blinds that contain it, Greenpeace asked whether other vinyl consumer products might also pose a lead hazard. Lead testing of vinyl consumer products began in Chicago, then widened to include 10 major US cities and Montreal, Canada. Preliminary studies of bioavailability were conducted to determine the consequence of accidental swallowing. An accelerated aging study was conducted to determine whether toxic dust might be formed during product degradation.

Materials and Methods

Please see Appendix V.

Results and Discussion

A random survey in Chicago reveals lead-containing vinyl products

An investigation of vinyl consumer products in Chicago showed that many of them contained lead (Table 1). Roughly 20% of the 131 products that were surveyed contained lead. Table 1 shows 28 lead-containing vinyl items that were purchased at national chain stores like Kmart, Toys R Us, Target, and Wal-Mart. The products include a variety of common school products, clothing, toys, and household items. Eighty-six percent of the lead-containing items in Table 1 exceeded the 200 ppm standard for lead in vinyl proposed by Consumer Product Safety Commission staff. In fact, more than half of the lead-containing items contained greater than 600 ppm lead and therefore would have been illegal and recalled if they had been painted wooden toys.

Children are the marketing target

The vinyl items containing lead included items with popular children's characters like Minnie Mouse, 101 Dalmations, Barbie, various Looney Tunes characters, and even Michael Jordan (Table 1). Other vinyl products containing lead included common household items used by children such as a cable for a Sega Genesis video game, various phone cords, a VCR cable, and headphone cables (Table 1). The lead-containing list even included vinyl placemats whose normal proximity to food is especially troubling. Various vinyl clothing items also contained lead. A Warner Bros. rain hat featuring Tweety contained over 4,000 ppm lead and a popular Columbia rain coat for children showed over 22,000 ppm lead (Table 1). Several toys easily available for sucking or even designed for it contained lead. A small hackey sack featuring Warner Bros. Looney Tunes characters contained over 1,600 ppm lead. A Kentucky Fried Chicken toy destined for children's mouths contained roughly 200 ppm lead. A simple vinyl pouch containing cosmetics had nearly 400 ppm lead and a doll stroller for little girls showed over 7,000 ppm lead.

Vinyl products also contain cadmium

All of the vinyl products containing lead also contained cadmium. In some cases the levels of cadmium were even higher than the lead levels. Since cadmium is even more toxic than lead, the results are especially surprising. Especially alarming is the presence of cadmium in a Kentucky Fried Chicken toy designed to put into children's mouths. Each gram of this toy contained over 340 μg cadmium (μg is a microgram or one-millionth of a gram). Both the Minnie Mouse and 101 Dalmations backpacks also contained unexpected levels of the carcinogenic metal. Each gram of the two backpacks contained 225 and 321 μg of cadmium respectively. The total amount of cadmium in the product adds up rapidly. For example, the 101 Dalmations backpack weighs roughly 300 g and therefore contains about 96,300 μg cadmium. To place the results in perspective, the limit for exposure to inhaled cadmium dust in California is 0.05 μg per day. This represents 1.8 billionths of an

ounce per day. The 101 Dalmations backpack contained nearly 2 million times this level of cadmium.

Table 1. Lead and cadmium in vinyl consumer products purchased in Chicago.

Item	Store	Lead (ppm)	Cadmium (ppm)
backpack; Minnie's Spring Fever	Disney	163	224.5
backpack; 101 Dalmations	Kmart	104	321.0
backpack; Barbie	Kmart	372	75.7
barbell; 2lb vinyl cover	Target	7,050	12.4
breast milk cooler; Medela	Target	375	29.3
cable; Sega Controller	Toys R Us	4,100	17.4
cable; Gemini 3' video coaxial	Kmart	7,505	10.3
cable; Gemini mod. Phone cord	Kmart	865	15.3
cable; Philco in-ear headphones	Kmart	3,770	11.0
cable; Philco headphones	Kmart	3,490	52.4
cable; AT&T 25' phone line cord	Kmart	213	6.5
cable; Gemini computer printer	Kmart	5,765	18.1
key ring; Disney Minnie Totes	Target	1,430	6.1
pencil case; Fun d Mentals	Kmart	197	25.6
placemat; Warner Bros. Space J.	Kmart	178	7.9
placemat; Barth & Dreyfuss	Kmart	398	6.2
placemat; Gloria Vanderbilt	Wal-Mart	505	12.0
purse; Pacific Kids	Wal-Mart	349	104.8
rain hat; Warner Bros. Tweety	Warner Bros.	4,060	35.4
rain coat; Columbia Youth Parka	Uncle Dan's	22,550	47.9
shower curtain; Springs Bath	Wal-Mart	864	105.5
tent pole; Barbie Slumber	Toys R Us	6,105	14.6
totebag; Tweety	Wal-Mart	459	228.5
toy; Looney Tunes hackey sack	Toys R Us	1,610	nd
toy; Kentucky Fried Chicken	Toys R Us	207	344.0
toy; Toteables cosmetics pouch	Target	392	152.0
toy; Dimples doll stroller	Toys R Us	7,115	22.6
umbrella; Looney Tunes	Toys R Us	817	27.0

Abbreviations: ppm, parts-per-million by weight in product; nd, not determined. Results shown are averages of duplicates. The average coefficient of variation was 9% and 22% for lead and cadmium respectively. The Consumer Product Safety Commission staff-recommended limit for lead in vinyl is 200 ppm. Cadmium is not regulated by the Consumer Product Safety Commission. Proposition 65 regulates cadmium at one-tenth the level set for lead.

Vinyl products in California contain lead and cadmium

Since lead and cadmium are stringently regulated in the state of California, a representative sample of the Chicago vinyl products was purchased in San Francisco and

Los Angeles and tested for the presence of lead and cadmium. Table 2 shows that the same pattern of lead and cadmium content that was observed in products from Chicago was seen in those from California. Seventeen different vinyl products contained between 180 and 7,780 ppm lead. More than 80% of the products violated the Consumer Product Safety Commission staff-recommended limit of 200 ppm lead in vinyl.

Though cadmium is more stringently regulated than lead in California, its presence was ubiquitous in items containing lead. In a few cases, the levels of cadmium rivaled or even exceeded the lead levels. The Disney Minnie Mouse backpack, Kmart 101 Dalmations backpack, Barbie backpack, shower curtain, and Kentucky Fried Chicken toy contained extremely high amounts of cadmium (Table 2). Each gram of the items contained between 235 and 650 μg cadmium even though they were purchased in a state that only permits 0.05 μg exposure to cadmium dust per day.

Agency	Lead	Cadmium
Consumer Product Safety Commission	paint: 600 ppm vinyl: 200 ppm*	not regulated
State of California	0.5 μg/day	0.05 μg/day inhalation

Abbreviations: ppm, parts-per-million by weight in product. *Proposed by Consumer Product Safety Commission staff following discovery of lead in vinyl blinds.

Disney, Warner Bros. and Mattel products contain toxic metals

Ironically, many of the California products containing lead and cadmium also featured children's icons of several California-based companies including Disney, Mattel, and Warner Bros. Backpacks featuring Minnie Mouse and 101 Dalmations containing both lead and high amounts of cadmium were available at the Disney Store and Kmart in Los Angeles and San Francisco respectively. Mickey Mouse is prominently featured on a children's key ring containing over 240 ppm lead. Mattel's Barbie is featured on a backpack containing over 400 ppm lead and 200 ppm cadmium. Barbie is also featured in a children's slumber tent that contains tent poles with 4,685 ppm lead in Los Angeles and 7,400 ppm lead in a tent purchased in San Francisco. Warner Bros. was also represented among lead-containing vinyl products for children. A rain hat containing over 2,700 ppm lead and featuring Tweety was purchased in the Warner Bros. store in both California cities. A Looney Tunes hackey sack contained either over 300 ppm lead in Los Angeles or over 1,600 ppm lead in San Francisco. A Space Jam placemat featuring Michael Jordan contained 180 ppm lead. Finally, a Looney Tunes children's umbrella available in Los Angeles and San Francisco contained over 650 ppm lead and 50 ppm cadmium in both cities. None of the products contained labels warning California consumers and children about the presence of either lead or cadmium.

Table 2. Lead and cadmium in vinyl consumer products purchased in California

Item	Store	Lead (ppm)	Cadmium (ppm)
backpack; Minnie's Spring Fever	Disney; LA	263	242.0
backpack, Minnie's Spring Fever	Disney; SF	255	238.5
backpack; 101 Dalmations	Kmart; LA	226	499.0
backpack; 101 Dalmations	Kmart; SF	288	440.0
backpack; Barbie	Kmart; SF	417	242.0
barbell; 2lb vinyl cover	Target; SF	7,780	7.2
cable; Sega Controller	Toys R Us; SF	4,755	14.1
cable; Gemini 3' video coaxial	Kmart; LA	4,910	9.6
cable; Gemini 3' video coaxial	Kmart; SF	6,965	24.3
cable; Gemini mod. phone cord	Kmart; LA	679	6.8
cable; Gemini mod. phone cord	Kmart; SF	5,290	36.7
cable; Philco in-ear headphones	Kmart; LA	5,910	5.6
cable; Philco in-ear headphones	Kmart; SF	4,700	27.3
key ring; Disney Minnie Totes	Target; SF	242	17.0
placemat; Warner Bros. Space J.	Kmart; LA	180	6.9
rain hat; Tweety	Warner Bros.; LA	2,755	39.0
rain hat; Tweety	Warner Bros.; SF	2,835	51.5
shower curtain; Springs Bath	Wal-Mart; LA	477	425.0
tent pole; Barbie Slumber	Toys R Us; LA	4,685	11.8
tent pole; Barbie Slumber	Toys R Us; SF	7,400	8.3
toy; Looney Tunes hackey sack	Toys R Us; LA	316	45.1
toy; Looney Tunes hackey sack	Toys R Us; SF	1,675	53.9
toy; Kentucky Fried Chicken	Toys R Us; LA	125	649.0
toy; Kentucky Fried Chicken	Toys R Us; SF	110	363.5
umbrella; Looney Tunes	Toys R Us; LA	656	46.7
umbrella; Looney Tunes	Toys R Us; SF	695	50.3

Abbreviations: ppm, parts-per-million by weight in product. LA, Los Angeles, CA; SF, San Francisco, CA. Results shown are averages of duplicates. Average coefficient of variation was 14% and 16% for lead and cadmium respectively. The Consumer Product Safety Commission staff-recommended limit for lead in vinyl is 200 ppm. Cadmium is not regulated by the Consumer Product Safety Commission. Proposition 65 regulates cadmium at one-tenth the level set for lead.

Pilot study in Canada confirms the US pattern

A small pilot study to determine whether vinyl products in Canada might also contain lead and cadmium was conducted by examining thirteen items purchased in Montreal, Quebec. Eight of the thirteen items contained significant amounts of lead (Table 3). The products included popular children's characters like 101 Dalmations, Barbie, and Minnie Mouse. Table 3 shows that half of the items violated the 600 ppm

standard currently in use in Canada. Health Canada is proposing a 15 ppm limit for lead in children's and other consumer products.[9] The 101 Dalmations and Barbie backpacks, Sega Genesis cable, and Columbia rain coat all contained lead as discovered in various US cities (Tables 3 and 4) The items also contain cadmium as discovered in the US sampling. A Minnie Mouse poncho, 101 Dalmations backpack, and waterproof jacket all contained greater than 45 ppm cadmium. As in the US, all the lead- and cadmium-containing items were readily available at common chain stores.

Table 3. Lead and cadmium in vinyl products purchased in Montreal

Item	Store	Lead (ppm)	Cadmium (ppm)
backpack; 101 Dalmations	Toys R Us	110	55.0
backpack; Barbie	Toys R Us	610	4.0
barbell; 2lb vinyl cover	Sports Experts	247	29.0
cable; Sega Controller	Toys R Us	5,415	6.0
jacket; black waterproof	Zellers	234	143.0
poncho; Minnie Mouse Suite 100	Wal-Mart	99	46.0
rain coat; Columbia	Sports Experts	18,600	8.0
rain pants; Columbia	Sports Experts	18,750	18.0

Abbreviations: ppm, parts-per-million by weight in product. Results shown are averages of duplicates. Average coefficient of variation was 14% and 15% for lead and cadmium respectively. The limit for lead in consumer products currently used in Canada is 600 ppm. Health Canada is currently proposing a 15 ppm limit for lead in children's and consumer products.

A national problem in the US

To determine whether lead in vinyl consumer products represented a national problem, a representative sample of the Chicago items was purchased in a variety of US cities and analyzed for lead. The US cities were: Boston, MA, Boulder, CO, Chicago, IL, Los Angeles, CA, Minneapolis, MN, New Orleans, LA, New York, NY, Portland OR, San Francisco, CA, Seattle, WA, and Washington, D.C. The results indicate that vinyl consumer products containing hazardous lead levels are widely available throughout the United States and in at least one major Canadian city (Table 4). Not all of the items were available in every city, but Table 4 shows that items originally found to contain lead in Chicago also contained lead in every city examined. Seventeen of the 19 items averaged greater than the 200 ppm standard recommended by the Consumer Product Safety Commission staff. The results also show a large variation in lead content throughout the US and even in different cities within the same state. This is surprising considering that almost all of the products were purchased at national chain stores like Kmart, Wal-Mart, Target, and Toys R Us. The data suggests that a low lead or cadmium level in one city may not guarantee the safety of the same product purchased in a different store, city, state, or country.

Table 4. Lead in vinyl products purchased in various cities

Item	Average Lead (ppm)	Low (ppm)	High (ppm)	Total Tests (n)	Cev (%)	Cities Tested
backpack; Minnie's	286	132	637	9	49	Bos, Chi, LA, Min, SF
backpack; 101 Dalm.	198	97	374	19	44	Bos, Bou, Chi, LA, Mon, Min, NO, SF, DC
backpack; Barbie	417	236	627	8	33	Chi, SF, Mon, DC
barbell; 2lb vinyl	5,271	824	8,610	6	67	Chi, SF, Sea
cable; Sega Genesis	5,045	2,490	7,160	15	21	Bos, Chi, Mon, NO, NY, SF, DC
cable; Gemini video	6,803	4,250	12,600	17	30	Bos, Chi, LA, NO, NY, SF, Sea, DC
cable; Gemini phone	2,130	393	5,350	13	91	Chi, LA, NO, NY, SF, DC
cable; Philco in-ear	4,845	3,610	6,150	13	18	Bos, Chi, LA, NO, SF, Sea, DC
cable; AT&T phone	4,203	207	7,260	6	76	Bos, Chi, Sea
key ring; Disney	836	234	1,570	4	83	Chi, SF
placemat; Space Jam	244	126	426	14	41	Bou, Chi, LA, Min, Sea, DC
rain hat; Tweety	2,631	680	4,200	8	49	Bos, Chi, LA, SF
rain coat; Columbia	23,211	16,800	28,600	9	15	Bos, Chi, LA, Mon, Por, SF
shower curtain; Spr.	665	445	889	12	27	Bos, Bou, Chi, LA, Min, NO, Sea, DC
tent pole; Barbie	5,962	2,830	12,500	17	41	Bos, Bou, Chi, LA, NO, SF, Sea, DC
totebag; Tweety	679	409	1,093	4	45	Bos, Chi
toy; L. Tunes hackey	1,774	190	7,490	8	136	Bou, Chi, NO, SF, DC
toy; KFC	177	97	420	12	50	Bos Chi, LA, Sea, SF, DC
umbrella; L. Tunes	661	416	852	14	19	Bos, Chi, LA, NO, NY, SF, Sea, DC

One item from each city was tested. Abbreviations as above plus: Cev, coefficient of variation, the standard deviation expressed as a percent of the mean; Bos, Boston, MA; Bou, Boulder, CO; Chi, Chicago, IL; LA, Los Angeles, CA; Mon; Montreal, Quebec, Canada; Min, Minneapolis, MN; NO, New Orleans, LA; NY, New York, NY; Por, Portland, OR; SF, San Francisco, CA; Sea, Seattle, WA; DC, Washington, D.C.

Lead and cadmium as ingredients in vinyl

The overall variation in lead and cadmium levels seen in the various consumer products could occur for various reasons. Since metal stabilizers are typically added at levels greater than 5,000 ppm, vinyl items containing high amounts of lead are probably stabilized with it. Items with lower amounts of lead may reflect contamination in the manufacturing process. In addition, cadmium and lead are both used in pigments that color vinyl. Items colored with these pigments could also contain high amounts of either metal. Unfortunately, vinyl always requires some sort of metal stabilizer to protect the plastic during processing due to the presence of chlorine in the polymer (see Appendix IV). This structural obligation for metal stabilizers makes the prospect of making a "clean" vinyl product doubtful.

Absorbing lead from chewing and swallowing vinyl

The chewing and swallowing behavior of children is a common source of lead exposure. However, swallowing is not even necessary for exposure. Simply chewing and sucking on plastic cables is a known source of lead poisoning.[10] Three commonly chewed products were incubated under mild acid conditions at body temperature using Consumer Product Safety Commission protocols. The experiment explored whether ingesting even a tiny amount of a Gemini phone cord, Disney Minnie Totes key ring, or Kentucky Fried Chicken toy could pose a risk.

The results show that swallowing less than one-hundredth of an ounce of vinyl would release measurable quantities of lead. In fact, Table 5 shows that all three items exceeded the ingestion limit used by the Consumer Product Safety Commission (15 µg/day). The agency limit is 30 times higher than the maximum ingestion level permitted in California under Proposition 65. The Commission uses it despite acknowledging that "..any ingestion of lead is undesirable because the effects of lead ingestion are cumulative, and other sources of lead may be available to children...."[11]

Table 5. Migration of lead under conditions that mimic swallowing 250 mg of an item (0.00881 ounces)

Item	Store	Available Lead (µg)
cable; Gemini modular phone cord	Kmart	21.6
key ring; Disney Minnie Totes	Target	34.2
toy; Kentucky Fried Chicken	Toys R Us	23.0

Abbreviations: µg, micrograms. All products were purchased in Chicago, Illinois. Items treated using Consumer Product Safety Commission protocols for extractable lead as described in Appendix V. The permitted exposure level for lead under California's Proposition 65 is 0.5 µg/day.

The data also demonstrates that swallowing less than one-thousandth of an ounce of any product in Table 5 would release enough lead to exceed the legal exposure limit of Proposition 65 by more than four-fold. The lead migration displayed by the vinyl Kentucky Fried Chicken is especially disturbing since the toy is actually designed to be put into children's mouths.

Contaminated dust is a health risk

Lead- or cadmium-contaminated dust is especially hazardous since it can easily enter the body in multiple ways. Routes of ingestion include licking, sucking, mouthing, inhalation, and hand-to-mouth behavior. Since real life behaviors encompass multiple exposure pathways, the data showing the presence of surface lead and cadmium was not confined to a specific route of ingestion. Instead, the results show the total amount of lead or cadmium that is available for ingestion on the product surface. Metals which are not ingested immediately can become part of the indoor environment and be ingested later by the same variety of pathways.

Lead dust is present on new vinyl products

Table 6 shows that lead dust was measured on the surfaces of seven products right out of the package. Lead levels on six products exceeded the Proposition 65 limit by 4 to 470 fold. Five of these products were purchased in California. More than half of the items contained lead dust at levels similar to, or greater than the Consumer Product Safety Commission ingestion limit.

Table 6. Lead and cadmium dust present on new vinyl children's products: exposures provided by average products

Item	Store	Lead (μg)	Cadmium (μg)
backpack; Minnie's Spring Fever	Disney; LA	1.984	nd
backpack; 101 Dalmations	Kmart; LA	19.430	1.740
backpack; Barbie	Kmart; SF	14.210	1.160
rain coat; Columbia	Columbia; Por	235.733	nd
rain hat; Tweety	Warner Bros.; LA	9.217	nd
tent pole; Barbie Slumber	Toys R Us; SF	13.404	nd
totebag; Tweety	Wal-Mart; Chi	0.240	nd

Abbreviations: μg, micrograms, nd, not detected. LA, Los Angeles, CA; SF, San Francisco, CA; Por, Portland, OR; Chi, Chicago, IL. Average exposures represent the mean of triplicates. The permitted exposure level for lead and cadmium under California's Proposition 65 is 0.5 μg/day and 0.05 μg/day respectively. Areas of products are as follows: Minnie's Spring Fever: 96 in^2; 101 Dalmations: 290 in^2; Barbie: 290 in^2; Columbia: 1,700 in^2; Tweety rain hat: 79 in^2; Barbie Slumber: 36 in^2; and Tweety totebag: 240 in^2

Two children's products were made even more dangerous by the additional presence of cadmium. Table 6 shows more than 1.1 μg of cadmium present on the surfaces of new 101 Dalmations and Barbie backpacks in addition to greater than 14 μg of lead dust. The cadmium levels are more than 23 to 35 times higher than the amount permitted in California, where they were purchased.

Vinyl products release toxic metals during degradation

Lead and cadmium were released as toxic dust during the degradation of vinyl children's products. Figure 1 shows the rapid release of lead from a Barbie tent pole under conditions used by the Consumer Product Safety Commission to demonstrate the liberation of lead from vinyl miniblinds. Figure 2 shows the increasing availability of cadmium on the surface of a 101 Dalmations backpack during aging. To our knowledge, Figure 2 represents the first demonstration of cadmium release from a vinyl consumer product during degradation.

Figure 1. Lead release during degradation of a Barbie tent pole

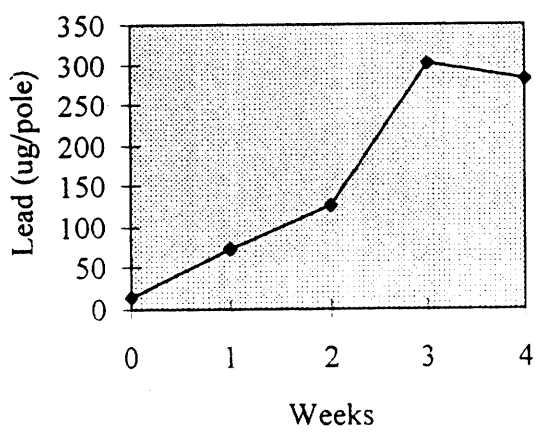
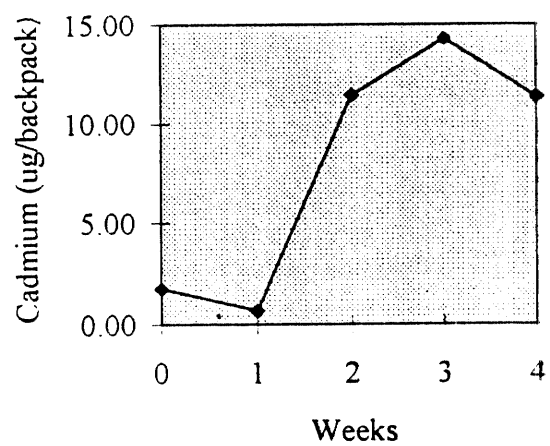


Figure 2. Cadmium release during degradation of a 101 Dalmations backpack



Abbreviations μg, micrograms. Average exposures representing the mean of triplicates were plotted for both figures. The permitted exposure level for lead and cadmium under California's Proposition 65 is 0.5 μg/day and 0.05 μg/day respectively.

Large amounts of lead and cadmium are released

Large amounts of lead-contaminated dust were released by vinyl children's products during four weeks of accelerated aging. Table 7 shows maximum surface lead levels between 5.6 μg and 336 μg. The lowest level occurred on the Disney Minnie's

Spring Fever backpack. However, this backpack still produced more than 11 times the amount of lead legally permitted in California where it was purchased. The two highest amounts of lead dust were present of the surfaces of the Columbia rain coat and the Barbie Slumber tent pole. Both products released over 300 μg of lead dust; more than 600 times the Proposition 65 limit. In fact, all seven products violated the legal limit set by Proposition 65. Four of the products exceeded the limit used by the Consumer Product Safety Commission.

Table 7. Maximum lead and cadmium dust levels observed on the surfaces of vinyl products: exposures provided by average products

Item	Store	Lead (μg)	Cadmium (μg)
backpack; Minnie's Spring Fever	Disney; LA	5.664	5.792
backpack; 101 Dalmations	Kmart; LA	19.430	14.307
backpack; Barbie	Kmart; SF	35.380	23.393
rain coat; Columbia	Columbia; Por	336.033	nd
rain hat; Tweety	Warner Bros.; LA	9.217	0.263
tent pole; Barbie Slumber	Toys R Us; SF	302.484	ND
totebag; Tweety	Wal-Mart; Chi	8.800	15.520

Abbreviations: μg, micrograms; nd, not detected; ND, not determined. LA, Los Angeles, CA; SF, San Francisco, CA; Por, Portland, OR; Chi, Chicago, IL. Average exposures represent the mean of triplicates. The permitted exposure level for lead and cadmium under California's Proposition 65 is 0.5 μg/day and 0.05 μg/day respectively. See Table 6 for product areas.

Large amounts of easily ingestable cadmium dust were also released during degradation of vinyl children's products. Table 7 shows that the amount of cadmium released by all six products that were tested was 5 to 460 times higher than the Proposition 65 limit.

The use or presence of several vinyl products would further increase the amount of lead and cadmium available for ingestion. Table 7 shows that combining a Barbie backpack, Columbia rain coat, and Tweety rain hat, could quickly increase exposure to both toxic metals, especially if they were used multiple times each week or month. The hazard comes from the cumulative nature of lead poisoning.[12]

The most dangerous 10% of items would provide significant exposures

A statistical calculation revealed the amount of lead or cadmium that the most hazardous subset of product samples could provide. The estimate assumed a normal distribution and used the variation observed among replicates to calculate the 90th percentile level of either metal. The most dangerous 10% of samples could provide at least this amount of lead or cadmium.

Table 8. Minimum lead and cadmium exposures provided by the most hazardous 10% of items: statistical estimate

Item	Store	Lead (μg)	Cadmium (μg)
backpack; Minnie's Spring Fever	Disney; LA	14.824	6.678
backpack; 101 Dalmations	Kmart; LA	24.284	15.852
backpack; Barbie	Kmart; SF	83.692	35.840
rain coat; Columbia	Columbia; Por	590.554	nd
rain hat; Tweety	Warner Bros.; LA	12.415	0.497
tent pole; Barbie Slumber	Toys R Us; SF	486.925	ND
totebag; Tweety	Wal-Mart; Chi	20.495	16.762

Abbreviations: μg, micrograms; nd, not detected; ND, not determined. LA, Los Angeles, CA; SF, San Francisco, CA; Por, Portland, OR; Chi, Chicago, IL. The 90th percentile was calculated by adding ($z_{1.28}$ x sample standard deviation) to the mean. A normal distribution was assumed for the data set. The permitted exposure level for lead and cadmium under California's Proposition 65 is 0.5 μg/day and 0.05 μg/day respectively. See Table 6 for product areas.

Table 8 shows that the most hazardous samples of each product would provide exceptional exposures to both toxic metals. The only product which did not liberate cadmium was the Columbia rain coat. The most dangerous 10% of these rain coats would provide nearly 1,200 times the legal limit for lead set by Proposition 65 and 40 times the limit used by the Consumer Product Safety Commission. Cadmium was not measured on the Barbie tent pole, but this product could liberate 970 times the amount of lead permitted by Proposition 65.

The remaining 5 products contained both toxic metals on their surfaces. Lead levels ranged from 25 to 170 times higher than the Proposition 65 limit. Cadmium levels exceeded the limit by 10 to 335 fold. The presence of both lead and cadmium on the product surface represents a significantly greater hazard than the considerable danger posed by either toxic metal alone.

Appendix I. Lead and Cadmium Toxicity, Exposure, and Regulation

Lead poisoning is a significant health problem

Lead poisoning is widely recognized to be one of the most serious preventable public health hazards in the US.[13] In the period from 1991 to 1994, 890,000 children had blood lead levels defined as lead-poisoned.[14] In addition, a national phone survey in 1994 showed that only 24% of parents had screened their young children for lead, indicating that the true magnitude of the problem may be much larger.[15]

Lead causes irreversible nervous system damage and decreased intelligence at extremely low doses.[16, 17] Children are especially susceptible to lead poisoning because they absorb and retain more lead in proportion to their weight than adults.[18] Lead exposure in childhood has been associated with lower vocabulary and grammatical-reasoning scores, increased absenteeism, poorer eye-to-hand coordination, and lower class standing in high school.[19] Unfortunately, most children with lead poisoning are not overtly symptomatic.[20]

Defining the hazard

What is considered a "safe" level of lead by the medical community has declined significantly over the last thirty years. In the 1960s the Centers for Disease Control (CDC) action level for lead in blood was 60 μg/dl. This lead level results in mental retardation, kidney damage, male infertility, impaired growth, and hearing loss.[21] A decade later the medical community lowered the level to 30 μg/dl. In 1985, the action level dropped to 25 μg/dl and in 1991 it was modified downward to 10 μg/dl.

Current blood level standards for lead do not protect children. Even though 10 μg/dl is considered "safe" by some medical professionals, reduced birthweight, hearing loss, and attention deficit in children and monkeys have been observed at blood lead levels of 5 μg/dl.[22] In fact, recent analyses of lead exposure and children's IQ could find no evidence of a threshold, suggesting that there is no safe lead level and that the current 10 μg/dl standard permits nervous system damage.[23] Blood lead levels in preindustrial humans were estimated from bone measurements to be 0.016 μg/dl.[24] The current "safe" level of 10 μg/dl is 600 times higher than natural concentration of lead in blood.

Exposure to lead

One of the largest sources of lead exposure in the US is lead-contaminated dust. A common source is decaying paint that contaminates house dust and soil.[25] The permitted lead level in residential paint in the US was lowered to 600 ppm in 1978 but a huge reservoir of leaded paint in older housing stock continues to pose a hazard. Recalls are normally implemented when painted consumer products contain greater than 600 ppm lead. However, each product or source of lead is regulated separately. This allows for

exposures from a variety of sources, all of which might be below the particular regulatory level.[26] The health hazard comes from the cumulative nature of lead poisoning.[27]

Lead ingestion and poisoning typically occurs by licking, mouthing, or swallowing the item, through hand-to-mouth activity, or inhalation. Both hand-to-mouth activity and mouthing were indicated in the lead poisoning cases caused by vinyl blinds.[28] Inhalation and hand-to-mouth activity have been implicated in the lead poisoning of plastics workers.[29, 30, 31] Finally, chewing on plastic-covered electrical cables was also identified as a source of lead intoxication.[32]

California and lead regulation

The most stringent regulation of lead in consumer products is in the state of California. The Safe Drinking Water and Toxic Enforcement Act of 1986 (also known as Proposition 65) regulates chemicals known to cause cancer or reproductive toxicity. Lead qualifies under both categories. The law regulates chemicals by requiring clear warning labels on products containing hazardous amounts of toxins and by prohibiting businesses from dumping listed chemicals onto the land or into water. The maximum acceptable intake level of lead under Proposition 65 as a reproductive and developmental toxin is 0.5 μg/day. This represents 0.8 millionths of an ounce of the children's Columbia rain coat in Table 1. The California lead level was adopted in 1987. It was determined by dividing the Occupational Safety and Health Administration (OSHA) limit of 500 μg/day by a 1,000-fold safety factor as required under the law.[33]

California and cadmium regulation

The State of California also recognized cadmium as a carcinogen under Proposition 65 in 1987.[34] The no-significant risk level for inhaled cadmium dust was set at 0.05 μg/day, one-tenth the level set for lead. The standard was based upon studies demonstrating an association between cadmium and respiratory cancer in both humans and laboratory animals.[35] Injection of cadmium in animals produced tumors both at the site of injection and at other sites including testicular interstitial cells and pancreatic islet cells.[36]

Cadmium was also recently nominated for listing as a reproductive toxin under Proposition 65.[37] Cadmium was positively correlated with reduced birthweight, premature birth, stillbirth, spontaneous abortion, and birth defects in humans.[38] In addition, cadmium and lead were both correlated with behavioral problems and learning disabilities. In utero exposure to both lead and cadmium was correlated with motor and perceptual problems in six year old children.[39] Since cadmium can be absorbed orally, by inhalation, or even by dermal contact, the lack of regulation in children's products by the Consumer Product Safety Commission is surprising.

Incineration and exposure to lead and cadmium

Incineration also represents a potentially significant source of lead and cadmium exposure. The ash produced by municipal waste incinerators contains both toxic metals.[40] In addition, both metals can be inhaled from combustion gases or ingested since they are easily leached out of ash into soil or water.[41] The EPA estimated that plastic contributes 71% of the lead and 88% of the cadmium in the combustible portion of the municipal waste stream.[42] This combustible portion of municipal waste is the major contributor to toxic air emissions and ash.[43] Both metals have been used as stabilizers and pigments in vinyl production for decades.

The final destination for lead- and cadmium-containing consumer products is the trash. This provides another legal avenue for exposure to both toxic metals. Currently, existing municipal waste combustors that process 1,000 tons per day are permitted to release 18 g/million dry standard cubic feet of cadmium and 200 g/million dry standard cubic feet of lead. A typical incinerator releases 3,670 dry standard cubic meters at 7% oxygen per ton of unprocessed municipal solid waste.[44] Therefore, an existing municipal waste incinerator of this size can legally emit 2,334 g of cadmium and 25,921 g of lead each day. For new incinerators the regulations are more "stringent". A 1,000 ton per day municipal waste incinerator can release 1,127 g of cadmium and 11, 274 g of lead per day. Assuming an 8,000 hour operating year, an existing 1,000 ton per day incinerator can legally emit 1,713 pounds of cadmium and 19,032 pounds of lead per year. A new incinerator of this size is permitted to emit 826 pounds of cadmium and 8,278 pounds of lead annually.

Prevention is the key

Lead poisoning is both extremely serious and preventable. The American Academy of Pediatrics has stated that, "Identification and treatment of the child poisoned with lead continues to be essential, but of greater importance is *identification of the source* and *prevention of subsequent exposures* for that child and other children in the future."[45] One overlooked source of lead (and cadmium) is vinyl consumer products. Fortunately, there are alternative materials for all consumer uses of vinyl. Parents can prevent toxic metal exposure to their children by returning vinyl items and buying products made of alternative materials.

Appendix II. Why Greenpeace Decided to Investigate Lead in Vinyl Products

Vinyl blinds as a source of lead

In 1996, the Arizona, North Carolina, and Virginia Departments of Health discovered unexplainable hazardous lead levels in children. The results were puzzling due to the lack of lead-based paint as an explanation for the poisoning. In the original Arizona case, a 1 ½ year old lead-poisoned child lived in a trailer that did not even have interior paint.[46] The investigation unexpectedly yielded vinyl miniblinds as the common source of lead dust. The mere presence of lead in vinyl blinds was surprising to the public even though it has been widely used as an additive in vinyl to retard decomposition since the 1950s.

An investigation by the Consumer Product Safety Commission revealed that ultraviolet (UV) light-degradation of the blinds caused the release of lead dust which was apparently subsequently ingested by children.[47] The agency found that new vinyl miniblinds contained from 7,700 to 12,300 parts-per-million (ppm) total lead.[48] In contrast, the limit for lead in painted toys and consumer products has been 600 ppm since 1978.[49]

Deterioration and toxic dust

The Consumer Product Safety Commission conducted accelerated aging experiments to demonstrate whether the lead in vinyl blinds could become available as dust as the blinds deteriorated in the sun. Blinds were exposed to UV light and heat over a period of eight weeks. During exposure, increasing levels of surface dust containing lead were measured on the blinds.[50] The results indicated that normal product use could make lead dust available to children. The deterioration of the vinyl and subsequent availability of lead continued despite washing or cleaning indicating that consumers were vulnerable despite good housekeeping practices.[51]

The vinyl blinds produced a surprising amount of toxic lead dust. The Environmental Protection Agency (EPA) standard limits for lead dust used in risk assessment of homes are 500 $\mu g/ft^2$ for window sills and 100 $\mu g/ft^2$ for floors.[52] In sharp contrast, lead levels over 14,000 $\mu g/ft^2$ were found on some blinds.[53] In Arizona, lead levels in all ten samples exceeded 100 $\mu g/ft^2$.[54] Seven of the ten samples had levels greater than 500 $\mu g/ft^2$.[55] In North Carolina, lead levels exceeded 100 $\mu g/ft^2$ in 45 of the 56 samples.[56] About half of the samples contained greater than 500 $\mu g/ft^2$ lead.[57] The vinyl blind sample with the highest level contained 66,440 $\mu g/ft^2$ lead dust.[58]

Vinyl blinds as a health risk

Lead-containing vinyl blinds represent a potentially significant health risk. Blood levels of lead greater than the 10 $\mu g/dl$ action level triggered the investigations in which

blinds were identified as the source of poisoning. Elevated blood lead levels in exposed children ranged from 17 to 58 μg/dl. A total of 31 lead-poisoned children in three states were investigated in the Consumer Product Safety Commission study. However, the agency estimated that 25 million vinyl miniblinds containing lead are imported into the US each year.[59] Taken together, the data suggests that lead poisoning due to vinyl blinds may be a significant preventable health problem in the US.

Publicity and response

Greenpeace and several other environmental organizations publicized the health risks associated with the vinyl blinds. Greenpeace wrote letters informing all the State Departments of Health in the US about the situation and contacted over 200 retailers. Local TV stations also picked up the story. The Chicago NBC affiliate, WMAQ, purchased ten different brands of vinyl blinds in Chicago and found lead levels between 940 ppm and 11,000 ppm.

Despite the publicity surrounding the health risk, the Consumer Product Safety Commission never issued a product recall of vinyl miniblinds. Instead, the agency entered into a dialog with the Window Covering Safety Council, the trade association of manufacturers. Initially, the industry group suggested that production be altered to meet the 600 ppm level specified for lead in paint under the Consumer Product Safety Act.[60] Despite the well-known toxicity of lead, the Window Covering Safety Council stated that "The industry is undertaking this program voluntarily understanding that there has been only cursory evaluation of the health risks of vinyl blinds to the public."[61]

A standard for lead in vinyl

In response to the trade association, the Consumer Product Safety Commission staff proposed a lower standard of 200 ppm lead in vinyl blinds and urged the industry to reduce lead content to the lowest level possible.[62] The trade association responded that, "The industry will work with the Consumer Product Safety Commission to develop an appropriate specification for vinyl window coverings through an accredited national standards organization, such as ANSI."[63] No specification currently exists.

In a follow-up memo the Consumer Product Safety Commission reiterated the position that lead levels below 200 ppm were technologically feasible.[64] In addition, the Consumer Product Safety Commission reminded the industry group that "...it is the responsibility of each manufacturer/importer to assure that the lead substitutes and other chemicals used in the manufacture of vinyl miniblinds do not present a hazard to consumers."[65]

The Consumer Product Safety Commission action and industry response were carried out quietly with no public attention. The agency never issued a mandatory standard for lead in vinyl blinds despite the 200 ppm level proposed by their own staff. Instead, the agency stated that "Because companies are discontinuing the use of lead, no

standard appears to be needed."[66] Unfortunately, the situation was different on the retail level.

The sale of a product containing lead is tolerated

The Consumer Product Safety Commission instructed retailers to place warning labels on brands known to contain lead but the recommendation was not enforced. Greenpeace found unlabeled, lead-containing miniblinds in Kmart stores across the US several months after the Consumer Product Safety Commission declaration. The lead-containing blinds were sold at deep discounts during the 1996 Labor Day Sale. It appears that vinyl blinds represent the first lead-containing consumer product whose sale was tolerated by governmental authorities, manufacturers, and retailers despite the demonstrated health risks to children.

Legal action in California

The State of California response to lead-containing vinyl blinds was more protective than the actions taken by the Consumer Product Safety Commission. Instead of issuing a recommendation, the state Attorney General, Dan Lungren, and Alameda County District Attorney, Tom Orloff, filed suit against 12 companies that made or sold the blinds since they failed to warn consumers as required by law.[67] The defendants included well-known national chains like Wal-Mart, Kmart, J.C. Penney, and Montgomery Ward & Co.

The problem remains unresolved

The lead content of all vinyl products remains unregulated even though lead poisoning causes irreversible nervous system damage to both children and adults. Cadmium has never been regulated by the Consumer Product Safety Commission despite its well-known carcinogenicity and kidney toxicity. Despite the clear health risk demonstrated by vinyl blinds, a Greenpeace follow-up evaluation revealed that no public agency appeared to be investigating other vinyl products to determine whether they might pose a similar health hazard as the miniblinds. Greenpeace initiated a study of lead in vinyl consumer products and found that vinyl blinds represent the tip of a much larger toxic iceberg.

Appendix III. Regulation of Lead in Vinyl

The adverse health effects of lead prompted a proposal for a specification limit in vinyl blinds. The Consumer Product Safety Commission staff recommended a lead limit of 200 ppm. The proposal was based on a maximum allowable ingestion limit of 15 μg/day lead, the surface area of half a hand of a typical child, and an assumption about vinyl degradation and subsequent availability of lead dust. The authors pointed out that "Staff believes that any ingestion of lead is undesirable because the effects of lead ingestion are cumulative and other sources of lead may be available to children, the staff urges the manufacturers to use the lowest amount of lead below 0.02% that is technologically feasible."[68]

The protection provided by the 200 ppm (0.02%) lead limit is questionable. To calculate the limit, the agency assumed a maximum allowable lead ingestion level of 15 μg/day. This is thirty times higher than the maximum level permitted in the state of California. In order to determine the likelihood of dust ingestion, the Consumer Product Safety Commission confined the exposure pathway to hand-to-mouth behavior. This presumes that the lead dust on blinds is somehow safe unless a child's hand touches them. Unfortunately, instead of disappearing, lead dust probably becomes part of the indoor environment. Failure to consider the fate of high levels of lead-contaminated dust inside a room underestimates the possible exposure and permits calculation of a more lenient specification limit.

The hand-to-mouth exposure scenario provided a method for calculating a maximum dust limit. The agency used anthropometric data to calculate the area of a typical child's hand between 2 and 6 years old. However, instead of using the area of both hands to determine possible dust exposure, the agency used half the area of only one hand. This means that a child might actually be exposed to four times the amount of lead dust the agency staff used in their calculation to determine the "safe" level. The staff then used this area to calculate the maximum dust limit that would maintain ingestion equal to or less than 15 μg/day. The agency proposed a lead dust limit of 3 $\mu g/in^2$, roughly equal to the EPA limit for windowsills of 3.472 $\mu g/in^2$ (500 $\mu g/ft^2$).

Finally, the Consumer Product Safety Commission used an association between total lead content of a vinyl blind and the amount of lead dust produced by a blind to arrive at a specification limit. Since the average total lead content was 6,900 ppm (0.69%) and the average lead dust level among the highest samples was 123 $\mu g/in^2$, an availability factor could be calculated assuming that all blinds degrade in a similar manner. The average dust level was 41 times higher than the suggested dust limit. Therefore, the agency divided the average total lead content of a blind by 41 (6,900/41) to yield 200 ppm lead as the standard.

A more protective specification limit could have been calculated using different assumptions and standards. Simply calculating the specification using the width of a whole

hand instead of half a hand would cut the limit in half to 100 ppm. Performing the calculation using two hands instead of one would have further reduced the limit to 50 ppm. Using the maximum permissible lead ingestion limit of 0.5 μg/day as mandated by California law would lower the specification from 200 ppm to 6 ppm even if only half of one hand was used in the calculation. The irreversible, cumulative health risks posed by lead poisoning warrant a more protective limit for lead in vinyl than 200 ppm.

Appendix IV. Stabilizers in PVC

Polyvinyl chloride (PVC) requires the addition of stabilizers to prevent degradation because it contains chlorine. Small faults in the polymer chain can serve as starting points for degradation. During the obligatory heating steps of PVC processing, chlorine can leave the polymer chain and combine with hydrogen to form corrosive hydrogen chloride which further degrades the polymer. At the same time, double bonds are formed in the polymer. As the process proceeds the polymer loses increasing amounts of hydrogen chloride and forms increasing numbers of double bonds.[69] A positively charged metal can act as a stabilizer by stabilizing bound chlorine and binding free hydrochloric acid, thereby effectively neutralizing it and preventing further damage to the polymer. Common heat stabilizers in PVC formulation include lead and mixed metal soaps containing barium, calcium, zinc, and cadmium, and organotin compounds.

Lead historically has been used as a cheap, effective stabilizer of PVC since the 1950s. From a chemical perspective, it is useful in both rigid and flexible PVC products and in electrical cables due to its electrical properties as demonstrated by its presence in Sega Genesis cables, video cables, computer cables, and headphone cables.(Table 1)

Mixed metal soaps like barium/cadmium, barium/zinc, and calcium/zinc act as stabilizers by replacing reactive chlorine atoms with less reactive atoms or groups.[70] In a barium/cadmium soap a carboxylate group substitutes for chlorine which reacts with cadmium forming cadmium chloride. The cadmium chloride then reacts with barium forming barium chloride and regenerating the cadmium metal soap. The other soaps operate in a similar manner. Unfortunately the least toxic metals make the poorest, most expensive stabilizers. Calcium/zinc stabilizers provide less long-term stability than those containing toxic cadmium or lead.[71] To solve this problem, organotins have been introduced as PVC stabilizers. While reasonably effective, the immunotoxicity of some organotins in animals has raised concerns about their effects in humans.[72, 73] Organotins are also known endocrine disruptors.[74]

In addition, the substitution of organotins illustrates a common corporate strategy. When consumer or regulatory pressure threatens a toxic additive, manufacturers can delay regulation and maintain profitability by substituting an additive that has not been rigorously tested for health and environmental impacts. Organotins are poorly characterized and therefore make good candidates for this strategy. By the time the additive is demonstrated to be hazardous, another substitution can be made thereby maintaining both corporate profitability and possibly perpetuating consumer health risks.

Appendix V. Materials and Methods

Lead and cadmium measurements

The total lead and cadmium content of all items was measured using atomic absorption spectroscopy by Stat Analysis, Chicago, Illinois AIHA proficient, NIST/NVLAP accredited. Laboratory work was supervised by Benjamin Ruth, PhD Testing was conducted blind to prevent possible bias introduced by product recognition. Sample preparation proved to be an important determinant of variation. Complete ashing of samples in a Thermolyne 48000 muffle furnace at 480C for two hours helped reduce variation though some sample heterogeneity was observed. Nitric acid, hydrogen peroxide, hydrochloric acid and methylene chloride were reagent or analytical grade. A test tube acid digestion lead preparation procedure (AOAC 5.001-3) was combined with an organic digestion using analytical grade methylene chloride (NIOSH9076). Measurements were performed using a Varian SpectrAA200 atomic absorption spectrophotometer. All appropriate laboratory QA/QC procedures regarding standard curve tolerances were followed for all samples including use of blanks and matrix spikes every 10 samples and NIST reference standards.

Acid extraction tests

Acid extractability was performed using procedures developed by the Consumer Product Safety Commission.[75] Studies were conducted by Stat Analysis, Chicago, Illinois. A test portion of plastic material was mixed with a 50-fold mass of 0.07 N hydrochloric acid and agitated for one hour at 37C in the dark. The material was re-extracted for two hours and then a third time for three hours. All three fractions were combined and analyzed for lead content.

Accelerated aging tests

Accelerated aging tests were performed using procedures developed by the Consumer Product Safety Commission.[76] Studies were conducted by personnel at the Environmental Quality Institute at the University of North Carolina-Asheville, AIHA, ELLAP accredited. The project was supervised by Richard Maas, PhD, associate director of the Institute.

A weatherometer was constructed according to specifications given in ASTM G53 without the condensation features and validated by lab personnel for temperature stability and illumination.[77] Even though moisture is a significant agent in degradation of materials, dry conditions were used in these aging studies to permit observations under milder, more realistic conditions. Natural and accelerated aging can be correlated if sufficient numbers of variables are compared in both methods.[78] Ultraviolet lights identical to those used in the UVCON brand weatherometer were purchased from Atlas

Electric Devices Company, Chicago, Illinois. The wavelength output in the 295-365 nm region provides excellent correlation with natural sunlight and outdoor exposure.

Samples were exposed to alternating cycles of six hours duration. The heat and light cycle was conducted at 50C under ultraviolet light illumination in the UV-A region. The alternate cycle was conducted at room temperature without illumination. Samples were tested in triplicate in a single blind design to prevent possible bias caused by product recognition among lab personnel. Samples were wiped at weekly intervals. One Pace Wipe was used to wipe each sample ten times. Wipes were digested and analyzed to yield lead and cadmium content per area wiped.

Lead and cadmium determinations were performed by graphite furnace atomic absorption spectrophotometry (GFAAS) using two Thermo-Jarrel-Ash Model 11 or Model 12 spectrophometers with TJA Model 774 graphite atomizers. All appropriate laboratory QA/QC procedures regarding standard curve tolerances and standard additions were followed for all samples.

Study design

Vinyl items purchased in Chicago area national chain stores were tested for total lead at Stat Analysis, Chicago, Illinois. Stores included Disney, Kmart, Target, Toys R Us, Uncle Dan's, Wal-Mart, and Warner Bros. In addition to information provided by product labels, all items containing lead were verified by the laboratory as PVC plastic by the Bellstein test for chlorine. Products containing above 100 ppm lead were also tested for cadmium content. A representative sample of 19 lead-containing items was selected for purchase in other cities. Not every item was found in every city. The sample included soft vinyl of various thickness, PVC-covered cable, and rigid PVC. Toys, clothing, household items, and cables were all included in the sample. Cadmium testing was restricted to items from Chicago, Los Angeles, San Francisco, and Montreal, Canada to control cost. A smaller sample of 7 products was submitted in triplicate for accelerated aging experiments as described above. The sample included soft and rigid PVC products designed for use by children. Most of the items submitted for the accelerated aging study were purchased in California.

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