

re has been
as yet to be
rs, regulatory
active risk to
ntradictory. In
substance and
primarily on
element to be
ely to cause
ne California
J a statement
1974) that "to
the basis of
e compounds
ganism." The
Environmental
I that humans
tive effects of
conclusion is
ent methylators
than humans
tific communi-
man teratogen,
despread contin-
guing industri-
oid's potential
studies should
ed teratogenesis
enic exposure,
ctive toxin.

inking water and the
geon caudal obtenu-
-29.
atural workers (letter).
effects in and around
isks in and around a
ter workers. *Hereditas*
with adult respiratory

- Börzsönyi, M., Bereczky, A., Rudnai, P., Csanady, M., and Horvath, A. 1992. Epidemiologic studies on human subjects exposed to arsenic in drinking water in Southeast Hungary. *Arch. Toxicol.* 66:77-78.
- Bower, C., Raymond, M., Lumley, J., and Bury, G. 1993. Trends in neural tube defects, 1980-1989. *Med. J. Aust.* 158:152-154.
- Brender, J. D., and Suarez, S. 1990. Paternal occupation and anencephaly. *Am. J. Epidemiol.* 131:517-521.
- Brook, F. A., Shum, A. S., Van Straaten, H. W., and Copp, A. J. 1991. Curvature of the caudal region is responsible for failure of neural tube closure in the curly tail (ct) mouse embryo. *Development* 113:671-678.
- Brouwer, O. F., Onkenhout, W., Edelbroek, P. M., de Kom, J. F., de Wolff, F. A., and Peters, A. C. 1992. Increased neurotoxicity of arsenic in methylenetetrahydrofolate reductase deficiency. *Clin. Neurol. Neurosurg.* 94:307-310.
- Buchanan, W. D. 1962. *Toxicity of arsenic compounds*. New York: Elsevier.
- Buchet, J. P., and Lauwerys, R. R. 1987. Study of factors influencing the in-vivo methylation of inorganic arsenic in rats. *Toxicol. Appl. Pharmacol.* 91:65-74.
- Burns, L. A., Spriggs, T. L., Fuchs, B. A., and Munson, A. E. 1994. Gallium arsenide-induced increase in serum corticosterone is not responsible for suppression of the IgM antibody response. *J. Pharmacol. Exp. Ther.* 268:740-746.
- Busam, K. J., Roberts, D. J., and Golden, J. A. 1993. Two distinct anterior neural tube defects in a human fetus: Evidence for an intermittent pattern of neural tube closure. *Teratology* 48:399-403.
- California Department of Health Services, Air Toxicology and Epidemiology Section. 1990. Proposed identification of inorganic arsenic as a toxic air contaminant. Part B. California Air Resources Board, Stationary Source Division. (Health Effects of Inorganic Arsenic Compounds.) May.
- Campbell, L. R., Dayton, D. H., and Sohal, G. S. 1986. Neural tube defects: A review of human and animal studies on the etiology of neural tube defects. *Teratology* 34:171-187.
- Chaîneau, E., Binet, S., Pol, D., Chatellier, G., and Meninger, V. 1990. Embryotoxic effects of sodium arsenite and sodium arsenate on mouse embryos in culture. *Teratology* 41:105-112.
- Cherry, F. F., Bennell, E. A., Bazzano, G. S., Johnson, L. K., Fosmire, G. J., and Barson, H. K. 1981. Plasma zinc in hypertension/toxemia and other reproductive variables in adolescent pregnancy. *Am. J. Clin. Nutr.* 34:2367-2375.
- Committee on Medical and Biological Effects of Environmental Pollutants, National Academy of Sciences. 1977. Effects of sodium arsenite on fetal development. *Bull. Environ. Contam. Toxicol.* 7:216-222.
- Copp, A. J., Brook, F. A., and Roberts, H. J. 1988. A cell-type-specific abnormality of cell proliferation in mutant (curly-tail) mouse embryos developing spinal neural tube defects. *Development* 104:285-295.
- Craig, J. C., Bennet, G. D., Dimichele, L. V., and Finnell, R. H. 1995. Screening for reproductive toxicity in *Fundulus heteroclitus* by genetic expression profiling (abstr.) *Teratology* 51:175.
- Dallaire, L., and Beliveau, R. 1992. Phosphate transport by capillaries of the blood-brain barrier. *J. Biol. Chem.* 267:22323-22327.
- Deneke, S. M. 1992. Induction of cystine transport in bovine pulmonary artery endothelial cells by sodium arsenite. *Biochim. Biophys. Acta* 1109:127-131.
- Dong, J., and Luo, X. 1993. Arsenic-induced DNA-strand breaks associated with DNA-protein crosslinks in human fetal lung fibroblasts. *Mutat. Res.* 302:97-102.
- Eastman, N. J. 1931. The arsenic content of the human placenta following arsphenamine therapy. *Am. J. Obstet. Gynecol.* 21:60-64.
- Edelman, G. M. 1992. Morphoregulation. *Dev. Dynamics* 193:2-10.
- Elwood, J. M., and Elwood, J. H. 1980. *Epidemiology of anencephalus and spina bifida*. Oxford: Oxford University Press.
- Fang, W. H., Li, G. M., Longley, M., Holmes, J., Thilly, W., and Modrich, P. 1993. Mismatch repair and genetic stability in human cells. *Cold Spring Harbor Symp. Quant. Biol.* 58:597-603.

- Ferm, V. H., and Carpenter, S. J. 1968. Malformations induced by sodium arsenate. *J. Reproduct. Fertil.* 17:199-201.
- Ferm, V. H., and Hanlon, D. P. 1983. Metal-induced congenital malformations. In *Reproduction and developmental toxicity of metals*, eds. T. W. Clarkson, G. F. Nordberg, and P. R. Sager, pp. 383-397. New York: Plenum Press.
- Ferm, V. H., and Hanlon, D. P. 1986. Arsenate-induced neural tube defects not influenced by constant rate administration of folic acid. *Pediatr. Res.* 20:761-762.
- Ferm, V. H., and Kilham, L. 1977. Synergistic teratogenic effects of arsenic and hyperthermia in hamsters. *Environ. Res.* 14:483-486.
- Field, B., and Kerr, C. 1979. Herbicide use and incidence of neural tube defects (letter). *Lancet* 1:1341-1342.
- Finnell, R. H., Moon, S. P., Abbott, L. C., Golden, J. A., and Chernoff, G. F. 1986. Strain differences in heat-induced neural tube defects in mice. *Teratology* 33:247-252.
- Finnell, R. H., Bennet, G. D., Karras, S. B., and Mohl, V. K. 1988. Common hierarchies of susceptibility to the induction of neural tube defects in mouse embryos by valproic acid and its 4-propyl-4-pentenoic acid metabolite. *Teratology* 38:313-320.
- Finnell, R. H., van Waes, M., Bennett, G. D., and Eberwine, J. H. 1993. Lack of concordance between heatshock proteins and the development of tolerance to teratogen induced neural tube defects. *Dev. Genet.* 14:137-147.
- Fisher, N. L., and Smith, D. W. 1981. Occipital encephalocele and early gestational hyperthermia. *Pediatrics* 68:480-483.
- Fowden, A. L. 1994. Fetal metabolism and energy balance. In *Textbook of fetal physiology*, eds. G. D. Thornburn and R. Harding, pp. 70-82. New York: Oxford University Press.
- Gale, T. F., and Layton, W. M., Jr. 1978. A variable embryotoxic response to lead in different strains of hamsters. *Environ. Res.* 17:325-333.
- Gilani, S. H., and Alibhai, Y. 1990. Teratogenicity of metals to chick embryos. *J. Toxicol. Environ. Health* 30:23-31.
- Golden, J., and Chernoff, G. 1993. Intermittent pattern of neural tube closure in two strains of mice. *Teratology* 47:73-80.
- Golden, J., and Chernoff, G. 1995. Multiple sites of anterior neural tube closure in humans: Evidence from anterior neural tube defects (anencephaly). *Pediatrics* 360:506-510.
- Gunn, T. M., Juriloff, D. M., and Harris, M. 1992. Further genetic studies of the cause of exencephaly in SELH mice. *Teratology* 45:679-686.
- Hall, J. G., Friedman, J. M., Kenna, B. A., Popkin, J., Jawanda, M., and Arnold, W. 1988. Clinical genetic, and epidemiological factors in neural tube defects. *Am. J. Hum. Genet.* 43:827-837.
- Hanlon, D. P., and Ferm, V. H. 1974. Possible mechanisms of metal ion-induced teratogenesis. *Teratology* 9:A18-A19.
- Hanlon, D. P., and Ferm, V. H. 1977. Placental permeability of arsenate ion during early embryogenesis in the hamster. *Experientia* 33:1221-1222.
- Hanlon, D. P., and Ferm, V. H. 1986. Teratogen concentration changes as the basis of the heat stress enhancement of arsenate teratogenesis in hamsters. *Teratology* 34:189-193.
- Harrison, W. P., and Hood, R. D. 1981. Prenatal effects following exposure of hamsters to sodium arsenite by oral or intraperitoneal routes. *Teratology* 23:40A.
- Hayes, W. J., Jr., and Laws, E. R., Jr., eds. 1991. *Handbook of pesticide toxicology*, vol. 1, *General principles*. New York: Academic Press.
- Holmes, L., Driscoll, S., and Atkins, L. 1976. Etiologic heterogeneity of neural tube defects. *N. Engl. J. Med.* 294:365-369.
- Hood, R. D., Vedel, G. C., Zaworotko, M. J., Tatum, F. M., and Mecks, R. G. 1987. Distribution, metabolism and fetal uptake of pentavalent arsenic in pregnant mice following oral versus intraperitoneal administration. *Teratology* 35:19-25.
- Hood, R. D., Vedel, G. C., Zaworotko, M. J., Tatum, F. M., and Mecks, R. G. 1988. Uptake, distribution and metabolism of trivalent arsenic in pregnant mouse. *J. Toxicol. Environ. Health* 25:423-434.

- Hopenhayn-Rich, C. the methylator 60:161-177.
- Huang, H., Huang, defense against 79:195-204.
- Hughes, M. F., Men arsenate in mic
- Hunter, F. T., Kip, as potassium at
- IARC. 1980. IARC humans. Some
- Jameson, S. 1976. genital malform
- Jha, A. N., Noditi, arsenite on hu
- Kaufman, M. H. 1 embryo. *Am. J.*
- Keeling, J. W., and examination. I
- York: Springer-
- Knowles, F. C., a 8:178-180.
- Lantzy, K., and Ma man's impact.
- Layton, M. W., Jr. associated diff
- Laurence, K. M. 1' and neonatal p
- Lee, H., Cortes, J. embryos. *Tera*
- Lee, T. C., Tanaki amplification
- Lemire, R. J., Bec Anencephaly.
- Leonard, A., and arsenic. *Muta*
- Li, W., and Chou tathione leve
- Li, J. M., and Ro mechanism c
- Lindhout, D., an *Lancet* 9:139
- Lindhout, D., On infants pren
- Lindgren, A., Dai and arsenate
- vitro. *Acta. F*
- Lugo, G., Cassad tal death. A
- Ma, L., Zhang, mice. *Chung*
- Marafante, E. an orally admi:

- um arsenate. *J. Reproduct.*
- ormations. In *Reproduction*
- ordberg, and P. R. Sager,
- defects not influenced by
- enic and hyperthermia in
- ube defects (letter). *Lancet*
- ff, G. F. 1986. Strain dif-
- 3:247-252.
- ommon hierarchies of sus-
- ryos by valproic acid and
1993. Lack of concordance
- teratogen induced neural
- arly gestational hyperther-
- ok of fetal physiology, eds.
- University Press.
- ponse to lead in different
- chick embryos. *J. Toxicol.*
- a closure in two strains of
- neural tube closure in humans:
- 360:506-510.
- udies of the cause of exan-
- l., and Arnold, W. 1988.
- effects. *Am. J. Hum. Genet.*
- ion-induced teratogenesis.
- arsenate ion during early
- ges as the basis of the heat
- y 34:189-193.
- 3 exposure of hamsters to
- JA.
- esticide toxicology, vol. 1,
- of neural tube defects. *N.*
- and Mecks, R. G. 1987.
- in pregnant mice following
- ecks, R. G. 1988. Uptake,
- se. *J. Toxicol. Environ. Health*
- Hopenhayn-Rich, C., Smith, A. H., and Goeden, H. M. 1993. Human studies do not support the methylation threshold hypothesis for the toxicity of inorganic arsenic. *Environ. Res.* 60:161-177.
- Huang, H., Huang, C. F., Wu, D. R., Jinn, C. M., and Jan, K. Y. 1993. Glutathione as a cellular defense against arsenite toxicity in cultured Chinese hamster ovary cells. *Toxicology* 79:195-204.
- Hughes, M. F., Menache, M., and Thompson, D. J. 1994. Dose dependent disposition of sodium arsenate in mice following acute oral exposure. *Fundam. Appl. Toxicol.* 22:80-89.
- Hunter, F. T., Kip, A. F., and Irvine, J. W. 1942. Radioactive tracer studies on arsenic injected as potassium arsenite. *J. Pharmacol. Exp. Ther.* 76:207-200.
- IARC. 1980. IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans. Some metals and metallic compounds. *IARC Monogr.* 23:39-141.
- Jameson, S. 1976. Variations in maternal serum zinc during pregnancy and correlation to congenital malformations, dysmaturity, and abnormal parturition. *Acta Med. Scand.* 593:21-37.
- Jha, A. N., Noditi, M., Nilsson, R., and Natarajan, A. T. 1992. Genotoxic effects of sodium arsenite on human cells. *Mutat. Res.* 284:215-221.
- Kaufman, M. H. 1979. Cephalic neurulation and optic vessel formation in the early mouse embryo. *Am. J. Anat.* 155:425-444.
- Keeling, J. W., and Boyd, P. A. 1993. Congenital malformations, prenatal diagnosis and fetal examination. In *Fetal and neonatal pathology, 2nd ed.*, ed. J. W. Keeling, pp. 111-147. New York: Springer-Verlag.
- Knowles, F. C., and Benson, A. A. 1983. The biochemistry of arsenic. *Trends Biochem. Sci.* 8:178-180.
- Lantzy, K., and MacKenzie, F. 1979. Atmospheric trace metals: Global cycles and assessment of man's impact. *Geochim. Cosmochim. Acta* 43:511-525.
- Layton, M. W., Jr., and Layton, M. W. 1979. Cadmium induced limb defects in mice: Strain associated differences in sensitivity. *Teratology* 19:229-236.
- Laurence, K. M. 1993. Hydrocephalus and malformations of the central nervous system. In *Fetal and neonatal pathology, 2nd ed.*, ed. J. W. Keeling, pp. 541-568. New York: Springer-Verlag.
- Lee, H., Cortes, J. L., and Levin, M. A. 1972. Teratogenic effects of phleomycin in early chick embryos. *Teratology* 6:201-206.
- Lee, T. C., Tanaka, N., Lamb, P. W., Gilmer, T. M., and Barrett, J. C. 1988. Induction of gene amplification by arsenic. *Science* 241:79-81.
- Lemire, R. J., Beckwith, J. B., Warkany, J. 1978. Incidences, etiology, and epidemiology. In *Anencephaly*. New York: Raven Press.
- Leonard, A., and Lauwerys, R. R. 1980. Carcinogenicity, teratogenicity and mutagenicity of arsenic. *Mutat. Res.* 75:46-62.
- Li, W., and Chou, I. N. 1992. Effects of sodium arsenite on the cytoskeleton and cellular glutathione levels in cultured cells. *Toxicol. Appl. Pharmacol.* 114:132-139.
- Li, J. M., and Rossman, T. G. 1989. Inhibition of DNA ligase activity by arsenite: A possible mechanism of its comutagenesis. *Mol. Toxicol.* 2:1-9.
- Lindhout, D., and Schmidt, D. 1986. In-utero exposure to valproate and neural tube defects. *Lancet* 9:1392-1393.
- Lindhout, D., Omtzigt, J. G. C., and Cornel, M. C. 1992. Spectrum of neural tube defects in 34 infants prenatally exposed to antiepileptic drugs. *Neurology* 42(suppl. 5):111-118.
- Lindgren, A., Danielsson, B. R. G., Dencker, L., and Vahter, M. 1984. Embryotoxicity of arsenite and arsenate: Distribution in pregnant mice and monkeys and effects on embryonic cells in vitro. *Acta. Pharmacol. Toxicol.* 54:311-320.
- Lugo, G., Cassady, G., and Palmisano, P. 1969. Acute maternal arsenic intoxication with neonatal death. *Am. J. Dis. Child.* 117:328-330.
- Ma, L., Zhang, C., and Liu, W. J. 1994. Effects of arsenic on the offspring development in mice. *Chung-Hua-Yu-Fang-I-Hsueh-Tsa-Chih* 28:20-23.
- Marafante, E. and Vahter, M. 1987. Solubility, retention and metabolism of intratracheally and orally administered inorganic arsenic compounds in the hamster. *Environ. Res.* 42:72-82.

- Martin, R. A., Fineman, R. M., and Jorde, L. B. 1983. Phenotypic heterogeneity in neural tube defects: A clue to causal heterogeneity. *Am. J. Med. Genet.* 16:519-525.
- McKinney, J. D. 1992. Metabolism and disposition of inorganic arsenic in laboratory animals and humans. *Environ. Geochem. Health* 4:43-48.
- Milunsky, A., Ulcickas, M., Rothman, K. J., Willet, W., Jick, S. S., and Jick, H. 1992. Maternal heat exposure and neural tube defects. *J. Am. Med. Assoc.* 268:882-885.
- Mirkes, P. E., and Cornel, L. 1992. A comparison of sodium arsenite- and hyperthermia-induced stress responses and abnormal development in post implantation rat embryos. *Teratology* 46:251-259.
- Moriss-Kay, G. M., Wood, H., and Chen, W. 1994. Normal neurulation in mammals. In *Ciba Foundation Symposium, Neural tube defects*, pp. 51-69. New York: John Wiley & Sons.
- Mottet, N. K., and Ferm, V. H. 1983. The congenital teratogenicity and perinatal toxicity of metals. In *Reproduction and developmental toxicity of metals*, eds. T. W. Clarkson, G. F. Nordberg, and P. R. Sager, pp. 93-125. New York: Plenum Press.
- MRC Vitamin Research Study Group 1991. Prevention of neural tube defects: Results of the MRC vitamin study. *Lancet* 338:132-137.
- Muckter, H., Islambouli, S., Docklea, E., Hopfer, C., Szinicz, L., Fichtl, B., and Forth, W. 1993. Isolated rat kidney tubules as a screening system for arsenic antidotes. *Toxicol. Appl. Pharmacol.* 121:118-128.
- Muller, F., and O'Rahilly, R. 1987. The development of the human brain, the closure of the caudal neuropore and the beginning of the second neurulation at stage 12. *Anat. Embryol.* 176:413-430.
- Muller, W. U., Streffer, C., and Fischer-Lahdo, C. 1986. Toxicity of sodium arsenate in mouse embryos in-vitro and its influence on radiation risk. *Arch. Toxicol.* 59:172-175.
- Munger, R., Isacson, P., Kremer, J., Hanson, J., Burns, T., Cherryholmes, K., and Hausler, W., Jr. 1992. Birth defects and pesticide-contaminated water supplies in Iowa. *Am. J. Epidemiol.* 136:959 (abstr.).
- Nagymajtenyi, L., Selyes, A., and Berencsi, G. 1985. Chromosomal aberrations and fetotoxicity effects of atmospheric arsenic exposure in mice. *J. Appl. Toxicol.* 5:60-63.
- Nordstrom, S., Beckman, L., and Nordenson, I. 1978a. Occupational and environmental risks in and around a smelter in northern Sweden. I. Variations in birthweight. *Hereditas* 88:43-46.
- Nordstrom, S., Beckman, L., and Nordenson, I. 1978b. Occupational and environmental risks in and around a smelter in northern Sweden. III. Frequencies of spontaneous abortion. *Hereditas* 88:51-54.
- Nordstrom, S., Beckman, L., and Nordenson, I. 1979a. Occupational and environmental risks in and around a smelter in northern Sweden. V. Spontaneous abortion among female employees and decreased birth weight among their offspring. *Hereditas* 90:291-296.
- Nordstrom, S., Beckman, L., and Nordenson, I. 1979b. Occupational and environmental risks in and around a smelter in northern Sweden. VI. Congenital malformations. *Hereditas* 90:297-302.
- Odanaka, Y., Matano, O., and Goto, S. 1980. Biomethylation of inorganic arsenic by the rat and some laboratory animals. *Bull. Environ. Contam. Toxicol.* 24:452-459.
- Pershagen, G. 1986. Sources of exposure and biological effects of arsenic. In *Environmental carcinogens, selected methods of analysis*, eds. I. K. O'Neill, P. Schuller, and L. Fishbein, pp. 45-61. Lyon: International Agency for Research on Carcinogens.
- Petres, I., Baron, D., and Hagedorn, M. 1977. Effects of arsenic cell metabolism and cell proliferation: Cytogenic and biochemical studies. *Environ. Health Perspect.* 19:223-227.
- Rasco, J. F., and Hood, R. D. 1994. Effects of maternal restraint stress and sodium arsenate in mice. *Reprod. Toxicol.* 8:49-54.
- Rein, K. A., Borrebaek, B., and Bremer, J. 1979. Arsenite inhibits β -oxidation in isolated rat liver mitochondria. *Biochim. Biophys. Acta* 574:487-494.
- Schoenwolf, G. C., and Smith, J. L. 1990. Mechanisms of neurulation: Traditional viewpoint and recent advances. *Development* 109:243-270.
- Scott, J. M., Weir, D. G., Molloy, S., McPartlin, J., Daly, L., and Kirke, P. 1994. Folic acid metabolism and mechanisms of neural tube defects. *Ciba Foundation Symp.* 181:180-187.

- Sever, L. E., Hesol, N. A., and Jorde, L. B. 1983. Congenital malformations associated with arsenic exposure. *Am. J. Med. Genet.* 16:251-254.
- Smith, A. H., Hopent, H. M., Wood, H., and Jorde, L. B. 1983. Drinking water. *Environ. Health Perspect.* 53:1-10.
- Stevens, J. T., Halle, J. W., and Jorde, L. B. 1977. Disposition of arsenic in the peroral administration. *Environ. Health Perspect.* 24:1-10.
- Stohrer, G. 1991. Arsenic and neural tube defects. *Environ. Health Perspect.* 89:1-10.
- Tabacova, S. 1986. Maternal arsenic exposure and neural tube defects: A review. *Environ. Health Perspect.* 68:1-10.
- Tabacova, S., and Hult, C. R. 1986. The role of arsenic in neural tube defects: A review of the literature. *Environ. Health Perspect.* 68:1-10.
- Tabacova, S., Little, J. B., and Jorde, L. B. 1986. Pregnancy in relation to arsenic exposure. *Reprod. Toxicol.* 1:1-10.
- Tabacova, S., Baird, D. D., and Jorde, L. B. 1986. Arsenic and cadmium in the per smelter area. *Environ. Health Perspect.* 68:1-10.
- Taubeneck, M. W., and Jorde, L. B. 1986. Arsenic metabolism in the Texas Department of Health. *Environ. Health Perspect.* 68:1-10.
- Thompson, D. J. 1991. Arsenic and neural tube defects. *Environ. Health Perspect.* 89:1-10.
- Tseng, W. P. 1977. Exposure to arsenic in drinking water. *Environ. Health Perspect.* 24:1-10.
- Umpierre, C. 1981. Arsenic and neural tube defects. *Environ. Health Perspect.* 43:1-10.
- U.S. Public Health Service. 1986. Profile for Arsenic. *Environ. Health Perspect.* 68:1-10.
- Vahter, M. 1981. Biotransformation of arsenic in rats. *Environ. Res.* 25:1-10.
- Vahter, M. 1994. Speciation of arsenic in urine. *Chem. Res.* 8:175-180.
- Vahter, M., and Maron, D. M. 1981. Arsenate in mice. *Environ. Health Perspect.* 43:1-10.
- Vahter, M., and Maron, D. M. 1981. Arsenite in mice. *Environ. Health Perspect.* 43:1-10.
- Van Allen, M. L., Kalay, M., and Jorde, L. B. 1986. The neural tube defect. *Environ. Health Perspect.* 68:1-10.
- Wang, C., Lin, J. M., and Jorde, L. B. 1986. 3T3 cells: Alteration of cell formation. *Arch. Environ. Health.* 41:1-10.
- Warkany, J., and Peterson, R. 1977. In rats produced. *Environ. Health Perspect.* 24:1-10.
- Webb, J. L. 1966. *Environ. Health Perspect.* 24:1-10.
- Weisenburger, D. D., Anderson, J. R., and Jorde, L. B. 1986. Ecologic study. *Environ. Health Perspect.* 68:1-10.
- White, F. M. M., Co, J. B., and Jorde, L. B. 1986. Arsenic and stillbirths in Iowa. *Environ. Health Perspect.* 68:1-10.

rogeneity in neural tube
225.

n laboratory animals and

Jick, H. 1992. Maternal
985.

id hyperthermia-induced
rat embryos. *Teratology*

on in mammals. In *Ciba*
ohn Wiley & Sons.

nd perinatal toxicity of
T. W. Clarkson, G. F.

defects: Results of the

B., and Forth, W. 1993.
antidotes. *Toxicol. Appl.*

rain, the closure of the
stage 12. *Anat. Embryol.*

ium arsenate in mouse
:172-175.

K., and Hausler, W., Jr.
Iowa. *Am. J. Epidemiol.*

erations and fetotoxicity.
4-63.

l environmental risks in
rt. *Hereditas* 88:43-46.

l environmental risks in
spontaneous abortion.

l environmental risks in
nong female employees

i environmental risks in
ions. *Hereditas* 90:297-

anic arsenic by the rat
59.

ic. In *Environmental car-*
; and L. Fishbein, pp.

ibolism and cell prolif-
9:223-227.

nd sodium arsenate in

ion in isolated rat liver

ditional viewpoint and

é, P. 1994. Folic acid
ymp. 181:180-187.

Sever, L. E., Hesol, N. A., Gilbert, E. S., and McIntyre, J. M. 1988. The prevalence at birth of congenital malformations in communities near the Hanford site. *Am. J. Epidemiol.* 127:243-254.

Smith, A. H., Hopenhayne-Rich, C., Bates, M. N., Goeden, H. M., Hertz-Picciotto, I., Duggan, H. M., Wood, R., Kosnett, M. J., and Smith, M.T. 1992. Cancer risks from arsenic in drinking water. *Environ. Health Perspect.* 97:259-267.

Stevens, J. T., Halle, L. L., Farmer, J. D., DiPasquale, L. C., Chernoff, N., and Durham, W. F. 1977. Disposition of ¹⁴C and/or ⁷⁵As-cacodylic acid in rats after intravenous, intratracheal or peroral administration. *Environ. Health Perspect.* 19:151-157.

Stohrer, G. 1991. Arsenic: Opportunity for risk assessment. *Arch. Toxicol.* 65:525-531.

Tabacova, S. 1986. Maternal exposure to environmental chemicals. *Neurotoxicology* 72:421-440.

Tabacova, S., and Hunter, E. S. 1993. Effects of arsenic on cultured mouse embryos in relation to valency, gestational rate and duration of exposure. *Teratology* 47:401 (abstr.).

Tabacova, S., Little, R. E., Balabaeva, L., Pavlova, S., and Petrov, I. 1994a. Complications of pregnancy in relation to maternal lipid peroxidation glutathione and exposure to metals. *Reprod. Toxicol.* 8:217-224.

Tabacova, S., Baird, D. D., Balabaeva, L., Lolova, D., and Petrov, I. 1994b. Placental arsenic and cadmium in relation to lipid and glutathione levels in maternal-infant pairs from a copper smelter area. *Placenta* 15:873-881.

Taubeneck, M. W., Daston, G. P., Rogers, J. M., and Keen, C. L. 1994. Altered maternal zinc metabolism following exposure to diverse developmental toxicants. *Reprod. Toxicol.* 8:25-40.

Texas Department of Health. 1992. An investigation of a cluster of neural tube defects in Cameron County, Austin, Texas.

Thompson, D. J. 1993. A chemical hypothesis for arsenic methylation in mammals. *Chem. Biol. Interact.* 88:89-114.

Tseng, W. P. 1977. Effects and dose response relationships of skin cancer and blackfoot disease with arsenic. *Environ. Health Perspect.* 109-119.

Umpierre, C. 1981. Embryolethal and teratogenic effects of sodium arsenite in rats. *Teratology* 23:66A.

U.S. Public Health Service, Agency for Toxic Substance and Disease Registry. 1991. Toxicological Profile for Arsenic (draft for public comment, October), p. 48.

Vahter, M. 1981. Biotransformation of trivalent and pentavalent inorganic arsenic in mice and rats. *Environ. Res.* 25:286-293.

Vahter, M. 1994. Species differences in the metabolism of arsenic compounds. *Appl. Organomet. Chem.* 8:175-182.

Vahter, M., and Marafante, E. 1983. Intracellular interaction and metabolic fate of arsenite and arsenate in mice and rabbits. *Chem. Biol. Interact.* 47:29-44.

Vahter, M., and Marafante, E. 1987. Effects of low dietary intake of methionine, choline or proteins on the biotransformation of arsenite in the rabbit. *Toxicol. Lett.* 37:41-46.

Van Allen, M. I., Kalousek, D. K., and Chernoff, G. F. 1993. Evidence for multisite closure of the neural tube in humans. *Am. J. Med. Genet.* 47:723-743.

Wang, C., Lin, J. M., and Lazarides, E. 1992. Methylations of 70,000-Da heat shock proteins in 3T3 cells: Alterations by arsenite treatment, by different stages of growth and by virus transformation. *Arch. Biochem. Biophys.* 297:169-175.

Warkany, J., and Petering, H. G. 1972. Congenital malformations of the central nervous system in rats produced by maternal zinc deficiency. *Teratology* 5:319-334.

Webb, J. L. 1966. *Enzyme and metabolic inhibitors*, vol. 3, pp. 595-895. New York: Academic Press.

Weisenburger, D. D., Ullrich, F. A., Spalding, M. E., Spalding, R. F., Buehler, B. A., and Anderson, J. R. 1992. Birth defects and well water contamination by agri-chemicals: An ecologic study. 3rd International Symp. Issues in Health Safety and Agriculture, Saskatoon, May 10-15.

White, F. M. M., Cohen, F. G., Sherman, G., and McCurdy, R. 1988. Chemicals, birth defects and stillbirths in New Brunswick: Association with agricultural activity. *Can. Med. Assoc. J.* 138:117-123.

- Widnell, C. C., Baldwin, S. A., Davies, A., Martin, S., and Pastenak, C. A. 1990. Cellular stress induces a redistribution of the glucose transporter. *FASEB J.* 4:1634-1637.
- Willhite, C. C., and Ferm, V. H. 1984. Prenatal and developmental toxicology of arsenicals. *Adv. Exp. Med. Biol.* 177:205-228.
- Wilson, D. B., and Center, E. M. 1974. Neural cell cycle in the *Looptail* mutant of the mouse (Lp). *J. Embryol. Exp. Morphol.* 32:697-705.
- Winship, K. A. 1984. Toxicity of inorganic arsenic salts. *Adv. Drug React. Acute Poisoning Rev.* 3:129-160.
- World Health Organization. 1991. Environmental Health Criteria: Arsenic. Washington, DC: WHO.
- Yamauchi, H., and Yamamura, Y. 1984. Metabolism and excretion of orally administered dimethylarsinic acid in the hamster. *Toxicol. Appl. Pharmacol.* 74:134-140.
- Yamauchi, H., and Yamamura, Y. 1985. Metabolism and excretion of orally administered arsenic trioxide in the hamster. *Toxicology* 34:111-121.
- Zierler, S., Theodore, M., Cohen, A., and Rothman, K. J. 1988. Chemical quality of maternal drinking water and congenital heart disease. *Int. J. Epidemiol.* 17:589-594.

PNEU
OF S

Melin

Depar
West

Denni

Depar
Lafaye

Gary

Depar
West

The pu
the lung
styrene
(CGT)
hepatot
Swiss /
500-10
with the
tal, an-
increas
due to
and the
pared.
especia
inhibit
foxamir
epoxide

Styrene, us
and resins (M
cytochrome P--
several detoxif
metabolized by

Received 20 Ju
This work wa:
Gateway Fellowship
Center.

Address corres
School of Pharmacy
USA.

Jour

19
CSP Petition
Committee

Re:Petition HP 01-3
Petition for Ban on Use of CCA-treated Wood in
Playground Equipment

Dear Chairperson Brown,

I urge the Consumer Product Safety Commission to implement an immediate ban on the use of CCA (chromated-copper-arsenate) on wood used for wooden play structures. I also urge your agency to begin a review of CCA-treated wood for other uses such as picnic tables and decks. The risks associated with arsenic, especially for children, are unnecessary. Arsenic is carcinogenic, and has been linked to nerve damage and other health problems.

Sincerely,
Terri Becker
Terri Becker

~~Adkins, Patricia H.~~

*cut
petition
comment
20*

From: Rhonda Roff [RRoff@semtribe.com]
Sent: Friday, August 24, 2001 2:48 PM
To: abrown@cpsc.gov
Subject: kids and arsenic



Rhonda Roff.vcf

Ann Brown, Chairperson
U.S. Consumer Product Safety Commission
Washington, D.C. 20207-0001
Email: abrown@cpsc.gov <mailto:abrown@cpsc.gov>
Dear Chairperson Brown,
Re: Petition HP G1-3

Petition for Ban on Use of CCA-treated Wood in Playground Equipment
I urge the Consumer Product Safety Commission to implement an immediate ban on the use of CCA (chromated-copper-arsenate) on wood used for wooden play structures. I also urge your agency to begin a review of CCA-treated wood for other uses such as picnic tables and decks. The risks associated with arsenic, especially for children, are unnecessary. Arsenic is carcinogenic, and has been linked to nerve damage and other health problems.
Sincerely,

Rhonda Roff
Seminole Tribe of Florida
Water Resource Management
6300 Stirling Road
Hollywood, FL 33024
tel 954.966.6300x1125
fax 954.967.3489

<<Rhonda Roff.vcf>>

*CCP
P
A*

21

Stevenson, Todd A.

From: Gregory S. Kidd [gkidd@beyondpesticides.org]
Sent: Wednesday, September 05, 2001 11:23 AM
To: CPSC
Cc: Bill Walsh; Jay Feldman
Subject: Petition HP 01-3, Petition for Ban on Use of CCA Treated Wood in Playground Equipment
September 5, 2001

Office of the Secretary
Consumer Product Safety Commission
Washington, DC 20207

Re: Petition HP 01-3, Petition for Ban on Use of CCA Treated Wood in Playground Equipment.

Dear Sir or Madam:

Thank you for the opportunity to comment on the Environmental Working Group (EWG) and Healthy Building Network (HBN) petition for the ban on the use of CCA-treated wood in playground equipment. Beyond Pesticides and its members urge the Consumer Product Safety Commission (CPSC) to ban the use of CCA-treated wood in playground equipment based on the extreme health risks associated with exposure to both arsenic and hexavalent chromium (chromium (VI)) leaching out of CCA-treated wood.

I am submitting these comments on behalf of Beyond Pesticides/National Coalition Against the Misuse of Pesticides (Beyond Pesticides). Beyond Pesticides is a national, environmental non-profit organization. The organization has been focusing on the issue of the heavy-duty wood preservatives, namely the inorganic arsenicals (such as chromated copper arsenate or CCA), pentachlorophenol, and creosote since the 1980s, as the Environmental Protection Agency (EPA) worked through its Special Review of these pesticides. One of the most serious limitations of EPA's review of the inorganic arsenicals was a failure on the part of the agency to consider the special hazard to children from playing on playground equipment made from CCA-treated wood. EPA acknowledged this gap in the review and determined that "with the available data the exposure of children to the end uses of the treated wood (playground equipment) cannot be estimated" (U.S. EPA, 1981).

Prior to 1978, the inorganic arsenicals were used in a significant number of pesticide products to control insects, fungi, weeds and rodents as well as in wood preservatives. EPA began investigating the inorganic arsenicals in 1978 because of concerns that this family of chemicals presented risks of cancer, genetic mutation, and birth defects (U.S. EPA, 1993). During that review, EPA separated the use of inorganic arsenicals as wood preservatives from all other uses. In 1988, the agency banned almost all uses of nonwood-preservative pesticide products containing inorganic arsenicals because EPA determined that arsenic posed an unacceptable risk to workers and others exposed to arsenic (U.S. EPA, 1993). As of 1993 all uses of inorganic arsenicals have been prohibited except for the use of arsenic in wood preservatives.

The Toxicity of CCA is Well Documented

Wood preservative arsenicals are a mixture of ingredients. The most commonly used arsenical, CCA, is a mixture of arsenic acid, chromium (VI), and copper oxide, plus unlisted "inert" ingredients in proportions that vary with the particular product. According to the United States Geological Survey

(USGS) approximately 34,000 metric tons of arsenic was consumed in the U.S. in 2000 and production of CCA accounted for more than 90% (or well over 30,000 metric tons) of domestic consumption of arsenic (USGS, 2001).

Arsenic is a known human carcinogen. Several studies have shown that inorganic arsenic can increase the risk of lung cancer, skin cancer, bladder cancer, liver cancer, kidney cancer, and prostate cancer (ATSDR, 2001). The International Agency for Research on Cancer (IARC) (WHO, 1993a), the U.S. Department of Health and Human Services (NTP, 2001a) and EPA have determined that inorganic arsenic is a human carcinogen based on sufficient evidence from human data. (U.S. EPA, 1998a). The National Research Council's recent report on arsenic in drinking water concludes that there is sufficient evidence from human epidemiological studies in Taiwan, Chile, and Argentina that chronic ingestion of inorganic arsenic causes bladder, lung and skin cancer (NRC, 1999).

Chronic exposure to arsenic also causes damage to internal organs. Arsenic poisoning damages mucous membranes, irritates eyes, causes darkening and lesions of the skin, damages and inflames the liver, damages the heart and causes hearing loss (WHO, 1981). Exposure to arsenic causes degeneration of the peripheral nervous system (WHO, 1981).

The most common route of exposure with arsenic is ingestion, so it is not surprising that gastrointestinal (GI) problems are associated with acute arsenic poisoning. The predominate GI effects of ingestion are vomiting, abdominal pain and diarrhea (U.S. EPA, 1999). Other GI effects include inflammation and eventual sloughing of the mucous membrane in the mouth, pharynx and esophagus (U.S. EPA, 1999). The central nervous system is also affected by acute exposure with symptoms ranging from headache, dizziness and confusion, progressing to muscle weakness and spasms, hypothermia, lethargy, delirium, coma, and convulsions (U.S. EPA, 1999). Acute exposure also damages the kidney, heart, and liver (U.S. EPA, 1999).

EPA has calculated an oral cancer slope factor for arsenic of 1.5 per mg/kg-day. This cancer slope factor is used to estimate cancer risks associated with exposure to arsenic from both oral and dermal routes. EPA has also developed a reference dose for non-cancer health effects of arsenic exposure of 0.0003 mg/kg-day. EPA has determined that this oral reference dose represents a safe dose for a chemical, i.e. one that a person could receive every day without unreasonable risk of non-cancer health effects (Roberts, 2001).

Chromium (VI) is a known human carcinogen. Several studies have shown that chromium (VI) compounds can increase the risk of lung cancer (ATSDR, 2001b). Animal studies have also shown an increased risk of cancer (ATSDR, 2001b). IARC (WHO, 1993b), the U.S. Department of Health and Human Services (NTP, 2001b), and EPA have determined that chromium (VI) is a known human carcinogen (U.S. EPA, 1998b).

ATSDR reports that breathing high levels of chromium (VI) can cause irritation to the nose, such as runny nose, nosebleeds, and ulcers and holes in the nasal septum. Ingesting large amounts of chromium (VI) can cause stomach upsets and ulcers, convulsions, kidney and liver damage, and even death. Skin contact with certain chromium (VI) compounds can cause skin ulcers. Some people are extremely sensitive to chromium (VI). Allergic reactions consisting of severe redness and swelling of the skin have been noted (ATSDR, 2001b).

Arsenic when ingested is readily absorbed. This is significant because scientists have established that children often stick their hands and other objects into their mouths. A recent study using video tape to monitor children found that children put their hands in their mouths an average of six times per hour,

ranging up to 45 times per hour for some children (Zartarian, 1997). Daily estimates of soil ingestion by children average 179 mg/kg (Stanek, 1995).

Scientific Studies Prove that CCA Leaches Out of Treated Wood

Studies conducted by scientists and media outlets have documented highly elevated levels of arsenic in the soil beneath structures made of CCA-treated wood and on the surface of the wooden structures. It is clear as a result of these studies that arsenic and chromium are leaching out of CCA-treated wood at rates that pose an unreasonable risk to human and environmental health. The National Research Council has determined that consuming arsenic at the current drinking water standard of 100 mg per person per day can lead to a cancer risk of between 1 additional case in 100 to 1 in 1,000 (NRC, 1999).

Hazardous Substance & Waste Management Research, Inc. (HSWMR) calculated a target quantity of dislodgeable arsenic on the surface of CCA-treated wood in a report prepared for the American Wood Preservers Institute (AWPI). Based on corrected calculations, Dr. Teaf, the author of this report, determined that the Wood Surface Target Quantities (WSTQs) for children (ages 2 to 6) was 21 mg/100 cm² and the aggregate child/adult WSTQ (age 2 to 31) was 2 mg/100 cm² (Teaf, 2001). The WSTQ was determined in order to achieve circumstances where the routinely accepted Target Hazard Quotient of 1.0 for noncancer effects and the Target Risk of 1×10^{-6} for cancer risk would not be exceeded (HSWMR, 2001).

Stephen Roberts, Ph.D., with the University of Florida's Center for Environmental and Human Toxicology, conducted an analysis of three formal assessments of risk resulting from dislodgeable arsenic exposure through direct contact with CCA-treated wood. The studies include the above referenced DHSSC study from 1987, and HSWMR study from 2001, as well as a study by the Consumer Product Safety Commission (CPSC) in 1990. As part of that analysis, Dr. Roberts calculated the risk of cancer, based on EPA's oral cancer slope factor, associated with a range of levels of dislodgeable arsenic, assuming daily exposure for five years, as shown in Table 1.

Table 1. Cancer risks and daily doses associated with exposure to CCA-treated wood with different levels of dislodgeable arsenic (Roberts, 2001)

Dislodgeable arsenic (mg/100 cm ²)	Dose(mg/day)	Cancer risk
1	0.76	4.22×10^{-6}
10	7.6	4.22×10^{-5}
25	18.9	1.06×10^{-4}
35	26.7	1.48×10^{-4}
50	38.1	2.11×10^{-4}
100	76	4.22×10^{-4}
250	191	1.06×10^{-3}
632	482	2.67×10^{-3}

The studies outlined below have found that children could receive doses of arsenic as high as 1,260 mg from touching CCA-treated wood.

The Department of Health Services of the State of California (DHSSC) conducted a study of playground

structures to determine the health hazard posed to children by the heavy-duty wood preservatives, including inorganic arsenicals, pentachlorophenol (penta) and creosote. DHSSC had volunteers rub CCA-treated playground equipment and measured the amount of arsenic and chromium found on their hands. DHSSC determined that a child could receive doses of arsenic ranging from 1,260 mg to 236 mg, and doses of chromium ranging from 575 mg to 351 mg (DHSSC, 1987). The average dermal loading (corrected for surface area) was 50 mg/100 cm² (Roberts, 2001). DHSSC concluded that theoretical increase in lifetime cancer risk from arsenic exposure from playgrounds is between 1.0×10^{-4} and 5.0×10^{-3} (DHSSC, 1987).

Health and Welfare Canada (HWC) conducted a study to determine the amount of arsenic and chromium leaching out of CCA-treated wood into the soil and on to the surface of the wood. HWC measured arsenic in the soil beneath CCA-treated structures as high as 9.573 mg/kg, with background levels less than .0371 mg/kg (Galarneau, 1990). The surface wipe samples contained from 0.5 mg to 322 mg (mean 42.9 mg) of arsenic, and 1.2 mg to 254 mg (mean 27.8 mg) of chromium collected from a total surface area of 10 cm² (Galarneau, 1990). Correcting for surface area, the average amount of dislodgeable arsenic was 429 mg/100 cm². A second, similar study prepared for HWC reported, "on all sampling occasions, there was significant leaching of copper, chromium and arsenic" (Doyle, 1992). Rainwater run-off samples contained concentrations ranging from 1.49 mg/kg to 9.19 mg/kg for arsenic, and from 0.6 mg/kg to 3.75 mg/kg for chromium (Doyle, 1992). Soil samples revealed arsenic levels as high as 80 mg/kg for arsenic, and 53.8 mg/kg for chromium, background levels did not exceed 3.4 mg/kg (Doyle, 1992).

The Connecticut Department of Analytic Chemistry (CDAC) has conducted studies to determine the levels of arsenic and chromium leaching out of CCA-treated wood decks. Overall the CDAC found levels of arsenic in the soil as high as 350 mg/kg with a background average of 3.7 mg/kg (Stilwell, 1997). Chromium was found in the soil at levels as high as 154 mg/kg, with background levels averaging 20 mg/kg (Stilwell, 1997). A slightly more recent study by CDAC reported on arsenic and chromium on the surface of CCA-treated wood. The levels of arsenic dislodged from the wood surfaces, after 1 month of weathering (nominal), ranged from 6-122 mg/100 cm², and averaged 40 mg/100 cm² (Stilwell, 1998).

The Maine Bureau of Health (MEBOH) conducted an evaluation of potential health risks to children arising from exposure to arsenic in CCA-treated wood, focusing on arsenic. MEBOH considered four exposure pathways including incidental ingestion of soil contaminated with CCA leachate, dermal contact with such soil, incidental ingestion of dislodgeable arsenic from contact with wood surfaces, and dermal exposure to dislodgeable arsenic. In a draft of their report, MEBOH determined that incidental ingestion of arsenic from hand-to-mouth activity after skin contact with wood surfaces and transfer of dislodgeable arsenic represents the greatest contribution to total exposure, comprising approximately 75% and ingestion of soil comprising approximately 20% of total daily exposure (Carlson-Lynch, 1998). MEBOH calculated cancer risks associated with exposure to dislodgeable arsenic that far exceed EPA's target of 1×10^{-6} and concludes:

Assuming the lower bound estimate of hourly frequency of skin-to-mouth contact of 2 times per hour (and assuming that the hand is the body part most frequently contacting the mouth), and a conservative estimate of 2 hours of per day [sic] during good weather, a child who plays on or around CCA-treated structures 30 days per year could receive arsenic exposure in excess of the reference dose for noncancer effects. A child who plays on or around CCA-treated structures 90 days per year will have an estimated cancer risk of 1×10^{-4} (Carlson-Lynch, 1998).

The Florida Center for Solid and Hazardous Waste Management (FCSHWM) in cooperation with the Florida Department of Environmental Protection (DEP), the University of Florida and the University of

Miami has been working on CCA-treated wood projects since 1997. FCSHWM sampled soil from under 65 decks made of CCA-treated wood in various sites in Gainesville, Tallahassee and Miami. FCSHWM found an average of 28.5 mg/kg of arsenic with an average background level of 1.5 mg/kg (Townsend, 2001).

Conclusion: CPSC Must Act to Prohibit the Use of CCA and Other Arsenic Based Wood Preservatives on Playground Equipment

Congress has directed the Commission to "protect the public against unreasonable risks of injuries and deaths associated with consumer products." Children face an unreasonable and unnecessary risk of cancer every time they play on or near CCA-treated wood playground equipment. Scientific studies have established that children regularly stick their hands and other objects into their mouths. Science has demonstrated the extreme toxicity of arsenic and chromium (VI). Studies have proven that arsenic and chromium (VI) are leaching out of CCA-treated wood, onto the surface of the wood and into the soil around the treated structure. Children are ingesting doses of these toxic chemicals that science has shown can cause cancer. CPSC must act to prohibit the future use of inorganic arsenic wood preservatives on playground equipment.

Sincerely,

Gregory S. Kidd
Science and Legal Policy Director

Endnotes

Agency For Toxic Substances and Disease Registry, 2001a. ToxFAQs for Arsenic.
<http://www.atsdr.cdc.gov/tfacts2.html>

Agency For Toxic Substances and Disease Registry, 2001b. ToxFAQs for Chromium.
<http://www.atsdr.cdc.gov/tfacts7.html>

Carlson-Lynch, H., A.E. Smith. 1998. Evaluation of Children's Health Hazards from Arsenic Exposure Associated with the Use of CCA-Treated Wood in Residential Structures and Municipal Playgrounds. Draft Report submitted to Bureau of Health, Maine Department of Human Services.

Department of Health Services for the State of California, 1987. Evaluation of Hazards Posed by the Use of Wood Preservatives on Playground Equipment. Report to the Legislature.

Doyle, E. 1992. Field Study to Investigate the Leaching and Dislodgeability of Copper, Chromium and Arsenic Residues from CCA-C Treated Lumber and to Evaluate Means for Reducing Environmental Contamination and User Exposure. Prepared for Health and Welfare Canada.

Galareau, D. et al., 1990. Residues of Arsenic, Chromium and Copper on and Near Outdoor Structures Built of Wood Treated with "CCA" Type Preservatives. Presented before the Division of Environmental Chemistry, American Chemical Society, Washington, DC.

Hazardous Substance and Waste Management Research, Inc. 2001. Development of Wood Surface Target Quantity for Arsenic Based on Exposure to Decks or Playground Equipment Constructed of CCA-Treated Wood. <http://www.preservedwood.com/safety/wsreport.pdf>.

National Research Council, 1999. Arsenic in Drinking Water. National Academy Press. Washington, DC.

National Toxicology Program, 2001a. 9th Report on Carcinogens: Arsenic Compounds, Inorganic. <http://ehis.niehs.nih.gov/roc/ninth/known/arseniccmpds.pdf>.

National Toxicology Program, 2001b. 9th Report on Carcinogens: Chromium Hexavalent Compounds. http://ehis.niehs.nih.gov/roc/ninth/known/chromium_hex_comps.pdf.

Roberts, S.M. and H.O. Ochoa. 2001. Letter dated April 10, 2001, addressed to John Ruddell, Director, Division of Solid Waste with Florida Department of Environmental Protection.

Stanek, E.J. III, E.J. Calabrese. 1995. Daily Estimates of Soil Ingestion in Children. Environmental Health Perspectives. 103(3): 276-285.

Stilwell, D., K.D. Gorny, 1997. Contamination of Soil with Copper, Chromium, and Arsenic Under Decks Built with Pressure Treated Wood. Bull. Environ. Contam. Toxicol. 58:22-29.

Stilwell, D. 1998. Environmental Issues On The Use Of CCA Treated Wood. Prepared for the Department of Analytic Chemistry for the State of Connecticut. <http://www.caes.state.ct.us/FactsheetFiles/AnalyticalChemistry/fsAC001f.htm>

Teaf, C.M. 2001. Letter dated April 9, 2001, addressed to Scott Ramminger, President and CEO, AWPI.

Townsend T., et al. 2001. New Lines of CCA-Treated Wood Research: In Service and Disposal Issues. Report #00-12. http://www.floridacenter.org/publications/solo-gabrielle_00-12.PDF.

U.S. Environmental Protection Agency, 1981. Creosote, Inorganic Arsenicals, Pentachlorophenol: Position Document No. 2/3. Washington, DC.

U.S. Environmental Protection Agency, 1993. International Pesticide Notice. EPA Cancels the Last Agricultural Use of Arsenic Acid in the United States. <http://www.epa.gov/oppfead1/17b/r2.htm>.

U.S. Environmental Protection Agency, 1998a. Integrated Risk Information System: Arsenic, Inorganic, CASRN 7440-38-2. <http://www.epa.gov/iris/subst/0278.htm#II>.

U.S. Environmental Protection Agency, 1998b. Integrated Risk Information System: Chromium(VI), CASRN 18540-29-9. <http://www.epa.gov/iris/subst/0144.htm#II>.

U.S. Environmental Protection Agency, 1999. Recognition and Management of Pesticide Poisoning, 5th ed.

U.S. Geological Survey, 2001. Mineral Commodity Summaries: Arsenic. <http://minerals.usgs.gov/minerals/pubs/commodity/arsenic/160301.pdf>.

World Health Organization, 1981. Arsenic, Environmental Health Criteria, 18. Cited in: Caroline Cox, 1991. Chromated Copper Arsenate. Journal of Pesticide Reform 11(1):23-27.

World Health Organization, 1993a. Guidelines for drinking water quality: Arsenic. 2nd ed. http://www.who.int/water_sanitation_health/GDWQ/Chemicals/arsenicsum.htm

September 5, 2001

Office of the Secretary
Consumer Product Safety Commission
Washington, DC 20207

Re: Petition HP 01-3 Requesting a Ban on Use of CCA Treated Wood in Playgrounds

The Leathers & Associates design firm is the leader in the field of community-built playgrounds. In the past three decades, we have worked with more than 1,600 communities in all 50 states as well as in Canada, Australia, Israel, and New Zealand. The vast majority of those playgrounds were built using CCA-treated lumber, and we have never had a single report of anyone becoming ill from the chemical in the wood.

In 30 years of building, our designs and materials have evolved. Our construction methods are unique and durable. We continually monitor developments to stay current with the research and development of new products. We will continue to be forward thinking in order to remain the leaders in the industry. Based on our experience and research, the materials we use for our projects have naturally evolved so that we now use only the highest quality products available.

Depending on the type and quality of the wood, test results measuring the amount of arsenic in the soil can vary tremendously. We continue to recommend CCA-treated wood, although the wood we specify for treatment is No. 1 dense select structural Southern Yellow Pine, kiln-dried after treatment. This is the best grade of Southern Yellow Pine available, which helps ensure low maintenance and reliability. The quality of the wood also allows the chemicals to fixate to the wood better. Kiln-dried after treatment reduces the amount of excess chemical in the wood and allows it to be sealed or painted immediately. If a ban on CCA is under consideration, the types of wood and the quality can't be ignored because the differences are enormous.

In addition to using the best quality lumber and requiring the wood to be kiln-dried after treatment, we also recommend that the projects be sanded and sealed annually. We use the plastic composite Trex for the decking and handrails of our playgrounds because we believe that it is easier to maintain and reduces the number of splinters that children may receive.

As you know, many playgrounds in Florida were closed after a series of newspaper articles were published stating that arsenic in the playground equipment was poisoning the children. However, most of those playgrounds have now reopened after the cities conducted their own independent tests. They realized that the articles were an

Founding Member: Community Built Association

Leathers & Associates, Inc. • 99 Eastlake Road, Ithaca, NY 14850 • 607 277-1650

Fax: 607 277-1433 • E-mail: leathers@leathersassociates.com • Web: www.leathersassociates.com

CCA
Comments
22
2001 SEP 10 AM 10:10

Office of the Secretary, CPSC

Page 2

September 5, 2001

overreaction to the word "arsenic." CCA wood is treated with arsenate, a form of arsenic. Arsenic is a naturally occurring element in soil everywhere. Naturally occurring levels are much lower in Florida than most of the rest of the country. However, in the independent testing, levels found were still far below a level that would endanger children. In fact, one of the cities found arsenic levels to be much higher in a nearby soccer field than it was under the playground.

We have no objection to helping communities work with one of the copper-based alternative treated-wood products, if they choose to do so, but we so far have seen no reason to change our basic recommendation from a wood that is economical and has proven itself to be effective and safe through decades of use. The type of lumber we require has proven to yield better test results than other types. We are mindful that the structural integrity of playgrounds is a major factor in their safe use, and we are not eager to abandon the construction material that has served us so well in the past.

It is our understanding that scientific research has not yet demonstrated that children have been or will be harmed from playing on CCA-treated wood, while on the other hand wood treated with CCA has proven its structural integrity over the long term. We feel strongly that no decision on banning CCA-treated wood should be made without proof that there is a health risk. Obviously, the current controversy over treated lumber is much greater than wood used to build playgrounds (i.e., arsenic in drinking water, varying levels of naturally occurring arsenic in soils in different parts of the country). If banning CCA-treated lumber is being considered, the different types of wood and treatment processes also need to be taken into consideration, as various processes and quality of wood result in different levels of exposure to the chemical. We feel that the best solution to this controversy is for a neutral body, such as the Environmental Protection Agency, to develop a standard from which everyone can gauge test results. Once this standard is developed, we can all make more responsible decisions.

Sincerely,



Marc Leathers
President

Ann Brown, Chairperson
U.S. Consumer Product Safety Commission
Washington, D.C. 20207-0001

CCA
document 23

Dear Ms. Brown,

I urge the Consumer Product Safety Commission
put an immediate ban on the use of CCA on
wood for play structures. I hope your
agency will also review CCA-treated wood
for picnic table and decks, etc.

My concern is for the risks for children
where there is an association with arsenic.

Sincerely,

Nina Lerda

2070 19th St

Wyandotte, MI 48192

CCA
petition
24



State of Wisconsin
Scott McCallum, Governor

Department of Agriculture, Trade and Consumer Protection
James E. Harsdorf, Secretary

September 9, 2001

Petition HP 01-3
**Petition for Ban on Use of CCA-Treated Wood in
Playground Equipment**

Office of the Secretary
Consumer Product Safety Commission
Room 501
4330 East-West Highway
Bethesda, Maryland 20814

Dear Secretary:

We write in support of Petition HP 01-3 requesting a ban on the sale and use of chromated copper arsenate (CCA) treated wood in playground equipment, as docketed by the U.S. Consumer Product Safety Commission under provisions of the Federal Hazardous Substances Act and published in the July 13, 2001 Federal Register.

The Department of Agriculture, Trade and Consumer Protection administers and enforces Wisconsin's Hazardous Substances Act, adopted in the early 1970's to complement and parallel federal law.

Like its federal counterpart, Wisconsin law defines a hazardous substance to include "any toy or other article intended for use by children which the department by rule determines . . . to present an electrical, mechanical or thermal hazard or to contain a toxic substance either in or on the toy or other article." If the Department imposes this definition on a substance or article, various remedies can be pursued, including labeling, restrictions on access to the general public or prescribed methods of sale, package safety standards (if applicable), or an outright ban on the sale and distribution of the substance or article.

Inorganic arsenic has been declared a known human carcinogen by various federal agencies, the International Agency for Research on Cancer, the American Conference of Governmental Industrial Hygienists and others. Inorganic arsenic compounds, such as CCA, have also been designated as "extremely hazardous substances" under applicable EPA regulations. When inorganic arsenic compounds are applied to products available for sale and use by the general public, then the products themselves may be deemed as hazardous substances under current state and federal regulatory authority.

As a water-borne wood preservative, we know that CCA is comprised of varying proportions of hexavalent arsenate, an inorganic arsenic compound, in a concentration ranging from 16-45%. We also know that the amount of CCA used as a preservative in wood products is defined by its retention level and the particular application of the wood product. The CCA retention level for lumber used in most children's playground equipment will vary from 0.25 to 0.60 lbs. of preservative per cubic foot.

According to the Toxicological Profile for Arsenic published by the Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health & Human Services, the minimal risk level for chronic exposure from oral ingestion of inorganic arsenic (a daily exposure for 365 days or more before symptoms have been observed) is 0.0003 mg/kg of body weight per day. Acute exposure symptoms have resulted from an oral ingestion rate of 0.005 mg of arsenic/kg of body weight per day. As a standard of reference, some publications have identified a concentration of inorganic arsenic in treated lumber as high as 3,000 mg/kg of wood.

Perhaps this data would not be cause for concern if the fixation of CCA to wood fibers was an accurate assumption. Contrary to literature of the American Wood Preservers Institute, the elemental components of CCA do, in fact, leach from the wood according to recent studies – studies that have relied on extensive soil tests and hand wipe samples.

Another variable in determining risks associated with CCA-treated wood relates to the treated wood production process itself.

According to a recent research article prepared by the USDA's Forest Products Laboratory, the fixation of CCA in pressure-treated wood is dependent upon both temperature and storage time. Although the American Wood Preservers Association has considered the development of "leaching minimization" standards for CCA and other wood preservatives, we are unaware of any nationally recognized standards to govern these production processes. While production monitoring is performed in some instances by the American Lumber Standard Committee (ALSC) and some state regulatory agencies, treated wood products used in children's playground equipment and playsets do not require ALSC oversight. Therefore, wood treatment processors may shorten storage times to achieve increased board feet production levels, thereby increasing the product's leaching potential.

Applying an appropriate sealant may be an effective remedy to minimize leaching for some applications. However, manufacturers discourage use of a sealant during the first year in order to allow for further chemical fixation.

Scientists and others may disagree with the perceived and calculated public health and environmental risks associated with CCA-treated lumber, but we who work to safeguard children's health and safety have a responsibility to proceed more expeditiously.

Children face a higher risk of exposure to CCA and inorganic arsenic because of inconsistent hygiene practices, hand-to-mouth habits, and the tendency to play in dirt. Referencing a 1993 National Research Council study on children's susceptibility to hazardous substances, the Agency for Toxic Substances and Disease Registry stated the following:

"Children are not small adults. A child's exposure may differ from an adult's exposure in many ways. Children drink more fluids, eat more food and breathe more air per kilogram of body weight, and have a larger skin surface in proportion to their body volume. A child's diet often differs from that of adults... A child's behavior and lifestyle also influence exposure. Children crawl on the floor; they put things in their mouths; they may ingest inappropriate things such as dirt or paint chips; they spend more time outdoors. Children also are closer to the ground, and they do not have the judgement of adults in avoiding hazards."

Clearly, there are legitimate uses for CCA-treated lumber. Children's playground equipment, playsets, and treehouses are not some of them. Thus, we urge the Commission to move swiftly to ban the sale and use of chromated copper arsenicals in playground equipment.

Sincerely,



William L. Oemichen, Administrator
Division of Trade and Consumer Protection

Cc: Consumer Protection Bureau Director Fran Tryon
Regulatory Specialist Tom Stocbig

September 11, 2001

CCA
amount 25

Office of the Secretary
U.S. Consumer Product Safety Commission
Room 501
4330 East-West Highway
Bethesda, MD 20814

re: **Petition HP 01-3**
Petition for Ban on Use of CCA Treated Wood in Playground Equipment

Dear CPSC Secretary:

This letter and attachment is intended for inclusion in the CPSC docket that is reviewing the proposal to ban chromated-copper-arsenate (CCA) pressure-treated wood in playground equipment. CPSC is to be commended for accepting the task of reviewing this important issue. The data collected in Connecticut and elsewhere points out that arsenic leaches from the wood on an ongoing basis leading to contamination of soil underneath decks/playscapes. This also results in a dislodgeable residue of arsenic on the surface of the wood that is readily available to children from casual contact.

The exposure potential to children from the dislodgeable residue appears to be on the order of 20-200 ug of arsenic on the hands at any point during a play activity, which is a large arsenic source relative to others in a child's environment (see attached ppt presentation). According to a recent risk assessment from a contractor for the wood industry (Gradient, 2001; accessible at preservedwood.com), an estimate of the arsenic concentration in the dislodgeable residue is 1900 ppm. This estimate is similar to calculations of arsenic concentration in the residue based upon the residues found on the wood surface (10-100 ug As/100cm² range across 5 studies) and the amounts of dust that can be expected on a surface (Hawley, Risk Analy. 5:289-302, 1985). These estimates indicate that the dislodgeable residue is a relatively concentrated source of arsenic, many times higher than soil remediation standards which are aimed to protect children. Another way of looking at the issue is to consider the total amount of arsenic available on an average-sized 8x10 deck. Scaling up from the range of 10-100 ug/100cm² to the surface area of the deck yields 7.5 to 75 mg of arsenic potentially available. Although an average child would not be expected to contact such high mg quantities of arsenic during a single play event, these considerations point out that CCA-treated wood surfaces can represent a substantial and ongoing arsenic source in a child's environment.

There are a number of uncertainties in calculating exposures and risks to arsenic from CCA-treated wood. For example, when extrapolating from what is on the hands of a child to what is actually ingested, there is too little information on children's behaviors (e.g. mouthing frequency; amount of hand entering mouth; mouthing of other objects that may be contaminated; frequency of play activities per year) to know the population distribution of exposure and what is a reasonable bounding estimate. Further complicating the assessment are considerations of exposure from contaminated soil underneath the playscape and uptake via the dermal route. Perhaps the upcoming SAP review of this issue for EPA will provide insights into how to better calculate these exposures and risks.

In the interim it appears that pressure-treated wood surfaces can be a substantial source of arsenic exposure to children on a frequent basis. Since this exposure pathway can be largely mitigated by use of sealants on the wood at regular intervals (e.g., every 2 years, as legislated in California in 1987), the State of CT has issued a fact sheet informing the public about the potential for children's exposures and the need to seal the wood. The CT fact sheet is available at www.state.ct.us/dph/publications (click on the fact sheet titled "Pesticides Used in Pressure-Treated Wood"). Even though the focus of CPSC deliberations is on CCA-treated wood used in playscapes, the commission should recognize that children's exposure to arsenic from CCA boards is also likely from other types of structures common in a child's environment (decks, treehouses, stair railings, picnic tables, etc.). Therefore, CPSC exposure and risk assessments should consider playscapes in an additive manner with these other sources.

While regular sealing of playscape and decking wood may protect children, this is a chore that many homeowners and town recreation departments do not currently engage in. One issue CPSC should consider in these proceedings is how large a public education campaign is needed to be confident that the wood will be regularly sealed and children adequately protected. Such an effort is needed regardless of whether or not the wood is banned for playscapes, given the number of existing playscapes that contain this type of wood. If CPSC determines that such an education campaign is unlikely to have the effectiveness needed to protect children, then it may want to seriously consider the ban option. If CPSC believes it can be effective, then we would expect that CPSC will become involved by ensuring that the wood is adequately labeled with a sealant message. Further, it would be highly beneficial for CPSC to develop fact sheets and media releases, and provide other outreach efforts. Public education is essential for existing structures as well as new construction. Since alternatives are now available in the marketplace, a description of these alternatives should also be part of the consumer educational process.

Thank you for considering these points. Please feel free to contact us if you need further information.

Gary Ginsberg, Ph.D., Toxicologist
Connecticut Dept. of Public Health
P.O.Box 340398, MS 11CHA
Hartford CT 06134-0308
860-509-7750
gary.ginsberg@po.state.ct.us

David Stilwell, Ph.D.
Connecticut Agricultural Experiment Station
PO Box 1106
New Haven, CT 06504
203-974-8457
david.stilwell@po.state.ct.us

September 11, 2001

Office of the Secretary
U.S. Consumer Product Safety Commission
Room 501
4330 East-West Highway
Bethesda, MD 20814

Dear CPSC Secretary:

This letter and attachment is intended for inclusion in the CPSC docket that is reviewing the proposal to ban chromated-copper-arsenate (CCA) pressure-treated wood in playground equipment. CPSC is to be commended for accepting the task of reviewing this important issue. The data collected in Connecticut and elsewhere points out that arsenic leaches from the wood on an ongoing basis leading to contamination of soil underneath decks/playscapes. This also results in a dislodgeable residue of arsenic on the surface of the wood that is readily available to children from casual contact.

The exposure potential to children from the dislodgeable residue appears to be on the order of 20-200 ug of arsenic on the hands at any point during a play activity, which is a large arsenic source relative to others in a child's environment (see attached ppt presentation). According to a recent risk assessment from a contractor for the wood industry (Gradient, 2001; accessible at preservedwood.com), an estimate of the arsenic concentration in the dislodgeable residue is 1900 ppm. This estimate is similar to calculations of arsenic concentration in the residue based upon the residues found on the wood surface (10-100 ug As/100cm² range across 5 studies) and the low amounts of dust that can be expected on a surface (Hawley, Risk Analy. 5:289-302, 1985). These estimates indicate that the dislodgeable residue is a relatively concentrated source of arsenic, many times higher than soil remediation standards which are aimed to protect children. Another way of looking at the issue is to consider the total amount of arsenic available on an average-sized 8x10 deck. Scaling up from the range of 10-100 ug/100cm² to the surface area of the deck yields 7.5 to 75 mg of arsenic potentially available. Although an average child would not be expected to contact such high mg quantities of arsenic during a single play event, these considerations point out that CCA-treated wood surfaces represent a substantial arsenic source in a child's environment.

There are a number of uncertainties in calculating exposures and risks to arsenic from CCA-treated wood. For example, when extrapolating from what is on the hands of a child to what is actually ingested, there is too little information on children's behaviors (e.g. mouthing frequency; amount of hand entering mouth; mouthing of other objects that may be contaminated; frequency of play activities per year) to know the distribution of exposure and what is a reasonable bounding estimate. Further complicating the assessment are considerations of exposure from contaminated soil underneath the playscape and uptake via the dermal route. Perhaps the upcoming SAP review of this issue for EPA will provide insights into how to better calculate these exposures and risks.

In the interim it appears that pressure-treated wood surfaces can be a substantial source of arsenic exposure to children on a regular or frequent basis. Since this exposure pathway can be largely mitigated by use of sealants on the wood at regular intervals (e.g., every 2 years, as

legislated in California in 1987), the State of CT has issued a fact sheet warning the public about the potential for children's exposures and the need to seal the wood. The CT fact sheet is available at www.state.ct.us/dph/publications (click on the fact sheet titled "Pesticides Used in Pressure-Treated Wood"). Even though the focus of CPSC deliberations is on CCA-treated wood used in playscapes, the commission should recognize that children's exposure to arsenic from CCA boards is likely from other types of structures common in a child's environment (decks, treehouses, stair railings, picnic tables, etc.). Therefore, CPSC exposure and risk assessments should consider playscapes in an additive manner with these other sources.

While regular sealing of playscape and decking wood may protect children, this is a chore that many homeowners and town recreation departments do not currently engage in. One issue CPSC should consider in these proceedings is how large a public education campaign is needed to be confident that the wood will be regularly sealed and children adequately protected. Such an effort is needed regardless of whether or not the wood is banned for playscapes, given the number of existing playscapes that contain this type of wood. If CPSC determines that such an education campaign is unlikely to have the effectiveness needed to protect children, then it may want to seriously consider the ban option. If CPSC believes it can be effective, then we would expect that CPSC will become involved by ensuring that the wood is adequately labeled with a sealant message. Further, it would be highly beneficial for CPSC to develop fact sheets and media releases, and provide other outreach efforts. Public education is essential for existing structures as well as new construction.

Thank you for considering these points. Please feel free to contact me if you need further information.

Gary Ginsberg, Ph.D., Toxicologist
Connecticut Dept. of Public Health
P.O.Box 340398, MS 11CHA
Hartford CT 06134-0308
860-509-7750
gary.ginsberg@po.state.ct.us

Arsenic Exposure Issues from Children's Contact with Pressure-Treated Wood

Gary Ginsberg, Ph.D.
CT Dept. Public Health

David Stilwell
CT Agricultural Expt. Station

September 6, 2001
ASTHO Electronic Seminar

WOOD PRESERVATIVES

- **Extends life of wood**
- **Protects wood from harmful organisms such as termites and fungi**
- **Reduces use of forest products**
- **In trade, potential for harmful environmental effects caused by the preservatives**

CCA IS THE MOST COMMONLY USED WATER-BORNE WOOD PRESERVATIVE

- ◆ **CCA - Chromated Copper Arsenate**
 - ◆ Inorganic As in pentavalent form
- ◆ **ACQ - Ammoniacal Copper Quaternary**
- ◆ **CDDC- Copper**
Dimethyldithiocarbamate
“Kodiak Wood”

Used in decks, fences, playscapes, picnic tables, docks, highway noise barriers, retaining walls

Concentration of Cu, Cr, and As in CCA Treated Wood*

<u>USE</u>	<u>Concentration (ppm- mg/kg)</u>		
	<u>Cr</u>	<u>Cu</u>	<u>As</u>
Above Ground	1997	1178	1792
Ground Contact	3120	1840	2800
Wood Foundation	4742	2797	4256
Marine	19970	11780	17920

* Based on Type C (47.5 CrO₃, 18.5% CuO, 34% As₂O₅)

ENVIRONMENTAL ISSUES ON THE USE OF CCA TREATED WOOD

- Translocation of CCA to Soil and Water via:
 - Leaching of CCA from wood
 - Runoff from lumber yards
 - Sawdust and physical wearing of the wood
- Human exposure to Arsenic in CCA
 - Dislodged from CCA wood surfaces (hand to mouth- children)
 - Exposure during construction (sawdust)
 - Plant uptake
- Impact on Beneficial Marine Organisms
 - Cu and As Toxicity
- Disposal of Old Wood

Cu,Cr, & As in Soil Under CCA-treated Decks

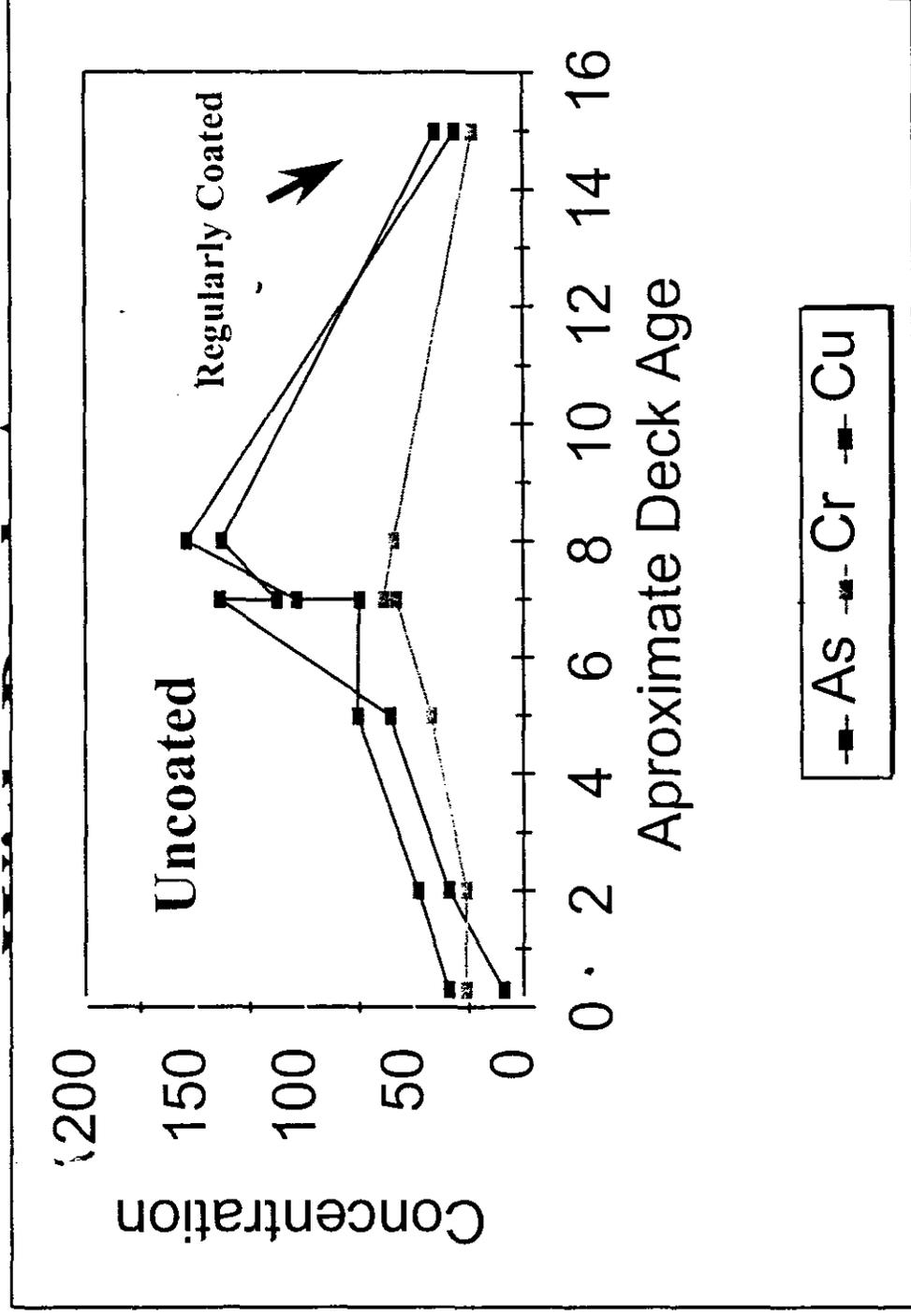
(Stilwell & Gorny, Bull Env.Contam.Tox. 58:22-29, 1997)

- **85 soil samples under 7 decks**
- **All but one of the decks not coated with a paint or stain.**
- **33 control samples -minimum 5 m from deck.**
- **Soil samples -upper 5 cm soil layer**

Arsenic Results in Soils (mg/kg)

<u>Deck #</u>	<u>Beneath Deck</u>	<u>Control</u>
1	9	3
2	34	4
3	61	5
4	139	5
5	113	3
6	138	4
7	40	2
OVERALL	76	3

Average Cu, Cr, and As in Soils Under Decks Tended to Increase



CONCLUSIONS – SOILS UNDER STRUCTURES

- **At each site the average Cu, Cr, and As content were higher in soils beneath the structure than the control soils.**
- **Age Effect noted for soils under decks.**
- **The Cu:Cr:As ratio found is indicative of leaching.**
- **Average As levels greater than State Guidelines.**
- **Contamination likely contained to areas right beneath structures (no evidence of significant horizontal movement of As)**

Estimates of Dislodgeable As on CCA Boards

- Cal Dept Health Services, 1987
 - 5 adult volunteers, hand rub x 5 min
 - playground wood
 - avg. As on hands = 236 ug; max = 1260 ug
 - approx surface area swiped (300 cm²)
 - range of hand coverage = 78 to 420 ug/100cm²
 - gauze wipe samples at park in Berkeley 31-314 ug/100cm²

Cal Dept Health Services, 1987 (cont)

- Cal DHS estimates:
 - high end exposure = 630 ug/visit;
 - mid range exposure = 60 ug/visit;
 - low end exposure = 24 ug/visit
 - based on 50% on hands is ingested and 20-100% of ingested is absorbed
- Uncertainties: sketchy info on how hand rubs done, condition of wood or how many playsets sampled

Estimates of Dislodgeable As on CCA Boards

- CPSC, 1990
 - 7 nylon gauze wipes on new playground wood
 - plus 1 sample from unfinished CCA wood
 - sampling done with dry media
 - 10 swipe passes over the tested area
 - nylon extracted in 0.01N HCl
 - Results: nondetectable ($<6.25\mu\text{g}/100\text{cm}^2$) in 5 samples
 - 22-32 $\mu\text{g}/100\text{cm}^2$ in 2 other samples
 - 69 $\mu\text{g}/100\text{cm}^2$ in one untreated wood sample

Estimates of Dislodgeable As on CCA Boards

- Canadian Data (Galerieau, 1990)
 - 10 outdoor structures sampled
 - dampened gauze swipes - 1 meter sample, 4 per structure
 - mean arsenic on swipe: 43 ug/100cm²
 - (range 0,5 to 322 ug/100cm²)
- SCS 1998 Data cited by Roberts, 2001
 - Swipes from CCA-wood: 4-96 ug/100cm²

Estimates of Dislodgeable As on CCA Boards

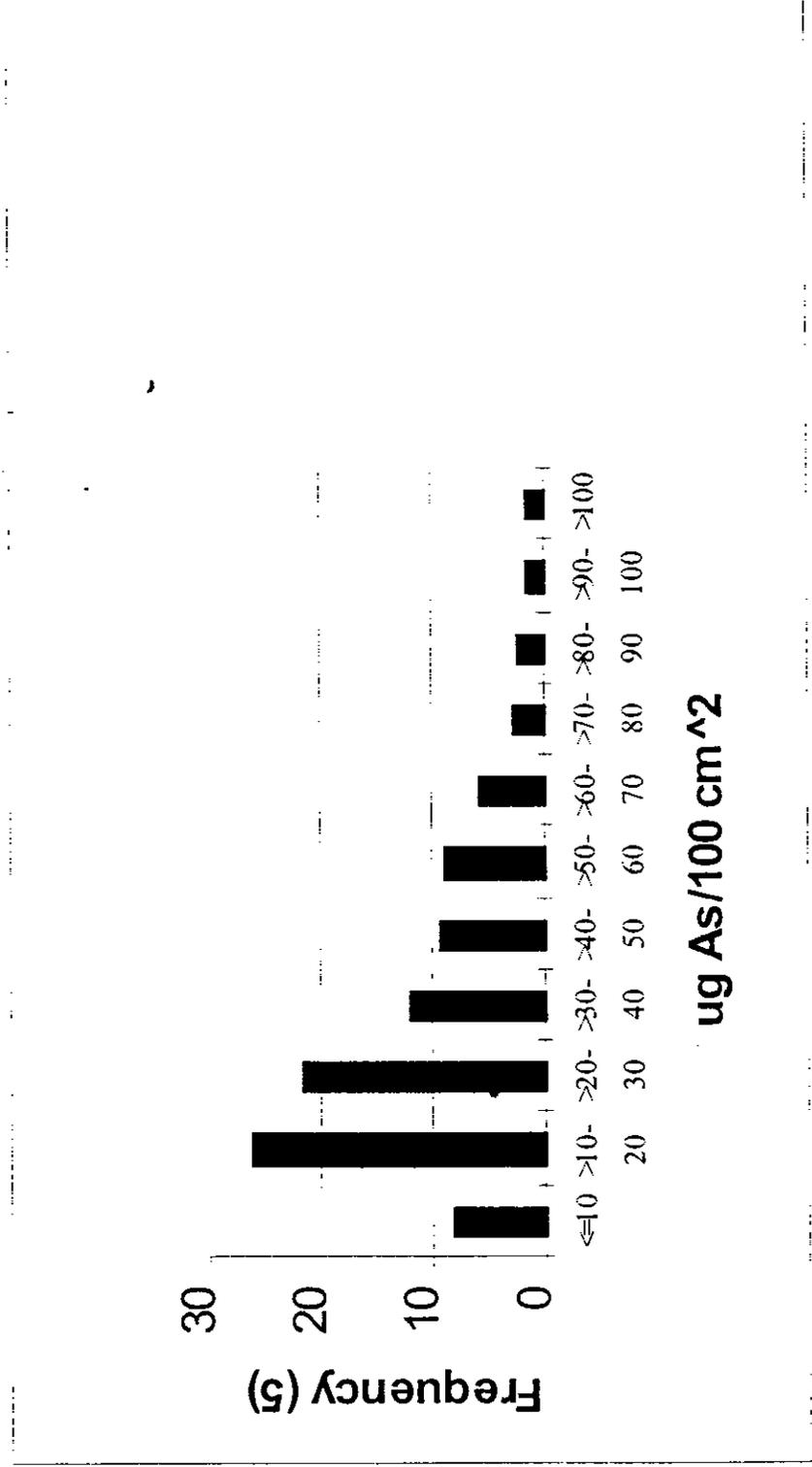
- CT Agricultural Expt. Station(ongoing)
 - New boards and 3 public playscapes sampled
 - Polyester cloth swiped across surfaces
 - Results with new wood
 - 5 - 122 ug/100cm²; avg. = 34 ug/100cm²
 - Results at playscapes
 - Horizontal surfaces - 2-45 ug/100cm²; avg = 8
 - Poles - 15-632 ug/100cm²; avg = 116
 - Poles swiped using hand support rather than wood block

Arsenic Dislodged ($\mu\text{g}/100 \text{ cm}^2$) From Municipal CCA Wood Playscape Surfaces

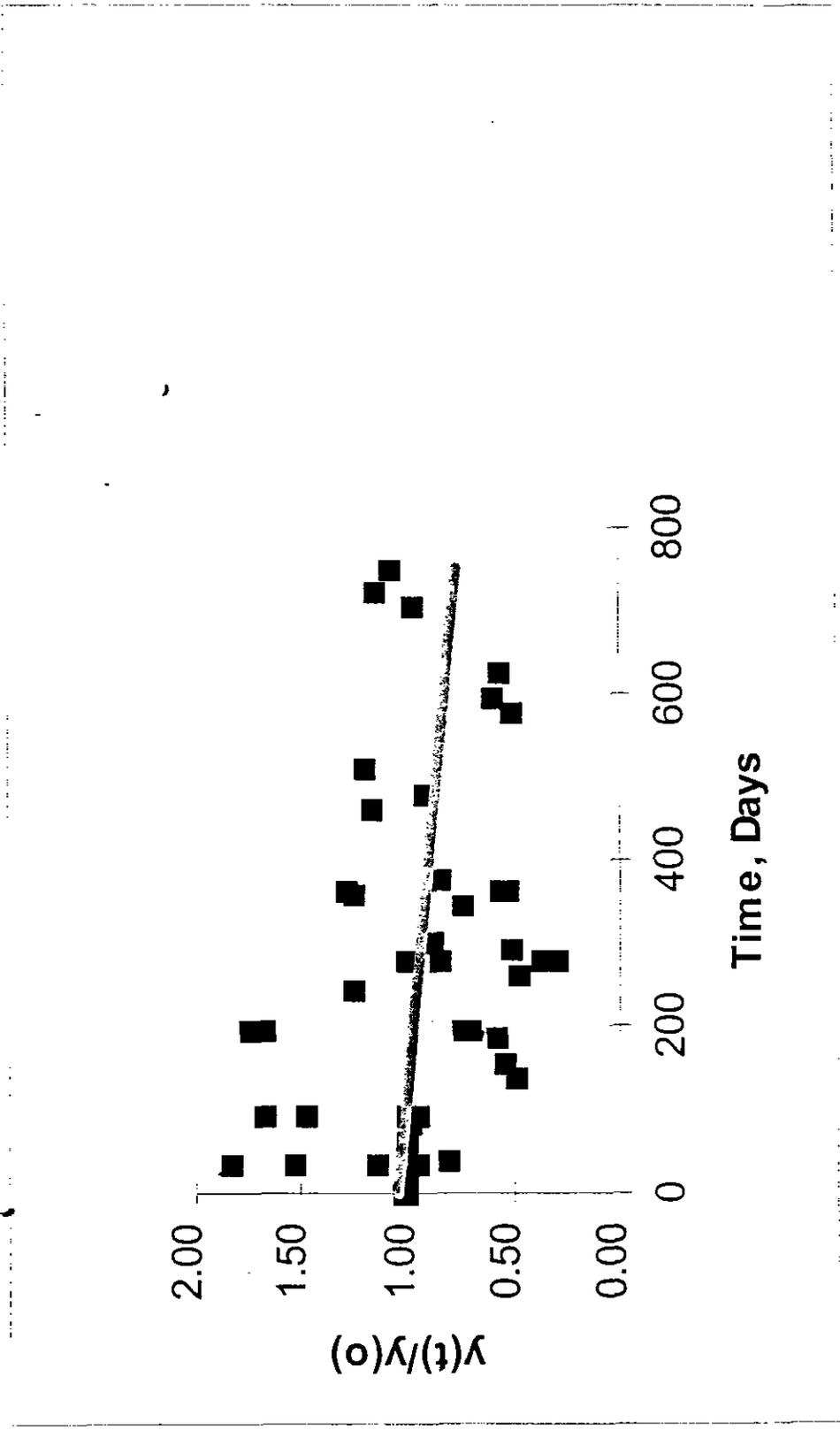
#	<u>Planks(Decking)</u>		<u>Supports (Poles)*</u>	
	n	Range Avg.	n	Range Avg.
1	14	2-45 10.5	3	15-67 36
2	16	2-17 7.8	4	51-632 216
3	15	3-22 8.2	3	21-135 63
Overall		2-45 7.6		15-632 116

Supports > Planks. But Supports Sampled In Different Manner (By Hand, Not Block). Pole Results Should Only be Considered Indicative.

Frequency Distribution of Dislodgeable As on Test Boards



Dislodgeable As on Test Boards Over Time



Summary of Dislodgeable As Estimates

- CalDHS (1987):
 - hand wipes = 78 to 420 ug/100cm²
 - gauze wipes = 31-314 ug/100cm²
- CTA GExpt Station (ongoing):
 - avgs range from 5-122 ug/100cm²
- CPSC (1990):
 - 20-30 ug/100cm² from coated surfaces
 - 69 ug/100cm² from uncoated surfaces
- Canadian Data (1990): 0.5 to 322 ug/100cm²
- SCS (1998): 4-96 ug/100cm²

As Available During Play Activities

- Most dislodgeable values in 10-100 ug/100cm² range
 - although some samples > 200 ug/100cm²
- Assume hands in equilibrium with wood
- Size of child's hand =
 - 200 cm² for two hands (palm side only)
- As on hands during play = **20 to 200 ug**
 - Considerably more if contact with poles

CCA Wood vs. other Inorganic As Sources

- As from diet - 46 ug/d (most as organic As)
- As from water
 - At current DW MCL: 50 ug/d
 - At proposed MCL: 5-10 ug/d
- As from air - 0.6 ug/d
- As from soil at CT cleanup std (10 ppm)
 - If 200 mg soil ingested per day: 2 ug/d

As Exposures from CCA Wood

- Source of 20-200 ug As on hands outweighs other sources in child environment
- Uncertain as to how much As on hands will be ingested
- Roberts and Ochoa (2001) estimate that dislodgeable residue of 10 to 100 ug/100cm² would result in:
 - 7.6 to 76 ug As ingested/day
 - Cancer risk of 4E-05 to 4E-04
 - Hazard Index of 1.4 to 14.

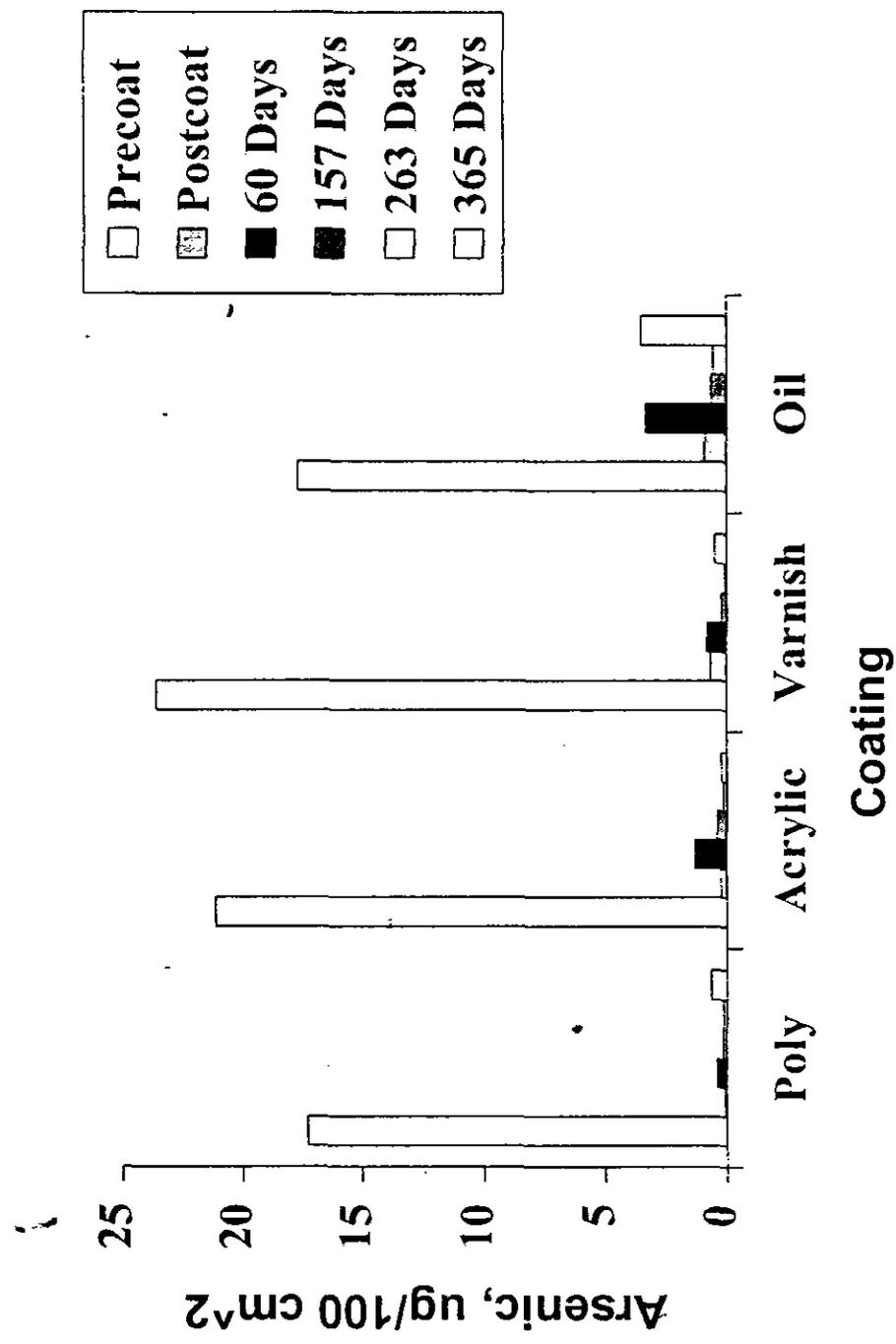
Perspectives on As Exposure from CCA

- While risks somewhat uncertain, exposure potential appears substantial
 - Daily exposure from multiple structures
- Important to minimize children's exposures to extent possible
- Research on sealants critical
 - CTAgExptStation (ongoing)
 - CalDHS (1987)
 - CPSC (1990)

***EFFECTS OF COATINGS ON
ARSENIC DISLODGED FROM THE
SURFACE***

- **The Coatings**
 - Polyurethane floor and deck enamel
 - Acrylic latex deck stain
 - Oil based deck stain (with alkyd resins)
 - Spar varnish- marine and door.
- **Coat top surface of 2x8 CCA boards.**
- **16 Coupons from 4 boards (4 replicates for each coating)**

Average Arsenic Dislodged from Surface Before, and up to One Year After Coating



Coatings Test Conclusions

- **Polyurethane, Acrylic, and Spar: >95% Reduction**
- **Oil Based Finish: 80-97% Reduction, Average=90%**
- **Application of these coatings eliminated dislodgeable arsenic for 1 year test.**
- **However, test did not determine how well these coatings would stand up to wear and tear.**
 - **Consult with paint dealer.**
 - **See Consumer Reports (June 1998) “Exterior Deck Treatments Test”**

Sealant Trials in California (1987)

Dislodgeable As/100cm²

- Pier samples: 1,131 ug
- Pier after polyU: <10 ug
- Pier after polyU
-2 yrs later: 12-65 ug
- Berkeley playscape: 31-314 ug
- Playscape after oil stain: 1-13 ug
- CalDHS recommendation to seal public play equipment every 2 years

Stevenson, Todd A.

From: Gary Ginsberg [gary.ginsberg@po.state.ct.us]
Sent: Monday, September 10, 2001 4:49 PM
To: cpsc-os@cpsc.gov
Cc: David.Stilwell@po.state.ct.us
Subject: Petition HP 01-3



CCA701.PPT



ARSENICCPSC.DOC

Please ignore my earlier e-mail today; I attached the wrong file. Please put the attached 2 files on the docket. The first file represents my comments, together with Dr. David Stilwell. The 2nd file provides supporting documentation; it is a powerpoint presentation that I gave on an ASTHO teleconference this past week. Sorry for the confusion and thank you very much. Gary Ginsberg