chemicalWATCH Factsheet

Chromated Copper Arsenate (CCA) Treated Wood -

Chromated copper arsenate (CCA) is an inorganic arsenical used as a wood preservative. In February 2002, the Environmental Protection Agency (EPA) announced a voluntary phase-out by industry of most residential uses of this arsenic-based wood preservative. The agreement states that after January 2004 CCAtreated wood can no longer be manufactured and sold for decks and patios, picnic tables, playground equipment, walkways/boardwalks, landscaping timbers, or fencing. However, already existing residential CCA-treated structures may continue to be used.

In 1988, when EPA decided to cancel most non-wood uses of the inorganic arsenicals, the agency stated its concern about oncogenicity (cancer), mutagenicity (genetic damage), teratogenicity (birth defects) and acute toxicity. EPA noted that its Office of Health and Environmental Assessment (OHEA) had prepared a report reviewing the existing scientific literature, including "[h]uman epidemiology studies ... [which] provided the most persuasive evidence linking exposure to inorganic arsenic to an increase in cancer in humans."1 When EPA finally canceled the last non-wood use of the inorganic arsenicals in 1993, it determined it would not modify its earlier risk/benefit assessment.

Routes of Exposure

One of the most important factors in determining the hazard of a toxic chemical is an evaluation of actual exposure to the chemical. In the case of CCA, chances of exposure are heightened since the chemical's use as a wood preservative is so widespread. Exposure through ingestion and inhalation, and to a lesser extent skin absorption, pose risks to both human health and the environment.²

Children face especially high risks from exposure since they take in more pesticides relative to body weight than adults. In addition, children have developing organ systems that are more vulnerable and less able to detoxify toxic chemicals.³ The Minnesota Arsenic Study (MARS), conducted by the Minnesota Department of Health (MDH), finds that

children accumulate arsenic at a higher rate then adults.⁴ Furthermore, the probability of an effect such as cancer, which requires a period of time to develop after exposure, is enhanced if exposure occurs early in life.5 Exposure to CCA is heightened by hand-to-mouth behavior, which is well-documented among children. According to the October, 2001 EPA Scientific Advisory Panel (SAP) recommendation, children have an average of 9.5 hand-to-mouth activities per hour for an average of 1-3 hours of play activity.6 This number has the potential to grossly underestimate the true exposure to active children and to high-risk groups predisposed to increased rates of handto-mouth behavior, such as children with Down syndrome (DS).

Exposure Through Direct Contact with Wood: The arsenic in CCA-treated wood can be dislodged so that direct contact with wood can lead to exposure. The amount dislodged depends on age and use of the wood, according to the Connecticut Agricultural Experiment Station. Wipe tests done on the horizontal surfaces of three municipal play structures found an average of 8.8 μ g/100 cm² arsenic dislodged from the wood. This was less than the average from new wood (40 μ g/100 cm²).⁷ Tests done on the vertical support beams found higher levels of arsenic compared to the horizontal surfaces and the new samples, showing levels as high as $632 \mu g/100 \text{ cm}^2$ and averaging 105 μ g/100 cm². A study conducted by the Environmental Working Group examined samples wiped from CCA-treated wood surfaces, collected from an area about size of a four-year old child's hands. In one quarter of the samples, the amount of arsenic wiped off the surface was at least three times the 10 micrograms EPA drinking water limit. Some samples showed up to 250 micrograms of arsenic.8 This arsenic can be ingested into the bodies of children participating in typical handto-mouth behavior or eating. Exposure to arsenic can dramatically increase with normal contact to decks, play sets and other wood treated with CCA.

Exposure Through Contaminated Soil: Leaching of CCA from wood into surrounding soil is well documented. Arsenic and chromium (VI) have been found to leach in substantial quantities from CCA treated wood. Carcinogenic hexavalent chromium (Cr VI) is known to account for up to 50% of the total chromium in CCA. This chromium is known to leach out of CCA treated wood. A 1997 report by Stilwell and Gorny found that soil around CCA treated wood structures ranging in age from four months to 15 years contained an overall average chromium concentration of 43 mg/kg (ppm), compared to 20 mg/kg (ppm) for the control soils. The same study found that the overall average arsenic concentration underneath treated decks was 76 mg/kg, compared to only 3.7 mg/kg for the control samples.⁹ In addition, a community group in Ithaca, NY found soil samples under a CCA treated playset with levels of arsenic up to 101 parts per million (ppm), over ten times the New York state clean-up standard of 7.5 ppm.10 The Connecticut Agricultural Experiment Station reported arsenic levels averaging 76 ppm under CCA treated decks, compared to an average level of 3.7 ppm of arsenic in control soils.¹¹

The Federal Insecticide, Fungicide and Rodenticide Act's (FIFRA) Scientific Advisory Panel (SAP), in October 2002, used a 25% availability factor for arsenic of consumed soils.¹² This factor does not take into account differing soil types. Testing for additional risks of contaminated soil with differing organic matter and pH levels is inadequate.

Exposure through Incomplete Fixation:

Pressure treated wood frequently fails to be fully dried before leaving production facilities. This can cause the CCA preservative to not fully fix to the wood. Workers have been exposed to massive quantities of arsenic, chromium, and copper causing skin irritation, and increasing risk of chronic health effects.

Exposure Through Food Grown In Contami-

nated Soil: Further exposure to CCA can occur by eating food raised in a garden containing CCA-treated wood. Research that examined lettuce in gardens with CCA-treated wood blocks showed the lettuce's arsenic uptake was

Beyond Pesticides

701 E Street, S.E., Suite 200 • Washington DC 20003 202-543-5450 (v) • 202-543-4791 (f) info@beyondpesticides.org • www.beyondpesticides.org more then 1.7 parts per million (ppm) of arsenic by dry weight.¹³ These levels alone may not cause acute poisoning, but when considered as an additive to other exposures, they are far from negligible.

Exposure Through Inhalation: Dust on a CCA-treated structure, or from the soil below, may be contaminated and contribute to total exposure to CCA if inhaled. In dry climates, dust is easily kicked up and inhaled. Even wet climates can have significant dust and may have higher contaminated levels in the soil due to past leaching. Most troubling is the possibility of inhaling sawdust or fly ash. Although these risks may not be present in normal use, its occurrence can have acutely toxic effects and dramatically increase any long term CCA (or its constituents) body burden. Children's increased respiration rates can dramatically exacerbate these exposures.

Acute Health Effects

Toxic effects of CCA can come from any of the chemical constituents, but most of the focus has been on arsenic. Most acute effects can be seen after inhalation or ingestion of arsenic or arsenic contaminated substrates. Symptoms of acute arsenic toxicity include pain, eye irritation, nausea, vomiting, and diarrhea, characteristic skin lesions, decreased production of red and white blood cells, abnormal heart function, blood vessel damage, liver and/ or kidney damage, and impaired nerve function causing a "pins-and-needles" feeling.14 In cases of extreme exposure, arsenic is fatal; a lethal dose can be a little as 1 to 25 mg arsenic per kg of body weight.15 Symptoms of acute poisoning from chromium (IV) include severe redness and swelling of the skin.16

Chronic Health Effects

Chronic effects of arsenic exposure have been seen in many body systems. Although some health effects are exposure specific, most are systemic and can result from any root. Arsenic poisoning damages mucus membranes, and it produces peripheral nervous system disturbances and degeneration and hearing loss.¹⁷ In addition, research links exposure with immune system suppression, leaving victims more vulnerable to other ailments.¹⁸ Studies on rats show increased fetal mortality, cleft pallet and increased ratio of male to female offspring.¹⁹

Children face particular risk from exposure to arsenic. A Thailand Health Research Institute study showed an inverse relationship between the levels of arsenic found in children's hair and their height. This relationship was significant for both high and low arsenic accumulations. This study represents defining data on lowlevel arsenic exposure's effect on the growth of children.²⁰

Chromium also poses long-term health threats. Studies of chromium (VI) from industrial emissions have found it to be highly toxic due to strong oxidation characteristics and ready membrane permeability.²¹ Cr (VI) has been known to cause damage to kidneys and liver. Skin contact with certain chromium (VI) compounds can cause skin ulcers. In addition, birth defects have been observed in animals exposed to chromium (VI).²²

Carcinogenic Effects

EPA Carcinogen Assessment Group classified inorganic arsenic as a Group A carcinogen. Arsenic ingestion and inhalation has been reported to increase the risk of cancer, especially in the liver, bladder, kidney and lung.²³ The form of chromium (hexavalent) found in CCA has also been found by EPA to be a known human carcinogen. An EPA "Product Matrix" on "Wood Preservatives" states that "inorganic arsenic compounds have been shown to cause cancer in humans."²⁴

Neurological Effects

Low dose neurological effects are well documented with arsenic exposure. Although

past studies have concluded that neurological function was not impaired below 1000 ppb, a recent EPA study found that vibrotacitle and pin-prick sensitivity were affected at levels as low as 300 ppb in drinking water.²⁵ EPA states that "there is a large body of epidemiology studies and case reports which describe neurotoxicity in humans after both acute and chronic exposures..."²⁶

Ecological Effects

In addition to human health effects, the arsenicals found in CCA pose grave ecological threats. Many aquatic organisms are extremely sensitive to arsenic exposure, which can result in serious health effects and even death at relatively low levels. Arsenic bioconcentrates in aquatic organisms - in fresh water organisms up to 17 times background levels, and in marine oysters 350 times background levels.²⁷ Because of bioaccumulation, low levels of arsenic pose devastating threats to larger animals including top predators that eat organisms exposed to arsenic. Studies have shown that quantities of arsenic that can leach from CCA treated wood are high enough to bioaccumulate. This is especially true in soils and water with slightly acidic pH.28

The copper in CCA can be toxic to aquatic life as well. The LC50 for aquatic invertebrates and fish ranges from 5 micrograms (μ g) per liter to 100,000 μ g /l.²⁹ Effects on aquatic invertebrates include decreased feeding and egg production and impairment of certain behaviors, such as the ability of clams to burrow.³⁰ In addition, fish growth, spawning and survival are all affected by the presence of copper. Salmon have been known to head back downstream without spawning due to high copper concentration. Gill lesions, kidney damage, and diabetes-like symptoms in a variety of fish species were also observed in association with copper concentrations.³¹

Chromated Copper Arsenate (CCA) chemicalWATCH Factsheet Bibliography

¹ Environmental Protection Agency, June 30, 1988. Federal Register 24787-89, volume 53.

² ATSDR, 1989. "Arsenic Public Health Statement March 1989." Url: *http:// atsdr1.atsdr.cdc.gov:8080/ToxProfiles/ phs8802.html*

³ US EPA, Office of the Administrator. September 1996. "Environmental Health Threats to Children." *EPA 175-F-96-001.* ⁴ Minnesota Department of Health (MDH), 2001. "The Minnesota Arsenic Study (MARS)" *Url: http://www.health.state.mn.us/divs/ eh/hazardous/arsenicstudy.pdf*

⁵ Vasselinovitch, S., et al., 1979. "Neoplastic Response of Mouse Tissues During Perinatal Age Periods and Its Significance in Chemical Carcinogensis," *Perinatal Carcinogenesis, National Cancer Institute Monograph 51*. ⁶ FIFRA Scientific Advisory Panel. 2001.

"Final Expo Document October 23-25," http://www.epa.gov/scipoly/sap/2001/october/final_expo_doc_927.pdf, p. 15

⁷ Stilwell, David E., 1998. "Environmental Issues On The Use Of CCA Treated Wood." Department of Analytical Chemistry, The Connecticut Agricultural Experiment Station, http://www.caes.state.ct.us/FactSheetFiles/

Beyond Pesticides 701 E Street, S.E., Suite 200 • Washington DC 20003 202-543-5450 (v) • 202-543-4791 (f) info@beyondpesticides.org • www.beyondpesticides.org

Chromated Copper Arsenate (CCA) chemicalWATCH Factsheet Bibliography

AnalyticalChemistry/fsAC001f.htm

⁸ Gray, Sean and Jane Houlihan, August 2002. "All Hands on Deck." Environmental Working Group, http://www.ewg.org/reports/ allhandsondeck AllHandsOnDeck.pdf.

⁹ Stillwell, D., and K. Gorny, 1997. "Contamination of soils with copper, chromium, and arsenic under decks built from pressure treated wood." *Bulletin of Environmental Contamination Toxicology*, 58(22-29).

¹⁰ Steingraber, S, "Arsenic and Old Spaces." *Pesticides and You*, Winter 2002-03, Reprinted Op-ed

¹¹ Stillwell, D., and Gorny, K, 1997. "Contamination of soils with copper, chromium, and arsenic under decks built from pressure treated wood." *Bulletin of Environmental Contamination Toxicology*, 58(22-29).

¹²FIFRA Scientific Advisory Panel, 2001. "Final Expo Document October 23-25." *http:// www.epa.gov/scipoly/sap/2001/october/ final_expo_doc_927.pdf*, p. 15

¹³ Stilwell, David, 1999. "Arsenic in Pressure Treated Wood." Department of Analytical Chemistry, The Connecticut Agricultural Experiment Station.

¹⁴ ATSDR, 1989. "Arsenic Public Health Statement, March 1989" *Url: http:// atsdr1.atsdr.cdc.gov:8080/ToxProfiles/ phs8802.html*

¹⁵ World Health Organization, 1981. "Arsenic, Environmental Health Criteria 18". Geneva, UNEP/ILO/WHO. Cited in: Caroline Cox,1991. "Chromated Copper Arsenate" *Journal of Pesticide Reform*, 11(1):23-27, Spring 1991, NCAP, Eugene, OR.

¹⁶ Agency for Toxic Subsistances and Disease Registry, U.S. Center for Disease Control, *http://www.atsdr.cdc.gov/tfacts7.html*

¹⁷ World Health Organization, 1981. "Arsenic, Environmental Health Criteria 18" Geneva, UNEP/ILO/WHO. Cited in: Caroline Cox, 1991. "Chromated Copper Arsenate," *Journal of Pesticide Reform.* 11(1):23-27, Spring 1991, NCAP, Eugene, OR

¹⁸ National Research Council, Committee on Medical and Biologic Effects of Environmental Pollutants, 1977. "Arsenic." Washington, DC: National Academy of Sciences; World Health Organization, 1981. Arsenic, Environmental Health Criteria 18. Geneva, UNEP/ILO/ WHO. Cited in: Cox, 1991.

¹⁹ Environmental Protection Agency. January 1981. "Creosote, Inorganic Arsenicals, Pentachlorophenol." *Position Docket No. 2/3.*

²⁰ Siripitayakunlit, Unchalee, Amara Thonghong, Mandhana Pradipasen, 2000, "Growth of Children with Different Arsenic Accumulation, Thailand," University of Denver Poster, financed by the Thailand Health Research Institute, National Health Foundation

²¹ Hazardous Substance Data Bank (HSDB), National Library of Medicine Specialized Information Service *http:// toxnet.nlm.nih.gov/cgi-bin/sis/search*

²² Agency for Toxic Substances and Disease Registry, U.S. Center for Disease Control, *http://www.atsdr.cdc.gov/tfacts7.html*

²³ ATSDR, 1989. "Arsenic Public Health Statement, March 1989." url: http:// atsdr1.atsdr.cdc.gov:8080/ToxProfiles/ phs8802.html

²⁴ Environmental Protection Agency. Copyright, 1996 by Purdue Research Foundation, West Lafayette, Indiana 47907. *Url: http:// www.epa.gov/grtlakes/seahome/ housewaste/house/woodpre.htm*.

²⁵ Mumford, Judy, PhD, Yajuan Xia, Mike Schmitt, Richard Kwok, Zhiyi Liu, Rebecca Calderon, David Otto, "Health Effects from Chronic Exposure to Arsenic via Drinking Water in Inner Mongolia." EPA Human Studies Facility, Research Triangle Park, NC.

²⁶ Timothy F. McMahon, Ph.D. and Jonathan Chen, Ph.D., Environmental Protection Agency. Septermber 25, 2001. "Hazard Identification and Toxicology Endpoint Selection for Inorganic Arsenic and Inorganic Chromium." FIFRA SAP Background Document. Url: http://www.epa.gov/oscpmont/ sap/2001/october/ hazard_final_document.pdf

 $^{\ensuremath{ \mathrm{ 27}}}$ ATSDR, 1993. "Toxicological Profile for Arsenic."

²⁸ ATSDR, 1993. "Toxicological Profile for Arsenic." April 1993, ATSDR Washington, DC.

²⁹ Hodson, Peter V., Uwe Borgmann, and



701 E Street, S.E., Suite 200 • Washington DC 20003 202-543-5450 (v) • 202-543-4791 (f) info@beyondpesticides.org • www.beyondpesticides.org

Harvey Shear. 1979. "Toxicity of copper to aquatic biota." *In* Nriagu, Jerome O. (ed.) *Copper in the environment. Part II: Health effects,* pp. 308-372. New York, NY: John Wiley and Sons.

³⁰ Ibid

³¹ Ibid