Chromated Copper Arsenate

By Caroline Cox

Chromated copper arsenate (CCA) is a mixture of metallic salts used as a wood preservative. As the name suggests, these salts contain arsenic, copper, and chromium. They are used to protect wood from decay by microbes, fungi, and wood-feeding insects.

Typical uses include treatment of fenceposts, decking, playground equipment, and structural lumber used where it will be in contact with concrete or the ground.

CCA is generally applied to wood by pressure treatment. Instead of applying the chemical to the surface of the wood, the wood is submerged in a cylindrical tank containing the preservative that is then subjected to high pressure to force the CCA into the cells of the wood. Common brand names of CCA formulations are Osmose, Wolman, and Rentokil.[1]

Unlike most synthetic pesticides, CCA is not an organic compound; its molecular structure does not contain carbon.

Various mixtures of salts can be used to make CCA so that its precise molecular composition may vary from formulation to formulation. However, most currently used formulations are CCA Type C which is a mixture of arsenic pentoxide (34 percent), chromic acid (47.5 percent), and cupric oxide (18.5 percent).[2]

The original type of CCA (CCA-A) began to be used in the United States in 1938. CCA-C was first introduced in 1968 and has become commonly used because it is more tightly retained by treated wood than the earlier types of CCA.[2]

Many metals can have more than one valence, a term used to describe the number of bonds one atom can form with other atoms. Arsenic usually is either trivalent, arsenic(III), or pentavalent, arsenic(V). Arsenic pentoxide, often used in CCA, contains arsenic(V). Chromium is usually either trivalent, chromium(III), or hexavalent, chromium(VI). CCA contains mostly chromium(VI).

Caroline Cox is JPR's editor.

Toxicology: Arsenic

Arsenic is widespread in the environment at low concentrations. For humans, the average daily intake is a few micrograms. Some seafood contains relatively large amounts (several milligrams) of arsenic per serving, mostly as relatively low toxicity organic compounds.

The lethal oral dose of arsenic for an adult human is between 1 and 2.5 milligrams per kilogram (mg/kg) of body weight. Symptoms of arsenic poisoning include damage to the digestive system, vomiting diarrhea, muscle cramps, facial swelling, and shock.[7]

> *"A high incidence of herpes infections and respiratory infections has been found in cases of subacute arsenic poisoning, suggesting that arsenic may suppress the immune system."*

Chronic effects include damage to mucous membranes; irritation of eyes; irritation, darkening and lesions of the skin; disturbances and degeneration of the peripheral nervous system; swelling and damage of the liver; abnormal heart function; and hearing loss.[7]

A high incidence of herpes infections and respiratory infections has been found in cases of subacute arsenic poisoning, suggesting that arsenic may suppress the immune system.[4] In experiments with virus-infected mice, large doses of arsenicals resulted in higher mortality from three different diseases.[7]

Exposure to arsenic has been associated with induction of cancer since the nineteenth century. Exposure to arsenic increased the risk of lung cancer in epidemiological studies of workers who manufactured or used arsenic-containing pesticides and workers in smelters who were exposed to high levels of arsenic. Exposed workers' risk of cancer is from two to 1000 times the risk of unexposed workers. Lung cancer risk increases with increasing exposure to arsenic. (See Figure 1.)[7]

Skin cancer has been associated with arsenic exposure in many parts of the world, and has been studied epidemiologically in a region of Taiwan where well water has high levels of arsenic. The incidence of skin cancer increased with increasing consumption of arsenic. (See Figure 2.)[7]

The U.S. Environmental Protection Agency (EPA) classifies arsenic as a Group A carcinogen, having evidence of human carcinogenicity.[8]

A small study of 62 patients, half of whom had previous arsenic exposure through medical use or through pesticide use, found that exposure to arsenic increased the frequency of abnormalities in the chromosomes of exposed subjects. Similar results were found in another small study of Swedish smelter workers who had been exposed to arsenic. A subsequent study of spontaneous abortion rates in workers employed at the same smelter found the rates were higher than in a reference population living more than 50 kilometers from the plant.[4]

Animal and bacteria tests for mutagenicity (the ability to cause genetic change) have given conflicting results, while injections of arsenic in golden hamsters, mice, and rats increased fetal mortality and birth defects. One study showed that three generations of low arsenic exposure increased the ratio of males to females in mice.[7] EPA is requiring further animal tests for reproductive effects.[8]

Toxicology: Chromium

The lethal oral dose of chromium(VI) for adult humans is 50 mg/kg of body weight. Clinical features

of acute chromium toxicity are vomiting, diarrhea, blood loss into the digestive tract, and cardiovascular shock followed by liver and kidney necrosis.[5]

Chronic toxic effects of chromium (VI) include irritation of the skin (chrome ulcers) and mucous membranes, sensitization of skin, irritation and corrosion of the lungs, and lung cancer.[5]

The first report that lung cancer might be related to chromium exposure was published in 1932. Since then the association has been confirmed by epidemiological studies of workers in chromium-using industries in the U.S., Japan, Germany, and the Soviet Union. For exposed workers, the risks of lung cancer as calculated in these studies was from 1.6 to 1510 times greater than the risk to unexposed workers. These cancer risks have generally declined as manufacturing processes have improved.[5] EPA has classified chromium as a Group A carcinogen.[8]

Bacterial tests, tests using mammalian cell cultures, and tests using isolated DNA (the molecule that stores and transmits genetic information) have shown that chromium(VI) causes changes to genetic material. In tests with golden hamsters, chickens, and mice, injections of chromium(VI) caused a number of birth defects.[5] EPA's assessment of these studies is that chromium(VI) has demonstrated the potential to cause birth defects and fetal mortality and that further tests are necessary using a route of exposure other than injection that is appropriate for human risk assessment.[8]

Mode of Action

Arsenic: Arsenic toxicity to living organisms results at a molecular level because it mimics phosphate ions. High energy bonds between phosphate ions are an important part of the respiratory process (the process by which a cell obtains energy from sugar compounds) in all higher organisms. If arsenic takes the place of phosphate in these high energy compounds they rapidly break down instead of performing their usual function.[3] Arsenic(III) is more toxic than arsenic(V) and there is some evidence that arsenic(V) is converted into arsenic(III) in living organisms.[4]

An indication of arsenic's broad-spectrum toxicity is the use of arsenic-containing compounds as insecticides, rodenticides, herbicides, and plant growth regulators.[3]

Chromium: Chromium(III) is thought not to produce toxic effects and is an essential nutrient in humans in amounts of 50-200 micrograms (μg) per day. Chromium(VI) is almost totally derived from human activities and is toxic because it is a strong oxidizing agent to which biological membranes are easily permeable.[5]

Copper: Copper inhibits some of the enzymes involved in respiration, photosynthesis, and nitrogen fixation. It also interferes with osmoregulation, the process by which an organism maintains an appropriate water balance. Copper is most toxic to organisms which concentrate copper, have high surface to volume ratios, or have permeable gill surfaces that facilitate rapid uptake of the metal.[6] For example, fungal spores concentrate copper to a level up to one hundred times that found in their immediate environment[3] and algae, in addition to having a high surface to volume ratio, accumulate up to 83,000 times the ambient copper concentration.[6]

Toxicology: Copper

Trace amounts of copper are required in the synthesis of hemoglobin and several human enzymes.[9] It is an essential nutrient and humans have "a natural, efficient homeostatic mechanism for regulating the body levels of copper ions over a wide range of dietary intake.... The adult human body burden of copper is typically 80 to 150 mg."[10] If copper in excess of this amount is ingested, it is excreted. If very large doses are consumed and homeostatic mechanisms break down, signs of acute toxicity include vomiting, diarrhea, jaundice, and urinary problems.[9]

Figure 1
Relationship Between Arsenic Exposure of Smelter Workers and Respiratory Cancer Deaths

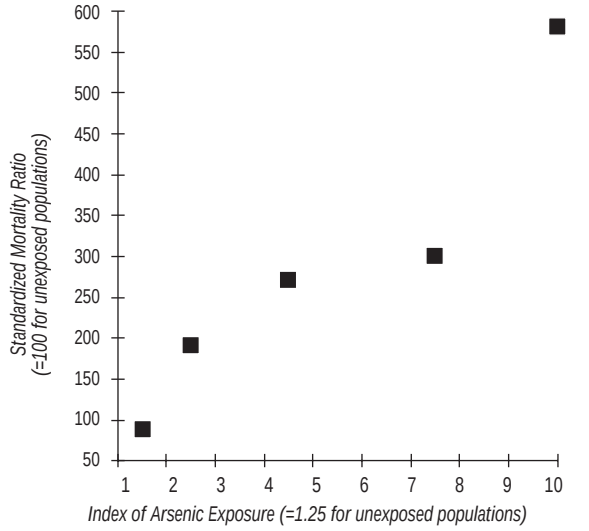
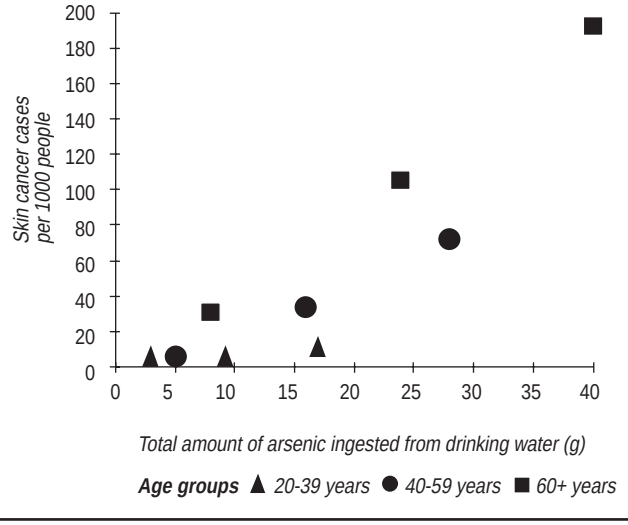


Figure 2
Prevalence of Skin Cancer in Relationship to Arsenic Ingestion



There are a few reports of green hair coloration being caused by occupational exposure to copper or drinking water with high copper levels.[9]

However, exposure to copper by inhaling copper dust appears to be more hazardous to health. Workers exposed to copper dust have reported symptoms resembling a common cold, including congestion and fever. In one instance these effects were reported in a factory where copper dust was less than one-tenth of the federal standard for copper dust. When new ventilation equipment was installed that reduced dust levels to .008 milligrams per cubic meter, the symptoms subsided.[9]

In Portugal, vineyard workers spraying copper sulfate 15 to 100 days per year developed lung lesions, some of which later became cancerous. In addition, liver damage, sometimes progressing to liver cancer, was noted.[9]

Impacts on Nontarget Organisms: Arsenic

A review of acute LC_{50}s* for various freshwater and marine fish and shellfish contained values that varied by several orders of magnitude. Bluegill appeared to be the most sensitive fish species (the lowest 48-hour LC_{50} was .5 ppm) while Channel catfish were relatively tolerant (the 24-hour LC_{50} was 47.9 ppm). Values for other species were between these extremes. Terrestrial wildlife, particularly birds, appears to be more tolerant of arsenic: the LD_{50}* for mallard ducks was estimated at over 300 mg/kg. Arsenic acid has caused deer kills following its use as an herbicide on farmland.[4]

As is generally true, chronic effects of arsenic exposure occur at lower doses than does acute toxicity. Reproductive impairment of the water flea, *Daphnia magna*, occurred at doses .07 times lower than the acute oral LD_{50} and weight loss occurred at .14 times the LD_{50}.[11]

Impacts on Nontarget Organisms: Chromium

Acute toxicity for most microorganisms ranges between .05 and 5 mg/kg of medium in which the organisms are living. Chromium is acutely toxic to soybean plants raised hydroponically if present at 30-60 milligrams per liter (mg/l) of nutrient solution. In general, aquatic invertebrates are more susceptible to chromium than are fish. LC_{50}s in EPA tests for aquatic invertebrates range from .066 to 64.0 mg/l of chromium(VI) while LC_{50}s for fish range from 17.6 to 249 mg/l. [5]

Chronic effects of chromium exposure have also been documented. Hydroponic soybeans showed a reduction in leaf weight and leaf chlorophyll concentration following chromium exposure 1/3000-1/6000 times that which caused mortality.[5] Exposure of *Daphnia magna*, the water flea, to chromium at 1/5 of the lethal dose inhibited reproduction and shortened lifespan.[11] Chromium has also been observed to increase the susceptibility of fish to infection.[5]

Impacts on Nontarget Organisms:

> *"For example, 1 µg/l of copper was sufficient to cause mortality of 6 percent of juvenile trout and 5 µg/l caused 6 to 13 percent mortality of five species of toads and frogs. Defects in juvenile trout occurred at concentrations between 1 and 5 µg/l."*

Copper

Acute toxicity (48-hour LC_{50}s) of copper to aquatic invertebrates ranges from 5 micrograms (µg) of copper per liter(l) to over 100,000 µg/l. Invertebrates with shells (for example, molluscs) or hard exoskeletons (marine arthropods, for example) are more tolerant of copper exposure than less well protected invertebrates. A similar range in acute toxicities is found in fish.[6]

Chronic effects of copper exposure on algae and aquatic invertebrates have been studied in detail. In algae, some depression of photosynthesis has been demonstrated at copper concentrations as low as 1 to 2 µg/l. Growth is inhibited at concentrations between 50 and 60 µg/l, and nitrogen fixation is reduced by concentrations of 5 µg/l. Decreased feeding and egg production by invertebrates has been found in concentrations as low as 5 µg/l and decreased growth at concentrations as low as 13 µg/l. Copper is also known to influence invertebrate behavior. For example, the burrowing activity of clams was impaired at 10 µg/l.[6]

In fish, spawning, growth, and survival are affected at copper concentrations between 5 and 40 µg/l in water low in organic matter and at higher concentrations (between 66 and 120 µg) in river water enriched with organic matter. Salmonids, channel catfish, and walleyes were the most sensitive fish species tested. Water hardness influenced toxicity. High copper concentrations have also caused salmon to return downstream from spawning areas without having spawned; inhibited oxygen uptake in bluegills; and caused a number of tissue and blood changes (including gill lesions, kidney damage, and diabetes-like symptoms) in a variety of fish species. Copper concentrations necessary to cause these responses varied from 50 to 300 µg/l.[6]

Copper appears to be more toxic to embryonic and juvenile stages of aquatic vertebrates than to adult stages. For example, 1 µg/l of copper was sufficient to cause mortality of 6 percent of juvenile trout and 5 µg/l caused 6 to 13 percent mortality of five species of toads and frogs. Defects in juvenile trout occurred at concentrations between 1 and 5 µg/l.[12]

Impacts on Nontarget Organisms: CCA

A recent Canadian study found that the acute toxicity of CCA to *Daphnia magna* and to a species of algae, *Selenastrum capricornatum*, was greater than might be predicted from the toxicity of copper, although copper was the most toxic of CCA's three metals when the test organisms were exposed to the metals individually. This suggests that the metals act jointly to cause toxicity. Additional experiments using the metals in pairs indicated that copper and chromium

*LC_{50} is the concentration in water of a chemical that will kill 50 percent of a population of test aquatic animals. LD_{50} is the amount of a compound that will kill 50 percent of a population of test animals.

have a synergistic toxic effect.[11]

Impacts on Water Quality

The Canadian study just mentioned also tested the rate at which CCA leached from treated wood when submerged in water of varying acidity (pH). The study showed that leaching was highly dependent on the acidity of the water when citric acid was used to adjust acidity. In acidic water (pH 3.5) 68 percent of the arsenic, 53 percent of the chromium, and all of the copper leached out during a forty-day period. If the water was neutral (pH 7.0) only 9 percent of the arsenic, 1 percent of the chromium, and less than 1 percent of the copper leached. Lower levels of leaching, but with a similar pattern, were found when acidity was adjusted with hydrochloric acid. These results suggest that CCA treated wood exposed to organic acids (in swampy ground or soil with high humus levels, for example) will leach readily.[11]

Another Canadian study found that runoff from a CCA treatment plant contained high concentrations of arsenic(III) (.04-1.14 milligrams per liter of water), chromium (3.5-66.3 mg/l), and copper (1.3-20.1 mg/l). The report concluded that "such waters may be of potential environmental concern. Observed concentrations of arsenic, chromium and copper all exceeded Canadian water quality criteria. The result was somewhat surprising because of the extensive design and procedural environmental controls at the facility."[12]

Human Exposure

Workers who suffered health effects following occupational exposure to CCA have successfully obtained compensation from preservative manufacturers. A United States Department of Agriculture employee who experienced internal bleeding and vomiting followed by complete disability after building picnic tables in an unventilated shop received a total of 767,000 dollars from CCA manufacturers in 1987. Evidence introduced during the trial included a memo from Koppers, Inc. (a CCA manufacturer) indicating that they had reports of illness from workers sawing treated wood as early as 1968.[13] In 1990, an employee of the Whatcom County (Washington) Parks and Recreation District whose nervous system was damaged during the installation of bridges constructed of CCA-treated wood was awarded 450,000 dollars in compensatory damages.[14]

Surveys of workers in wood treatment plants have found high levels of arsenic in Hawaii and Nigeria. In the Hawaiian study, wood treatment workers had urinary arsenic levels 1.4 times those of unexposed workers.[15] In the Nigerian study, arsenic concentrations in the hair of wood treatment workers were 1.6 times those of unexposed workers.[16]

Nonoccupational exposure has also caused health problems. For example, a Wisconsin family of eight suffered recurring health problems during heating seasons in three years. The family used a woodstove to heat their house and had been using mostly CCA-treated plywood scraps as fuel. Members of the family reported rashes, respiratory problems, seizures, hair loss, extended "blacking out," eye irritation, and muscle cramps. High arsenic levels were found in hair, air, ash, and dust samples.[17]

Regulatory History

EPA began a Special Review (known then as Rebuttable Presumption Against Registration) of CCA on October 18, 1978. EPA's action was based on their determination that CCA and other inorganic arsenical pesticides met or exceeded EPA's risk criteria for oncogenicity, mutagenicity, and teratogenicity.[8]

In 1988, EPA issued a registration standard for inorganic arsenicals, and concluded the Special Review. The registration standard imposed a number of conditions on the use of CCA, but did not place any restrictions on the use of treated wood.[8]

CCA-treated Wood Playground Equipment

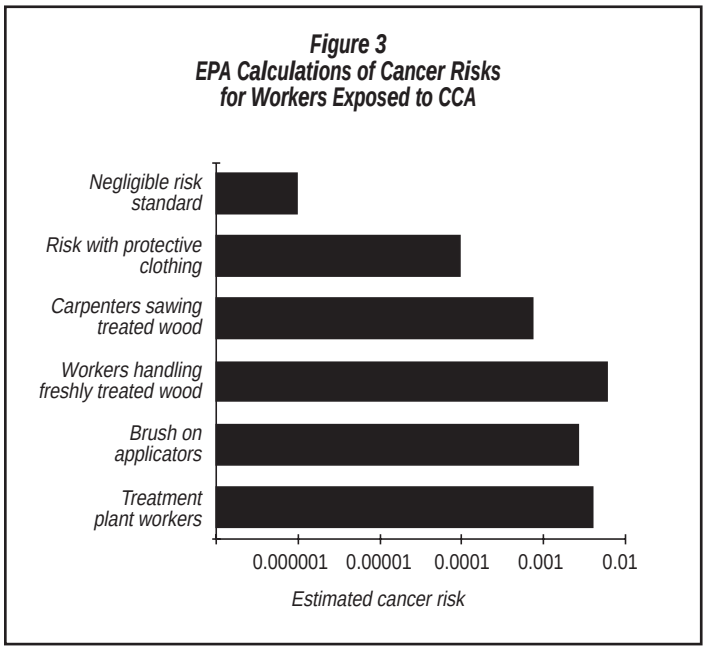
Special concerns about children's exposure to toxic chemicals have prompted studies of their exposure to CCA caused by playing on pressure-treated wood playground equipment. Two studies, one done in the U.S.[1] and the other in Canada[2], have recently been released. Both studies conclude that increased exposure to arsenic results from children's contact with play structures.

In the Canadian study, soil sampling showed that arsenic concentrations at the base of the playground equipment were from 1.8 to 23.5 times the concentration in soil 10 meters away from the equipment. Using cloth to wipe the structures showed that dislodge–able arsenic was present on all ten of the play structures studied, in concentrations ranging from 4.8 to 149.3 micrograms (µg) per wipe (250-500cm^2). The wipe samples were also analyzed for chromium and copper; concentrations of chromium varied from 4.2 to 555.8 µg per wipe and concentrations of copper varied from 5.0 to 132.2 µg/wipe.

The U.S. study used a similar wiping technique to sample seven play structures. Five of the seven showed no detectable arsenic residues while two showed residues of 21.9 and 32.1 µg/100 cm^2. The study then calculated the excess risk of skin cancer resulting from use of the play equipment. Risks (from touching the equipment and not considering possible mouthing of wood or eating playground dirt) were negligible for the equipment without dislodgeable residues and between three and four per million for the equipment with residues.

While both studies stress that arsenic exposure from play equipment is small relative to dietary exposure to arsenic, the Canadian study concludes that "inorganic arsenic and chromium are poisonous and carcinogenic when absorbed in excessive amounts and that while small amounts may be harmless, it would be best to avoid any unnecessary exposure to arsenic or to chromium."

1. Lee, Brian C. 1990. Dislodgeable arsenic on playground equipment wood and the estimated risk of skin cancer. Memorandum. Washington, D.C.: U.S. Consumer Product Safety Commission.
2. Riedel, D., D. Galarneau, J. Harrison, D.C. Gregoire, and N. Bertrand. 1991. Residues of arsenic, chromium and copper on and near playground structures built of wood pressure-treated with "CCA" type preservatives. Ottawa, Canada: Environmental Health Center, Health and Welfare Canada.



CCA (with the exception of commercial brush-on use of the preservative) was classified as a restricted use pesticide, so that only certified applicators and people under their direct supervision could treat wood. In addition, workers who actually enter the pressure treatment cylinder must wear protective clothing and respirators unless the arsenic concentration in the air is less than 10 micrograms per cubic meter. EPA also required the use of closed systems for emptying treatment tanks and mixing formulations and required that no residues of CCA be visible on treated wood.[8]

EPA required an air monitoring study for chromium in wood treatment plants. Determination of the need for teratology and reproduction studies, environmental fate studies, testing for chronic effects on aquatic organisms, and an assessment of impacts on endangered species was deferred.[8]

As part of its Special Review process, EPA estimated risks of cancer to workers exposed to arsenic in wood treatment and fabrication plants. The calculated risks varied from about four cases of lung cancer per 1000 workers exposed in treatment plants to about three cases of skin cancer per 10,000 workers sawing or fabricating treated wood. (See Figure 3.) EPA believed that the protections established during the Special Review would reduce these risks to "the orders of 10^{-4} [1/10,000] to 10^{-5} [1/100,000]" and concluded that "this level of risk does not outweigh the benefits of continued registration of these products."[8]

Conclusion

In 1988 (the most current year for which statistics are available), wood treatment plants in the United States treated 444 million cubic feet of lumber and plywood with 154 million pounds of CCA.[18] This was 50 percent more than had been treated with CCA in 1984[18] and over a 400 percent increase since 1978, when the Special Review of CCA began.[2]

All of this CCA-treated wood is a concentrated source of metals that are acutely and chronically toxic to a wide range of organisms, from single celled algae to humans. In humans, the ability of CCA's ingredients to cause disorders of the nervous system, damage to various organs, cancer, and birth defects is well documented. The sensitivity of other organisms varies from species to species, but the most susceptible species are damaged at concentrations of several parts per billion. Although chromium(VI) can be transformed to the less toxic chromium(III), the toxicity of arsenic and copper is a permanent legacy of treated wood. ■

References

1. U.S. Environmental Protection Agency. 1990. Unpublished list of all pesticide formulations registered for use on wood and wood products. Washington, D.C.: Office of Pesticide Programs.
2. U.S. Department of Agriculture. 1980. *The biologic and economic assessment of pentachlorophenol, inorganic arsenicals, creosote. Volume 1. Wood preservatives.* Technical Bulletin Number 1658-1.
3. Corbett, J.R., K. Wright, and A.C. Baillie. 1984. *The biochemical mode of action of pesticides.* Second edition. London: Academic Press.
4. National Research Council, Committee on Medical and Biologic Effects of Environmental Pollutants. 1977. *Arsenic.* Washington, D.C.: National Academy of Sciences.
5. World Health Organization. 1988. *Chromium.* Environmental Health Criteria 61. Geneva, Switzerland: Published under the joint sponsorship of the United Nations Environment Program, the International Labor Organization, and the World Health Organization.
6. Hodson, Peter V., Uwe Borgmann, and 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.
7. World Health Organization. 1981. *Arsenic.* Environmental Health Criteria 18. Geneva, Switzerland: Published under the joint sponsorship of the United Nations Environment Program, the International Labor Organization, and the World Health Organization.
8. U.S. Environmental Protection Agency. 1988. *Guidance for the reregistration of wood preservative pesticide products containing chromated and non-chromated arsenicals as the active ingredient.* Washington, D.C.: U.S. EPA Office of Pesticide Programs.
9. Cohen, Steven. 1979. Environmental and occupational exposure to copper. *In* Nriagu, Jerome O. (ed.) *Copper in the environment. Part II: Health effects*, pp. 1-16. New York, NY: John Wiley and Sons.
10. U.S. Environmental Protection Agency. 1987. *Guidance for the reregistration of wood preservative pesticide products containing Group II copper compounds as the active ingredient.* Washington, D.C.: U.S. EPA Office of Pesticide Programs.
11. Solomon, Keith R. and John E. Warner. 1990. Persistence, leaching, and bioavailability of CCA and pentachlorophenol wood preservatives. Final report to the Ontario Ministry of the Environment. July 2.
12. Birge, Wesley J. and Jeffrey A. Black. 1979. Effects of copper on embryonic and juvenile stages of aquatic animals. *In* Nriagu, Jerome O. (ed.) *Copper in the environment. Part II: Health effects*, pp. 374-397. New York, NY: John Wiley and Sons.
13. McCrea, David. 1988. Arsenic in lumber. Citizen's Clearinghouse for Hazardous Wastes, Inc. *Environmental Health Monthly* (September 30): 2-7.
14. The Bureau of National Affairs. 1990. $450,000 award won by worker who developed arsenic poisoning. *Occupational Safety and Health Reporter* (November 7): 984.
15. Takahashi, Wataru, Karl Pfenninger, and Lyle Wong. 1983. Urinary arsenic, chromium, and copper levels in workers exposed to arsenic-based wood preservatives. *Archives of Environmental Health* 38(4):209-214.
16. Ndiokwere, Ch.L. 1985. A survey of arsenic levels in human hair and nails - Exposure of wood treatment factory employees in Nigeria. *Environmental Pollution (Series B)* 9:95-105.
17. Peters, Henry, William Croft, Edwin Woolson, Barbara Darcey, and Margaret Olson. 1984. Seasonal arsenic exposure from burning chromium-copper-arsenate-treated wood. *Journal of the American Medical Association* 251(18): 2393-2396.
18. Micklewright, James T. 1990. *Wood preservation statistics, 1988: A report to the wood preserving industry in the United States.* Stevensville, MD: American Wood-Preservers' Association.