ENVIRONMENTAL HEALTH SECRETS

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20. PERSISTENT ENVIRONMENTAL CONTAMINANTS

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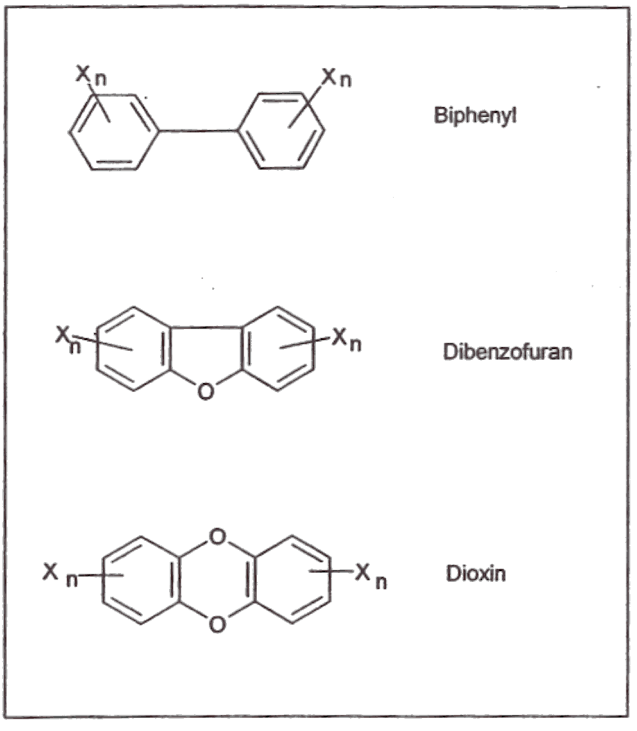
HALOGENATED DIOXINS/FURANS

1. What are halogenated dioxins and furans?

Dioxins and furans are related compounds characterized by a similar backbone that varies in the number and position of halogen atom substitutions, as shown in the figure below. Although there are many forms of these compounds, the best known is 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). TCDD is the most potent of this class of chemicals, and as a result the biological activities of the other dioxins and furans are often described relative to that of TCDD. Only a small subset of the total dioxins and furans, however, are thought to induce health effects through the same mechanism of TCDD.



2. How do these compounds get into the environment?

Halogenated dioxins and furans are not thought to be naturally occurring, rather they are formed as by-products of various industrial and combustion processes. Potential sources of release to the environment include chlorination processes such as paper bleaching, manufacture of chlorinated organic chemicals, including some pesticides; and combustion of fossil fuels and municipal and hazardous waste. Chlorinated dioxins and furans can also be present in cigarette smoke.

Once these compounds are released into the environment they are resistant to being degraded, and thus they tend to be persistent. They are generally hydrophobic and for this reason they can bioaccummulate in the fatty tissues of animals that have ingested contaminated vegetation, soil, or water. The diet, primarily in meat and dairy products, is thought to be the major pathway for chronic low-level exposure in humans.

3. What are the primary health effects from exposure to dioxins and furans?

TCDD is noted for the wide array of physiological systems affected. A hallmark symptom of high TCDD exposure in humans is chloracne, a severe skin disease characterized by acnelike lesions. Chloracne has been associated with body burdens of TCDD ranging from 18 to 2357 ng/kg body weight. Erythematous or red skin rashes, discoloration, and excessive body hair have also been reported to occur in people following exposure to high concentrations of TCDD. Mild and reversible alterations in the ability of the liver to metabolize hemoglobin, lipids, sugar, and protein have been reported to occur in people following exposure to high concentrations of TCDD. Other effects have been less consistently reported in people. It is not clear whether TCDD can induce adverse reproductive or developmental effects in humans and the carcinogenicity data for TCDD have provided mixed results, with varying interpretations. The International Agency for Research on Cancer concluded that TCDD can cause cancer in humans and the U.S. Environmental Protection Agency (EPA) classifies TCDD as a possible human carcinogen.

TCDD dosing has been shown to induce adverse effects in many target organs in animal toxicity studies, including a wasting syndrome, thymic atrophy and other immunological effects, and effects on reproduction and development. It should be noted that there is considerable variability in species sensitivity to the effects of TCDD, therefore for many end points the applicability to exposed humans remains unclear.

POLYCHLORINATED BIPHENYLS

4. What are polychlorinated biphenyls?

The polychlorinated biphenyls (PCBs) are a group of 209 different compounds, referred to as congeners, characterized by a similar chemical backbone that varies in the number and position of chlorine atoms (see figure, question 1). In most cases, PCBs are described based on the toxicity of several PCB mixtures that have been commercially available. These mixtures, called arochlors, are unique mixtures identified by a number indicating the number of carbon atoms per molecule and percentage of chlorine in the mixture. For example, the U.S. EPA has reviewed the toxicity data for the development of human health risk values for arochlors 1016, 1248, and 1254.

5. How do these compounds get into the environment?

PCBs are not known to be naturally occurring. They once were manufactured extensively for use as electrical insulators in transformers and capacitors. Although no longer used in these products, PCBs can be released into the environment by leaking from old electrical equipment that still contains PCBs. These compounds can also be generated during the combustion of municipal and industrial waste.

Due to their hydrophobic nature, PCBs are resistant to degradation in the environment, where they associate with organic matter in sediment. Similarly to dioxins and furans, PCBs bioaccumulate in the adipose tissues of animals that have ingested contaminated vegetation, soil, or water and in the diet, primarily in meat and dairy products. This is thought to be the major pathway for chronic low-level exposure in humans.

6. What are the primary health effects of exposure to PCBs?

Acute exposures to PCBs have been associated with a variety of symptoms in exposed people. These include skin irritation, acne and rashes, neurological effects such as general weakness and numbness, respiratory irritation following exposure by the inhalation route, altered immune function, and liver damage. Toxicity studies in animals have shown a similar array of effects following acute exposures.

In chronic animal exposure studies, PCB treatment administered orally resulted in effects on the liver, stomach, and thyroid gland and decreased reproductive ability. The human evidence is inconclusive regarding the ability of PCBs to cause cancer, but animal studies have reported increased tumor incidence in PCB-treated animals. Based on these data, the U.S. EPA classifies

s as probable human carcinogens and the International Agency for Research on Cancer has mined that PCBs may reasonably be anticipated to be human carcinogens.

MERCURY

What is mercury?

Each year, anywhere from 2700 to 6000 tons of mercury is released into the environment natural sources, and another 2000 to 3000 tons is contributed by human activities. Three s of mercury exist, each having different physical and chemical properties, routes of expo- and health effects. The first is metallic mercury, or mercury in its elemental form. It appears r and silver and is a liquid at room temperature. Mercury vapor is released from the metal in tate. The second form is a group of inorganic mercury compounds, or mercury salts. These rmed when mercury combines with other elements such as oxygen, sulfur, or chlorine. Most as powders or crystals. The third form is organic mercury, which is created when mercury bines with carbon-based molecules. The most common organic mercury compound is ylmercury.

How are people exposed to inorganic (metallic and the inorganic salts) mercury?

Broken thermometers, shattered fluorescent tubes, or damaged electrical switches can ex- people to metallic mercury through inhalation of the vapors. Breathing mercury vapors is ncern in closed areas, because 80% of inhaled mercury vapor is absorbed directly into the dstream. Swallowing metallic mercury is not as much of a health concern, because the ach or intestines absorb less than 0.01% of the dose. Dermal contact with the metal can e rashes.

How are people exposed to methylmercury?

Microorganisms in the water can convert metallic mercury into methylmercury. Methylmer- bioaccumulates, and can be present in significant concentrations in the tissues of organisms the tops of food chains—shark, swordfish, and large tuna being several examples. Cooking not appreciably reduce the methylmercury concentration in food. Methylmercury causes fe- ethylmercury poisoning, and can be passed in breast milk. Children and fetuses are the most itive to exposure.

What are the effects of mercury exposure?

Mercury impacts the nervous system. Remember the Mad Hatter in Alice in Wonderland? d as a hatter!" is a saying describing milliners who had developed mercury poisoning from exposure to mercurious nitrate, once used in the manufacture of hats. A variety of symp- s are possible for exposure to the various forms of mercury, and depend on degree of ex- ure. Short-term exposure to metallic mercury vapor can irritate and damage the tissue lin- the mouth and lungs, causing a burning sensation and tightness of breath. Paresthesia, a bness and tingling sensation around the fingers, toes, and lips, is usually an early nervous em manifestation of exposure. The next progressive symptom is characterized by a diffi- walking and talking, followed by a constriction of visual fields, and finally tunnel vision loss of hearing. People may demonstrate nervousness, irritability, shyness, an inability to centrate, and memory loss. Severe exposures can lead to tremors or jerks. Kidney damage occur, especially after exposure to toxic concentrations. Coma or death is the end to this gression.

How can exposure to mercury be measured?

Mercury levels in the body can be measured in blood, urine, or hair samples. Breast milk can be tested for mercury concentrations. Urine samples can indicate exposure to metallic mer- vapor and to inorganic forms of mercury, whereas whole blood and scalp hair mercury con- rations correlate to methylmercury exposure.

12. Is there a health risk from mercury in dental fillings?

Metallic mercury is often present in dental amalgam, which is used in fillings. Although U.S. government evaluations conclude there is no health threat posed to the general population from dental amalgam, there is currently an investigation on the health risks for sensitive populations and the possibility of subtle immune or behavioral effects.

PESTICIDES

13. What are pesticides, and what categories of pesticides exist?

The United States Environmental Protection Agency defines pesticide as any substance or mixture of substances intended for preventing, destroying, repelling, or mitigating any pest. There are five broad categories. The first category is **insecticides,** containing the organochlorine, anticholinesterase, pyrethroid, and botanical insecticides. **Herbicides**, the second category, encompass the chlorophenoxy compounds and bipyridyl derivatives. The third category is **fungicides**, grouping hexachlorobenzene, pentachlorophenol, phthalimides, and dithiocarbamates. **Fumigants** compose the fourth category and include phosphine and ethylene dibromide/dibromochloropropane. The final and fifth category is the **rodenticides**, including the zinc phosphide, fluoroacetic acid and derivatives, α-naphthyl thiourea (ANTU), and anticoagulants.

14. How long have pesticides been used?

Pesticide use has been recorded for millennia. Homer recounted the use of sulfur for fumigation in 1000 B.C. and in 200 B.C. Cato wrote about using sulfur to destroy vine pests. By 900 A.D., the Chinese were using arsenic to control garden insects. However, widespread chemical pesticide use did not become widely established until the mid-19th century. These precursors to modern pesticides included primarily inorganic compounds such as copper and mercury salts, sulfur, arsenic, and cyanide. Some organic compounds were used and included tar distillates and natural plant extracts such as dersis (a root extract), nicotine, and pyrethrum.

15. What are the relative benefits and concerns of pesticide use?

Many people believe that the use of pesticides has brought about positive changes for humans such as improved food quality, decreased food prices, improved animal welfare and human health, and increased productivity. However, overuse of pesticides, contamination of water supplies, and the appearance of residual pesticides in foodstuffs have raised the level of concern of health scientists and the public to the acute and chronic effects from pesticides (carcinogenicity, mutagenicity, teratogenicity, immune system effects, and central nervous system effects). As we better understand the potential human and ecological health effects of the pesticides, governmental regulatory agencies can apply more prudent measures to balance the concern of pesticide use with the perceived societal benefits.

16. How do people come into contact with pesticides?

Pesticides are in use in agriculture, home gardens and lawns, in homes, and in some public places such as parks or airplanes. The most likely population to be exposed to potentially unsafe levels is the occupationally exposed, such as farmworkers or workers in a pesticide manufacturing plant. Exposure can occur by dermal, oral, or inhalation routes. In extreme situations, poisonings can occur. Residues of pesticides have been found on food, such as produce.

17. Why are pesticides considered toxic, and what are the modes of action?

Pesticides were developed to be toxic to specific living things, be it vertebrates, invertebrates, or microorganisms. The chemical compounds that make up each pesticide have a certain effect on biological systems, ideally impacting only the target organism. Unfortunately, nontarget species frequently are affected because their physiological and/or biochemical systems are simi-

› those of the target species. Each of the different classes of pesticides has a different mode tion. For example, exposure to organochlorine and anticholinesterase insecticides, of which ʻdane, dieldrin, and methoxychlor are members, can result in central nervous system effects. urces for pesticide poisoning guidance include the National Pesticide Telecommunications ʻork (NPTN) at 800-858-7378 (internet site: www.ace.orst.edu/info/nptn/index.html) and the ician's Guide to Pesticide Poisoning, available online at www-aes.tamu.edu/doug/MED/ P.htm.

DDT (P,P'-DICHLORODIPHENYLTRICHLOROETHANE)

s DDT still a pollutant of concern in the United States?

)DT was recognized to have insecticide properties in the late 1930s and was used exten- y in the United States until its ban in 1972. Since that time, levels of DDT in the environ- in many parts of the United States have decreased; however, DDT is still used in other of the world and atmospheric deposition of DDT represents a source of current contami- n. In addition, DDT undergoes a gradual breakdown in the environment to DDE. There- contaminated sites can still have significant levels of both DDT and DDE arising from pesticide use. Because of its continued international use and environmental persistence, sure to DDT and DDE can result from consumption of contaminated fish, crops grown in minated soil, or imported crops that were sprayed with DDE. Lactational transfer is a path- or exposure of infants.

Vhat caused DDT to be banned?

n 1962, Rachel Carson published "Silent Spring," warning the world about the widespread r more appropriately, misuse) of pesticides. Subsequently, DDT was shown to interfere with ıell-making capability (reproduction) of avian wildlife, causing a dramatic decrease in the ation of many birds, including the American bald eagle. As a result, DDT was banned for ı the United States.

Vhat health effects are associated with DDT and DDE?

imilarly to other persistent chlorinated pollutants, DDT has been associated with liver and logical effects. The evidence for carcinogencity is mixed in human epidemiology studies, ısed on positive results in animal studies, the U.S. EPA has classified DDE as a probable hu- arcinogen. A primary effect of concern is the potential adverse reproductive effects of DDT)DE. Although the human evidence for adverse effects are not conclusive, a variety of ad- effects have been reported in animal toxicology studies.

CONTROVERSY

ʻan eating sport fish lead to a significant health risk?

ſany persistent environmental pollutants can bioaccumulate in wildlife that are exposed to ninated environments. Based on the recognition that human exposures to pollutants can be cant from eating fish, the U.S. EPA and many state agencies establish fish consumption ad- ɛs. Advisories have been established for many bodies of water and are based on levels of ants such as mercury, PCBs, dioxins, chlordane, and DDT, among others. Fish consump- ſvisories are available though the U.S. EPA data bases or from state health or environmen- ɛncies.

lthough the potential for adverse effects of eating highly contaminated fish is clearly rec- ɔd, the trade-off between risk from low levels of these pollutants and the health risk from tuting fish with less healthy dietary alternatives is uncertain and an area of active research. ıt work focuses on development of methods to assess these comparative risks.

BIBLIOGRAPHY

1. Agency for Toxic Substances and Disease Registry: Toxicological profile for chlorinated dibenzo-*p*-dioxins. U.S. Dept. of Health and Human Services. Washington, D.C., U.S. Govt. Printing Office, 1998.
2. Agency for Toxic Substances and Disease Registry: Toxicological profile for 4,4′-DDT, 4,4′-DDE, 4,4′-DDD. U.S. Dept. of Health and Human Services. Washington, D.C., U.S. Govt. Printing Office, 1994.
3. Agency for Toxic Substances and Disease Registry: Toxicological profile for mercury. U.S. Dept. of Health and Human Services. Washington, D.C., U.S. Govt. Printing Office, 1999.
4. Agency for Toxic Substances and Disease Registry: Toxicological profile for polychlorinated biphenyls. U.S. Dept. of Health and Human Services. Washington, D.C., U.S. Govt. Printing Office, 1997.
5. Arbuckle TE, Sever L.E: Pesticide exposures and fetal death: A review of the epidemiologic literature. Crit Rev Toxicol 28(3): 229–270, 1998.
6. Ecobichon DJ: Toxic effects of pesticides. In Klassen CD (ed): Casarett and Doull's Toxicology: The Basic Science of Poisions. New York, McGraw-Hill, 1996.
7. Orloff KG, Fagliano J, Ulirsch G *et al.*: Human exposure to elemental mercury in a contaminated residential building. Arch Environ Health 52(3):169–172, 1997.
8. Toxicology Excellence for Risk Assessment: Comparative dietary risks: Balancing the risks and benefits of fish consumption, 1999. Internet site: http://www.tera.org.
9. U.S. Environmental Protection Agency: National listing of fish and wildlife advisories, EPA-823-F-99-005, 1999. Internet site: http://www.epa.gov/ost/fish/.
10. U.S. Environmental Protection Agency: Unified Air Toxics Website, 1999; DDE (*p,p*-dichlorodiphenyldichloroethylene). Internet site: http://www.epa.gov/ttnuatw1/hlthef/dde.html.
11. U.S. Food and Drug Administration: Mercury in fish: Cause for concern? FDA Consumer report, 1999. Internet site: http://vm.cfsan.fda.gov/~dms/mercury.html.