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Profiles of Selected Chemicals

Industrial Chemicals and Byproducts

Chemical Name: DIOXIN AND DIOXIN-LIKE COMPOUNDS
[POLYCHLORINATED DIBENZO-*p*-DIOXINS (PCDDs) and POLYCHLORINATED DIBENZO-*p*-FURANS (PCDFs)]

Summary: The chemical 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD, or simply, "dioxin") is the most highly toxic and well-studied chemical in a family of compounds termed "dioxin-like" compounds. This family includes polychlorinated dibenzo-*p*-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), polybrominated dioxins (BDDs), polybrominated dibenzofurans (BDFs), and certain polychlorinated biphenyls (PCBs). These compounds share similar chemical structures, properties, and common toxic effects, and often occur in mixtures in the environment. For simplicity, this report refers to this family of compounds as "dioxins." Dioxins are unintentional byproducts of high temperature combustion (e.g., incinerators and boilers), fires, and certain industrial processes. They bioaccumulate in the food chain and can contaminate meat, dairy products, and fish. Food is the primary source of dioxin exposure for the general population. Dioxins are carcinogens and can have harmful effects on hormone function, the immune and reproductive systems, and fetal development.

Uses and Sources: Chlorinated dioxins and furans are not intentionally manufactured, rather, they are unintentionally formed as byproducts of fossil fuel and wood combustion; industrial, municipal, medical, and domestic waste incineration; metal smelting; and chlorine bleaching and manufacturing processes. Chlorinated dioxins and furans are also formed by combustion of polyvinyl chloride (PVC, commonly used in consumer product packaging and medical devices), and polychlorinated biphenyls (PCBs). Uncontrolled combustion processes, such as fires in buildings, can lead to formation of both chlorinated and brominated dioxins and furans. Polybrominated dioxins and dibenzofurans are formed when flame-retardants used in certain plastics (e.g., textiles and foam furniture cushions) are burned.

The Stockholm Convention will require governments to take steps to minimize and, where feasible, eliminate releases of dioxins and furans.

Major Consumer Products: Dioxins and furans are not intentionally produced for any consumer uses. Chlorinated forms of dioxins and furans do occur as low-level contaminants in some commercial products, including the wood preservative pentachlorophenol (PCP) and in some pesticides and herbicides.

Routes of Human Exposure: In the environment, dioxins and furans are found in soil, sediment, and food as a result of industrial and combustion processes. Once in the environment, these compounds can remain for years and even decades before breaking down through natural processes. Dioxins accumulate in the fat of food-producing animals and are transferred into meat, eggs, and dairy products. EPA estimates that most (95%) dioxin exposure occurs through eating these animal fats. People who eat higher than average amounts of contaminated foods (e.g., sport or subsistence fishermen) can be exposed to significantly higher amounts of dioxins (as well as other persistent chemicals) than the general public. Once in the human body, dioxins can be stored in fatty tissues for

long periods. They can be released into human breast milk, leading to exposure to nursing infants. Despite the presence of dioxin in breast milk, medical experts still strongly support breastfeeding, based on its clear health benefits for infants. Additional study is needed to evaluate long-term effects of exposure to dioxin and other persistent chemicals in breast milk. Other populations that may have dioxin exposures higher than the general population include cigarette smokers and people living near incinerators.

In addition to food, people are exposed to dioxins and furans through contact with contaminated soils, urban air, and water sources, although the levels of contamination found in these sources are typically much lower.

Health Effects: Dioxins and related compounds are highly toxic chemicals that can cause a number of different health effects, depending on the extent of exposure and individual sensitivity. Effects range from subtle biochemical and cellular changes to more serious long-term effects, such as cancer and delays in childhood development. Both short-term, high-level (acute) exposures and low-level daily exposures can lead to chronic effects. These may include long-term effects on human reproduction, abnormal hormonal function, toxic effects on the nervous system, and weakening of the immune system. Animal studies and human evidence suggest that these long-term effects can result from exposure to low levels of dioxins found in the ambient environment. The developing fetus, infants, and young children may be particularly vulnerable to adverse effects of dioxins, because their bodies are rapidly growing. Prenatal exposure to one form of dioxin (2,3,7,8-tetrachlorodibenzo-*p*-dioxin, or TCDD) by laboratory animals has resulted in adverse effects on learning and behavior, physical deformities, retarded growth, and changes in physiology.

One of the most serious health effects of dioxin exposure is cancer. Studies have shown that workers exposed to high levels of dioxins in the workplace over many years have a higher risk of cancer than the general population. EPA has determined that the mixtures of dioxins to which people are typically exposed are likely human carcinogens. Both EPA and the International Agency for Research on Cancer (IARC) have classified TCDD as a known human carcinogen. The compound 2,3,7,8-TCDD is significantly more toxic than other forms of dioxin and is one of the most potent human carcinogens.

Detecting Dioxin Exposure: Dioxins tend to accumulate in fat and liver tissue. Blood serum lipids and tissue samples can be analyzed to determine dioxin levels in the body.

Regulations for Protection of Human Health:

Air – Dioxin (2,3,7,8-TCDD) is regulated by EPA as a Hazardous Air Pollutant under the Clean Air Act.

Drinking Water – EPA has established an MCL of 0.00000003 mg/L (or $3x10^{-8}$ mg/L) for 2,3,7,8-TCDD in drinking water.

Food – Levels of dioxin in food are not currently regulated.

Occupational Exposures - OSHA has not established a PEL-TWA for dioxins.

Sources of Information:

ATSDR. *Toxicological Profile for Chlorinated Dibenzo-p-dioxins (CDDs).* Agency for Toxic Substances and Disease Registry, Atlanta, GA.1998. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp104.html

EPA. Draft Dioxin Reassessment. Office of Research and Development, Washington, DC. 2001. Accessed online at http://cfpub.epa.gov/ncea/cfm/dioxreass.cfm

Weber LWD and H. Greim. The toxicity of brominated and mixed-halogenated dibenzo-p-dioxins and dibenzofurans: An overview. *Journal of Toxicology and Environmental Health* 50:195-215 (1997).

Mason G et al. Polybrominated dibenzo-p-dioxins and related compounds: Quantitative in vivo and in vitro structure-activity relationships. *Toxicology* 44:245-255 (1987).

Chemical Name: POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)

Summary: Polycyclic aromatic hydrocarbons (PAHs) are a group of more than 100 different chemicals that are formed by incomplete combustion processes. Exposure to PAHs can occur through inhalation of tobacco smoke or smoke and soot from fires (residential and industrial), skin contact with contaminated soot or soil, or ingestion of contaminated food or drinking water. Long-term exposure to PAHs can lead to serious health effects including cancer. EPA has determined that several PAHs are probable human carcinogens.

Uses and Sources: Most PAHs have no commercial uses. PAHs generally occur as mixtures of multiple compounds, and they adhere to soot and dust particles in the air. PAHs are present in smoke, formed by incomplete combustion of fuels such as coal, oil, gas, and wood. Tobacco smoke is a major source of PAH exposure for many people. Other sources of PAHs in the environment include waste incineration, vehicle exhaust, cigarette smoke, and releases from wildfires and volcanoes. PAHs can also be formed on food that has been cooked at high temperatures, such as by grilling or charbroiling.

Major Consumer Products: A few PAHs are used in certain medicines and to make dyes, plastics, pesticides, asphalt, creosote, and roofing tar.

Routes of Human Exposure: Workers in industries such as coal tar production plants, coking plants, asphalt production plants, coal-gasification sites, coal-tarring facilities, and municipal trash incinerators may have exposures to PAHs. Firefighters can also be exposed to PAHs in smoke and soot.

For most people, exposure to PAHs occurs by breathing tobacco smoke, wood smoke, and ambient air. PAHs are found in some foods, including food grown in contaminated soil, food that is grilled or charbroiled, dairy products, and breast milk.[1] People can also be exposed to PAHs from contact with creosote-treated wood products, soil where certain fuels have been burned, and by consuming drinking water contaminated with PAHs.

Health Effects: Cancer is the health effect of greatest concern with respect to PAHs. Animal and human studies have shown that long-term PAH exposure can cause lung cancer (from inhalation of PAHs), skin cancer (from direct skin contact), and possibly cancer at other sites in the body. EPA has determined that several PAHs, including benzo[a]pyrene, are probable human carcinogens.

Little is known about the adverse health effects of short-term (acute) and intermediate duration (typically defined as less than one year) exposure to PAHs. Acute skin exposure can cause skin irritation and lesions, and long-term dermal exposure can cause chronic dermatitis, an abnormal thickening of the skin (called hyperkeratosis), and potentially skin cancer. By the inhalation route, short-term exposure can cause temporary effects on

[1] The health benefits of breast-feeding normally greatly outweigh any small risk from contaminants in mothers' milk. Local health departments can be a good source of information about breast-feeding of infants.

the respiratory system (such as chest pains and difficulty breathing), while long-term inhalation exposure can lead to lung cancer.

Detecting PAH Exposure: PAHs and their metabolites can be measured in urine, blood, and tissue samples, although they are usually excreted within a few days.

Regulations for the Protection of Human Health:

Air – PAHs are regulated as Hazardous Air Pollutants under the Clean Air Act.

Drinking Water – EPA has established an MCL of 0.0002 mg/L for the PAH benzo[a]pyrene in drinking water.

Occupational exposure – OSHA has established a PEL-TWA of 0.2 mg/m^3 for coal tar pitch volatiles and 5 mg/m^3 for mineral oil mist. Both exposure limits are regulated as average concentrations over an 8-hour exposure period.

Source of Information:

ATSDR. *Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHs*). Agency for Toxic Substances and Disease Registry, Atlanta, GA.1995. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp69.html

Chemical Name: POLYCHLORINATED BIPHENYLS (PCBs)

Summary: PCBs have been used as insulating chemicals, coolants, and lubricants. Production of new PCBs is banned in the U.S., but existing PCB-containing equipment remains in use here and around the world. PCBs in the environment can persist for decades. A number of human studies have linked PCBs to adverse health effects including cancer and neurological effects on the developing fetus. In addition, there is ample evidence from animal studies that PCBs can cause immune deficiencies, impaired reproduction, anemia, various skin conditions, and neurological effects.

Uses and Sources: PCBs were extensively used as coolants and lubricants in capacitors, transformers, and other electrical equipment; hydraulic fluids; flame-retardants; and for many other industrial and commercial purposes. Concern about environmental contamination with PCBs led to a ban on manufacture in the U.S. in 1977. However, hundreds of thousands of tons of PCB-containing fluids are still in use in electrical and industrial equipment and are also found in industrial wastes. PCBs are released into the environment from leaking equipment, electrical transformer fires and accidents, and from hazardous waste sites where PCBs have been disposed. PCBs can also be released during combustion of some wastes in municipal and industrial incinerators. PCBs usually occur as mixtures of individual chlorinated biphenyl compounds. They are extremely persistent and can remain for years in soils and sediments. They also move easily between air, soil, and water and can travel long distances in the environment. According to EPA's Toxic Release Inventory, industries released nearly 1.5 million pounds of PCBs into the environment in 2000. In addition to ongoing releases, past dumping by companies has left reservoirs of these chemicals in river sediments and soils across the country. PCBs are continuously re-released from such reservoirs into the general environment. The Stockholm Convention, when implemented, will prohibit the production of PCBs. It also calls on countries to phase out the use of PCBs by 2025.

Major Consumer Products: PCB-containing products are no longer manufactured in the U.S. However, old consumer products such as refrigerators, televisions, and fluorescent light fixtures made before 1977 may still contain PCBs.

Routes of Human Exposure: For the general population, the vast majority of PCB exposure occurs through the diet. PCBs have a strong tendency to bioaccumulate in fatty tissues of organisms and can reach high concentrations in animals. Fatty fish (particularly freshwater fish), meat and meat products, and milk typically have higher concentrations of PCBs than other types of food. People consuming large quantities of these foods, such as Great Lakes sport fishermen and Arctic indigenous populations, can have PCB body burdens many times higher than the general population. PCBs are also found in human breast milk, which can lead to infant exposure through breastfeeding. People can also be exposed to PCBs by breathing contaminated air or dust, drinking contaminated water, and by skin contact with contaminated soil or PCB-containing items. PCBs are common contaminants in and around hazardous waste sites and landfills and pose a health threat to people living near these areas.

Health Effects: PCBs are toxic chemicals that can cause a wide range of adverse health effects. EPA has classified PCBs as probable human carcinogens. PCBs also have a variety of other adverse effects including immune system suppression, nervous system effects, disruption of normal hormone function, and reproductive abnormalities such as reduced birth weight and reduced fertility. Early life exposure to PCBs can cause harmful neurological effects, leading to learning deficits, poor memory, and behavioral effects.

Detecting PCB Exposure: PCBs can be measured in blood, body fat, and breast milk. The CDC National Exposure Report analyzed body burdens of over twenty different PCBs.

Regulations for Protection of Human Health:

Air – PCBs are regulated by EPA as Hazardous Air Pollutants under the Clean Air Act.

Drinking Water – EPA has established an MCL of 0.0005 mg/L for PCBs in drinking water.

Food –FDA has established tolerances for PCBs in various foods, animal feed products, and certain paper food packaging materials. Tolerances are measured in parts per million (ppm), which is the same thing as mg/kg. Food tolerances are: infant and junior foods (0.2 ppm); eggs (0.3 ppm); milk and manufactured dairy products (1.5 ppm, fat basis); fish and shellfish (2 ppm in edible portions); and poultry and red meat (3 ppm, fat basis). A tolerance of 10 ppm has been established for PCBs in paper food-packaging material in direct contact with human food or animal feed.

Occupational Exposure – OSHA has established a PEL of 1 mg/m^3 for 52% chlorine PCBs, and 0.5 mg/m^3 for 54% chlorine PCBs.

Sources of Information:

ATSDR. *Toxicological Profile for Polychlorinated Biphenyls (PCBs)*. Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services, Atlanta, GA. 2000. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp17.html

Chemical Name: PHTHALATES

Summary: Phthalates are a group of industrial chemicals commonly found in consumer products, including flexible plastics, cosmetics, children's toys, pesticides, furniture, pharmaceuticals, and lubricants. Biomonitoring studies by CDC have confirmed that everyone is exposed to phthalates—especially women of childbearing age. The human health effects of phthalates are not well understood, but evidence from animal studies has raised concerns that they may affect reproduction and early fetal development, particularly in males.

Uses and Sources: Phthalates are used to make polyvinyl chloride (PVC) plastics softer and more flexible and to increase moisturizing properties of some personal care products. The most commonly used phthalates are benzylbutyl phthalate (BBP), dibutyl phthalate (DBP), di-2-ethylhexyl phthalate (DEHP) and di-isononyl phthalate (DINP). BBP is added to cosmetic products, flooring, paints, adhesives, and printing inks. DBP is used in cosmetics, toys, flooring, adhesives, furniture and a variety of other products. DEHP is widely used in medical devices, flooring, auto parts, and many other products. Among the uses of DINP are children's toys, flooring, fabrics, garden hoses, and food packaging. According to EPA's Toxics Release Inventory for 2000, reporting industries released about 285,000 pounds of DBP, nearly 250,000 of DEHP, and about 325,000 of dimethyl phthalate (DMP) into the environment. These are the only phthalates reported in the TRI, and the estimates do not reflect environmental releases from finished products.

Major Consumer Products: The vast majority of phthalates are used in production of polyvinyl chloride products, including construction material (such as pipes and vinyl siding), medical equipment, and some children's toys. DEHP is also used to produce cosmetics, lotions, hair products, pesticides, paints, inks, adhesives, lubricants, furniture, and other common plastic products. DBP, diethyl phthalate (DEP), and BBP are commonly used in cosmetic products, including perfume, soap, shampoo, hand lotion and nail polish.

Routes of Human Exposure: People can be exposed to phthalates directly from use of certain consumer products, or indirectly by exposure to phthalates released into the environment. Phthalates (particularly DEHP) can leach out of PVC materials, such as blood bags and vinyl siding. Phthalates can also be absorbed into the body following use of certain cosmetic products, and children can be exposed to phthalates from toys and other objects placed in the mouth. Phthalates are common environmental contaminants as a result of their manufacture, use, and disposal. However, phthalates are not highly persistent in the environment or in the body.

Biomonitoring conducted as part of CDC's first National Exposure Report (2001) showed widespread exposure to phthalates in the population. Although DEP and DBP are produced in much lower quantities than other phthalates, metabolites of these compounds were found at much higher levels in urine of the study population, especially women. The reason for higher exposures among women is not known, but the available evidence

suggests that cosmetics may be a significant source of exposure. Hospital patients who undergo long-term care, such as trauma patients, can be exposed to high levels of DEHP.

Health Effects: The human health effects of phthalate exposure have not been well studied. However, animal studies have raised serious concerns about potential adverse effects of these compounds on humans, particularly for the developing fetus and young children. The increased vulnerability of the fetus to phthalates is particularly troubling, given the evidence indicating relatively high levels of exposure to these compounds by women of reproductive age. Studies have shown that several phthalate compounds, including DEHP, DBP, BBP, and DINP can have a wide range of adverse effects on reproduction and development, including decreased fertility, birth defects, hormone disruption, and reproductive malformations, particularly in the male reproductive tract. Animal studies have shown that exposure to phthalates can also damage the liver, kidney, heart, and lungs.

Although the toxic and carcinogenic effects of DEHP have been well established in laboratory studies, its ability to produce similar effects in humans is controversial. An expert panel recently convened by the National Toxicology Program (NTP) found that DEHP causes harm in experimental animals, including effects on testicular development and sperm production in young animals.

Detecting Phthalate Exposure: Phthalate exposure is most easily determined by measuring specific metabolites in urine. The table below identifies the specific metabolites associated with each of the major phthalate compounds.

Phthalate name	**Abbreviation**	**Primary urine metabolite**
Diethyl phthalate	DEP	Mono-ethyl phthalate
Dibutyl phthalate	DBP	Mono-butyl phthalate
Benzylbutyl phthalate	BBP	Mono-benzyl phthalate
Dicycolhexyl phthalate	DCHP	Mono-cyclohexyl phthalate
Di-2-ethylhexyl phthalate	DEHP	Mono-2-ethylhexyl phthalate
Dioctyl phthalate	DOP	Mono-n-octyl phthalate
Di-isononyl phthalate	DINP	Mono-isononyl phthalate

Regulations for Protection of Human Health:

Air – DEHP, DBP, and dimethylphthalate (DMP) are regulated by EPA as Hazardous Air Pollutants under the Clean Air Act.

Drinking Water – EPA has established an MCL of 0.006 mg/L for di-2-ethylhexyl phthalate in drinking water.

Food – The FDA has established specific regulations controlling the use of DEHP in food packaging.

Occupational Exposures – OSHA has established a PEL for DEHPs during an 8-hour workday of 5 mg/m^3. The ACGIH has established 8-hour limit of 5 mg/m^3 for diethyl phthalate.

Sources of Information:

ATSDR. *Toxicological Profile for Di(2-ethylhexyl) phthalate (Update).* Agency for Toxic Substances and Disease Registry, Atlanta, GA. 2002. Accessed online at: http://www.atsdr.cdc.gov/toxprofiles/tp9.html

ATSDR. *Toxicological Profile for Di-N-butyl phthalate.* Agency for Toxic Substances and Disease Registry, Atlanta, GA. 2001. Accessed online at: http://www.atsdr.cdc.gov/toxprofiles/tp135.html

FDA. Medical Devices Made With Polyvinylchloride (PVC) Using the Plasticizer di-(2-Ethylhexyl)phthalate (DEHP); Draft Guidance for Industry and FDA. Accessed online at: http://www.fda.gov/cdrh/ode/guidance/1407.html

FDA. Safety Assessment of Di(2-ethylhexyl) phthalate (DEHP) Released from PVC Medical Devices. Accessed online at: http://www.fda.gov/cdrh/ost/dehp-pvc.pdf

HEAVY METALS

CDC's National Exposure Report includes data on the body burdens of a number of so-called "heavy" metals. Heavy metals include those metals from periodic table group IIA to VIA, which are characterized by a specific gravity (a measure of density) at least five times that of water. The heavy metals most often linked to human poisoning are lead, mercury, arsenic, and cadmium. Other heavy metals, including copper, zinc, and chromium, are required by the body in small amounts, but can be toxic in larger doses.

For most of the metals included in the study, urine specimens were collected and analyzed for persons 6 years of age and older. However, because of known health effects of lead, cadmium, and mercury, additional populations are included in the study. Young children are highly susceptible to the toxic effects of exposure to these metals, particularly serious psychological and neurological effects. Mercury levels in women of childbearing age are included because they provide an indication of potential fetal exposure levels. The table below summarizes the specific population groups and analyses conducted for lead, cadmium, and mercury.

Heavy Metal	Sample Type	Population/Age
Lead	Blood	$\geq$ 1 year old
	Urine	$\geq$ 6 years old
Cadmium	Blood	$\geq$ 1 year old
	Urine	$\geq$ 6 years old
Mercury	Blood	Children, 1-5 years old Females, 16-49 years old
All other metals	Urine	$\geq$ 6 years old

Sources of Heavy Metals and Routes of Exposure:

Heavy metals have been widely used in industry since the 1800s and as a result are common environmental contaminants. Sources of heavy metals include air emissions from coal-burning plants, smelters, and other industrial facilities (e.g., cadmium and arsenic); waste incinerators (mercury and cadmium); process wastes from mining and industry; pesticides and wood preservatives (e.g., arsenic and chromium); fertilizers (e.g., cadmium is found in phosphate fertilizers); and lead in household plumbing and old house paints. While certain types of industrial facilities are required by the EPA to report their releases of some heavy metals to the Toxics Release Inventory (TRI), other major sources—including power plants and waste incinerators—are not. Consequently, the TRI may significantly under-report actual environmental releases of some metals.

Heavy metals can also enter the environment through natural processes. For example, in some parts of the U.S., naturally occurring geologic deposits of arsenic dissolve into groundwater, potentially resulting in unsafe levels in drinking water supplies. Once released to the environment, metals can remain for decades or centuries, increasing the likelihood of human exposure.

Humans are exposed to heavy metals through inhalation of air pollutants, consumption of contaminated drinking water, exposure to contaminated soils or industrial waste, or consumption of contaminated food. Food sources such as vegetables, grains, fruits, fish, and shellfish can become contaminated by accumulating metals from surrounding soil and water.

Health Effects of Heavy Metals:

Heavy metals cause serious health effects, including reduced growth and development, cancer, organ damage, nervous system damage, and in extreme cases, death. Exposure to some metals, such as mercury and lead, may also cause development of autoimmunity, in which a person's immune system attacks its own cells. This can lead to joint diseases, such as rheumatoid arthritis, and diseases of the kidneys, circulatory system, and nervous system. Metals are particularly toxic to the sensitive rapidly developing systems of the fetus, infants, and young children. Some metals, such as lead and mercury, easily cross the placenta and can damage the fetal brain. Childhood exposure to some metals can result in learning difficulties, memory impairment, damage to the nervous system, and behavioral problems such as aggressiveness and hyperactivity. At higher doses, heavy metals can cause irreversible brain damage. Children may receive higher doses of metals from food than adults, since they consume more food for their body weight than adults. Also, children absorb metals more readily through their intestinal tract than adults.

Sources of Information

Klaassen CD, ed. 1996. Casarett and Doull's Toxicology: The Basic Science of Poisons. New York: McGraw-Hill**.**

World Resources Institute (WRI). *Heavy Metals and Health.* Accessed online at http://www.wri.org/wri/wr-98-99/metals2.htm.

OSHA. *Heavy Metals.* Occupational Safety and Health Administration. Accessed online at http://www.osha-slc.gov/SLTC/metalsheavy/index.html.

Chemical Name: **LEAD**

Summary: Lead is a heavy, soft gray metal with a wide variety of industrial and consumer uses. The EPA classifies lead as a probable human carcinogen.
Lead poisoning is one of the most common environmental health problems in the U.S., with particularly severe effects on young children. Prenatal or childhood exposure to lead can result in long-term damage to the brain and nervous system and behavioral problems lasting into adulthood.

Uses and Sources: Lead has a wide variety of uses due to its properties of high density, softness, low melting point, resistance to corrosion, and ability to stop gamma and x-rays. Lead is commonly used in storage batteries for automobiles and industry; electrical and electronic equipment; machine bearings; cable coverings; and in pipes, traps, solder, and sheets for building construction. In the U.S., lead and zinc processing facilities account for a large portion of lead releases to the environment. These operations and various industrial sources have contributed to widespread environmental contamination by lead. According to EPA's Toxics Release Inventory, reporting industries released more than 370 million pounds of lead and lead compounds into the environment in 2000.

Major Consumer Products: Lead is used for many consumer products, including lead-acid batteries, metal products, ammunition, and ceramic glazes. Use of lead in gasoline was phased out in the U.S. and is now banned for transportation purposes. Lead is also used in some paints, although the amount has been reduced because of health concerns.

Routes of Human Exposure: The risk of lead poisoning is highest for children who live in housing containing lead-based paint. Many older homes were painted with leaded paint that can release lead dust and chips as it ages. (The lead content of paint was not regulated until 1977.) This lead dust accumulates on window ledges, floors, and other surfaces, where children come into contact with it while crawling and playing. Through normal hand-to-mouth activity, children can then ingest this lead-contaminated dust. Some children with pica, a behavior of eating non-food items, can also be exposed to lead by eating contaminated paint chips.

Exposure to lead can also result from breathing contaminated air, contacting lead-contaminated soils, or drinking contaminated water. Lead gasoline additives, smelters, and battery plants are the most significant contributors to lead emissions into the atmosphere. Tap water contamination with lead occurs when water (especially acidic water) passes through older pipes containing lead, lead solder, or brass fixtures that contain lead.

Health Effects: The health effects of lead exposure have been extensively studied. When lead enters the body, it travels through the blood to the soft tissues, such as brain, liver, and kidneys. In adults, most lead taken into the body is excreted, with a small amount stored in bones and teeth, where it can accumulate with repeated exposure. Chronic exposure to lead can cause a variety of adverse health effects in adults, including brain and kidney damage, poor reaction time, joint weakness, anemia (a disorder of the blood),

memory impairment, and possibly increased blood pressure. These effects can persist long after lead exposure ends. Lead can be released from the bones into blood under certain circumstances, such as during pregnancy and breastfeeding. These releases can potentially expose the developing fetus or nursing infant. Lead is classified by EPA as a probable human carcinogen, based on evidence from rodent studies. Large doses of lead have caused tumors in rats and mice. There is not yet enough information to determine whether lead causes cancer in humans.

Children are highly sensitive to the effects of lead largely because lead affects the child's developing nervous system. Children absorb and retain more lead than adults (in proportion to their body weight), and most of the lead that enters a child's body remains stored in the body. Because lead accumulates in the body faster than it is excreted, repeated exposure to small amounts of lead can result in poisoning. Prenatal exposure has been linked to a variety of effects in the infant, including premature birth, low birth weight, decreased mental ability, learning difficulties, and slow growth. Childhood exposure to lead has been associated with anemia, learning disabilities, attention deficits, lower intelligence scores, slowed or stunted growth, and effects on kidney function—effects that can persist as the child grows older. Lead exposure has also been associated with behavioral effects such as impulsiveness and aggression.

Detecting Exposure: Lead is one of the few chemicals for which health effects can be directly linked to specific measured levels in the blood. Measuring the amount of lead in the blood has become a common way of determining the amount of lead in the body and for screening children for lead poisoning. The CDC considers children to have an elevated level of lead if the amount in the blood is 10 micrograms per deciliter (µg/dL) or higher. However recent information suggests that effects on the nervous system and developmental effects in children can occur at blood lead levels as low as 2.5 µg/dL. CDC's first National Exposure Report (2001) reported geometric mean blood lead levels of 2 µg/dL in children ages 1-5 years, and 1.6 µg/dL for all age groups. The geometric mean of urine lead concentration across all age groups was 0.80 µg/L.

Regulations for Protection of Human Health:

Air – EPA regulates lead as a "criteria air pollutant" under the Clean Air Act, and has designated lead as a hazardous air pollutant. Lead emissions are controlled for various industrial operations and in motor vehicle emissions. EPA limits concentrations of lead in the air to 1.5 micrograms of lead per cubic meter of air (1.5 $\mu g/m^3$).

Drinking Water – EPA has established an "action level" of 0.015 mg/L for lead in drinking water. Action levels are not enforceable standards; rather, they are intended to serve as guidance for public health decision-making. Water utilities are required to replace lead service lines when more than 10% of homes exceed the action level (if corrosion cannot be controlled).

Dust and Soil – To help protect children from lead poisoning, EPA has established new standards to identify dangerous levels of lead in paint, dust, and contaminated soil. These

standards provide specific targets that can be used in cleaning up lead found in homes, yards, businesses, schools, and play areas. The standards are: 40 micrograms of lead in dust per square foot on floors; 250 micrograms of lead in dust per square food on interior window sills; and 400 parts per million of lead in bare soil in children's play areas.

Lead-Based Paints – To prevent exposure to lead-based paints, the Consumer Product Safety Commission limits the amount of lead in most paints to less than 0.06% lead.

Food – FDA has set action levels of 1.5 ppm lead in crustacea and 1.7 ppm in clams, oysters, and mussels.

Occupational Exposure – OSHA has established a PEL of 0.050 mg/m^3 for lead in workplace air.

Sources of Information:

ATSDR. *Toxicological Profile for Lead (Update).* Agency for Toxic Substances and Disease Registry, Atlanta, GA. 1999. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp13.html.

ATSDR *Case Studies in Environmental Medicine. Lead Toxicity.* U.S. Department of Health and Human Services, Atlanta, GA. 2000. Accessed online at http://www.atsdr.cdc.gov/HEC/CSEM/lead/index.html.

EPA. 2000 Toxics Release Inventory (TRI) Public Data Release Report. Office of Environmental Information, Washington, DC. EPA-260-R-02-003. Accessed online at http://www.epa.gov/tri/tridata/tri00/pdr/index.htm.

EPA. National Primary Drinking Water Regulations, Consumer Factsheet on Lead. Office of Groundwater and Drinking Water, Washington, DC. 1998. Accessed online at www.epa.gov/OGWDW/dwh/c-ioc/lead.html.

EPA Integrated Risk Information System. Lead and compounds (inorganic). National Center for Environmental Assessment, Washington, DC. Accessed online at http://www.epa.gov/iris/index.html.

National Library of Medicine (NLM). TOXNET Database, Hazardous Substances Databank (HSDB). National Institutes of Health, Bethesda, MD. Accessed online at http://toxnet.nlm.nih.gov/.

Physicians for Social Responsibility. "Drinking water fact sheet #8, Lead: What health care providers should know."

Chemical Name: MERCURY

Summary: Mercury is a silvery, heavy, odorless liquid best known for its use in fever thermometers. This persistent heavy metal occurs in elemental form and in various organic forms, most notably, methylmercury. Environmental contamination with mercury is widespread as a result of industrial activity, particularly fossil fuel combustion. Highly toxic methylmercury accumulates in many edible fish species, which are a major source of mercury exposure in the U.S. Exposures by pregnant women are of particular concern, because methylmercury can cross the placenta and enter the fetal brain. Children exposed to even low levels of mercury before birth can experience serious neurological and development deficits.

Uses and Sources: Mercury is used to make a variety of chemicals, industrial equipment, and consumer products. Most of the mercury associated with human activity is released to the air from fossil fuel power plants. Mercury is also released into the environment by mining, smelting, and waste incineration. According to EPA's Toxics Release Inventory, reporting industries released more than 4 million pounds of mercury and mercury compounds into the environment in 2000.

Major Consumer Products: In addition to thermometers, mercury is found in barometers, batteries, vapor lamps, mirror coatings, dental amalgams, and various medical devices.

Routes of Human Exposure: Metallic and inorganic mercury are the most common forms found in the environment. Mercury released to the atmosphere can be deposited onto soil or into waterways, where biological processes can change inorganic mercury to organic forms. The most common organic form of mercury is methylmercury. Methylmercury is of particular concern because it is more toxic than other forms of mercury. It tends to accumulate in plants, animals, fish, and in the human body. People who eat certain types of fish, such as tuna and swordfish, may be exposed to higher levels of methylmercury. In addition to fish consumption, exposure to methylmercury can result from drinking contaminated water, breathing contaminated air, or through skin contact with contaminated soil or water. In the body, methylmercury is rapidly absorbed and moves into the brain. Mercury in the mother's body can be passed to the fetus, where it moves into the fetal brain. Once mercury enters the human body, it can remain for months.

Health Effects: The developing fetus and young children are most sensitive to the effects of mercury exposure, even at low levels. The developing nervous system and brain are highly sensitive to the effects of mercury in any form, but particularly to methylmercury and mercury vapors. Maternal exposure to organic mercury may lead to brain damage, mental retardation, poor coordination, speech difficulties, and other serious effects on the fetus and newborn. Children born to women exposed to methylmercury have shown a variety of abnormalities including delays in development and learning ability. Recent epidemiological studies found that children exposed to low levels of mercury before birth experienced subtle effects on motor skills, learning capacity, memory, and other

symptoms of neurological damage. The National Research Council (NRC) has estimated that neurobehavioral effects in the fetus could occur at methylmercury levels as low as 58 ppb in cord blood. The NRC cautions that children of women who consume large amounts of fish and seafood during pregnancy are at particular risk. Methylmercury has also shown potential to affect the cardiovascular system. In adults, chronic exposure to either organic or inorganic mercury can permanently damage the brain and kidneys. Exposure to metallic or organic mercury may affect the brain, resulting in effects such as personality changes, tremors, vision problems, poor muscle coordination, and memory loss.

Animal studies have shown that long-term exposure to inorganic mercury salts can cause kidney damage; effects on the nervous system and immune system; effects on blood pressure and heart rate; and effects on the stomach. Long-term animal studies have also found that exposure to organic mercury at high levels can cause nervous system damage; damage to the kidneys, stomach, and large intestine; changes in blood pressure and heart rate; adverse effects on the developing fetus, sperm, and male reproductive organs; and increases in the number of spontaneous abortions and stillbirths.

EPA has classified methylmercury and mercuric chloride as possible human carcinogens.

Detecting Exposure: Blood and urine are commonly used to measure recent exposure to mercury. Hair is a biomarker of long-term exposure to methylmercury. CDC's first National Exposure Report (2001) reported geometric means of mercury in blood of 0.3 µg/L for children ages 1- 5 years and 1.2 µg/L for women ages 16-49 years. Of the women tested by CDC, approximately 10% were found to have mercury levels very close (within one-tenth) to the level of concern (58 µg/L) reported by the National Research Council.

Regulations for Protection of Human Health:

Air – Mercury is regulated by the EPA as a Hazardous Air Pollutant under the Clean Air Act. EPA has indicated that it intends to begin regulating mercury emissions from power plants, although these regulations have not yet been developed.

Drinking Water – EPA has established an MCL of 0.002 mg/L for inorganic mercury in drinking water. The FDA allows no more than 2 mg/L of mercury in bottled water.

Food – In January 2001, EPA and FDA issued advisories cautioning that both commercially available and non-commercial fish may be contaminated with mercury. The FDA advised that pregnant women, nursing mothers, women of childbearing age, and young children should not to each shark, swordfish, king mackerel, and tilefish because they may contain potentially high levels of methylmercury. EPA recommended that women who are pregnant or may become pregnant, nursing mothers and young children limit consumption of non-commercial fish to one meal per week.

The FDA has set a maximum allowable level of 1 ppm methylmercury for all fish.

At least 40 states have issued fish advisories warning pregnant women or women of reproductive age to limit or avoid consuming freshwater fish that may be contaminated with mercury. Each state, Native American tribe, or U.S. Territory establishes its own criteria for issuing fish and wildlife advisories.

Occupational Exposure – OSHA has established a PEL of 0.1 mg/m^3 for mercury in workplace air.

Sources of Information:

ATSDR. *Toxicological Profile for Mercury*. Agency for Toxic Substances and Disease Registry, Atlanta, GA. 1999. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp46.html.

EPA. Mercury Study Report to Congress. Volume V: Health Effects of Mercury and Mercury Compounds. Office of Air Quality Planning and Standards; Office of Research and Development. December 1997. Accessed online at http://www.epa.gov/ttn/oarpg/t3/reports/merover.pdf

EPA. 2000 Toxics Release Inventory (TRI) Public Data Release Report. Office of Environmental Information, Washington, DC. EPA-260-R-02-003. Accessed online at http://www.epa.gov/tri/tridata/tri00/pdr/index.htm.

National Research Council. *Toxicological Effects of Methylmercury*. National Academy Press, Washington, DC. 2000.

Physicians for Social Responsibility (PSR). "In Harm's Way: Toxic Threats to Child Development". Greater Boston PSR. 2000.

Chemical Name: **CADMIUM**

Summary: Cadmium is a heavy metal that occurs naturally in the environment. It also has many industrial uses, which can result in its release to air, water, and land. Cadmium accumulates in the body and can cause serious health effects such as lung disease, kidney damage, and lung cancer. Exposure to cadmium, even at low levels, may also have serious effects on the fetus, infants, and young children. This heavy metal is classified as a known human carcinogen, but few human studies have been conducted to assess other effects of cadmium at low levels of exposure.

Uses and Sources: Cadmium is a naturally occurring element, used for a variety of purposes including silver solder; metal plating; pigments in plastics, ceramics and glass; nickel-cadmium batteries; electronic devices; PVC stabilizers; coatings on steel and non-ferrous metals; components of specialized alloys; and as a catalyst. The largest sources of cadmium to the environment are fossil fuel emissions and incineration of municipal wastes. Cadmium is also released in smelter emissions. According to EPA's Toxics Release Inventory, reporting industries released more than 9 million pounds of cadmium and related compounds into the environment in 2000.

Major Consumer Products: Cadmium is used in nickel-cadmium (Ni-CD) batteries, pigments, and plastics.

Routes of Human Exposure: Because of cadmium's strong tendency to accumulate in plants and other living organisms, consumption of contaminated food is the primary non-occupational route of exposure to cadmium. People can also be exposed to cadmium by drinking contaminated water, by breathing contaminated air (including cigarette smoke), or by swallowing small quantities of contaminated soil or dust. Cigarette smokers can have approximately twice the cadmium in their bodies as non-smokers. Cadmium released into the air by industry, incinerators, smelters, and fossil fuel emissions is deposited onto soils or surface water where it tends to accumulate in plants (including food crops), fish, and shellfish. Cadmium is also a contaminant in sewage sludge from industrial sources. Crops grown on soils treated with sewage sludge can take up cadmium, increasing the potential for human exposure.

Health Effects: Once cadmium enters the body, it accumulates in soft tissues, primarily the liver and kidneys. Exposure to cadmium over time can result in accumulation of the metal in the body, which can have significant health significance for children. Human studies have shown that chronic exposure to cadmium can lead to serious health effects including lung cancer, emphysema and other lung diseases, and kidney damage. Limited evidence suggests that maternal exposure to cadmium can cause lower birth weight in infants. Psychological and neurological effects have been observed in children exposed to low levels of cadmium. Acute exposure to cadmium is rare, but can cause severe irritation of the stomach (by ingestion) and the lungs (by inhalation).

The U.S. National Toxicology Program now classifies cadmium and cadmium compounds as known human carcinogens based on evidence of lung and prostate cancer in humans.

Animal studies have shown that cadmium affects development, including low fetal weight, skeletal malformations, changes in fetal metabolism, and behavioral and neurological effects. Decreased reproduction and testicular damage have also been observed in animals exposed to cadmium. Other adverse effects shown in chronic animal studies include effects on the kidneys, liver, lungs, bone, immune system, blood and nervous system. There is little evidence for many of these effects in humans. It is important to note, however, that few human studies have assessed the effects of cadmium at low levels of exposure.

Detecting Exposure: Cadmium exposure can be measured by its presence in blood or urine. The amount of cadmium in blood is a good indicator of recent exposure, whereas the amount of cadmium in urine is more representative of total body burden. CDC's first National Exposure Report (2001) found that blood levels of cadmium were below the analytical detection limit for many participants. In urine samples, CDC reported a geometric mean urinary cadmium level of 0.32 µg/L in persons 6 years of age and older.

Regulations for Protection of Human Health:

Air – Cadmium compounds are regulated by EPA as Hazardous Air Pollutants under the Clean Air Act.

Drinking Water – EPA has established an MCL of 0.005 mg/L for cadmium in drinking water.

Food – FDA has set action levels of 3 ppm cadmium in crustacea and 4 ppm in clams, oysters, and mussels. FDA also limits cadmium in food colors and ceramic dishware.

Occupational Exposure – OSHA has established a PEL-TWA of 0.005 mg/m^3 (5 $\mu g/m^3$) for cadmium in workplace air.

Sources of Information:

ATSDR. *Toxicological Profile for Cadmium (Update).* Agency for Toxic Substances and Disease Registry, Atlanta, GA. 1999. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp5.html.

California Environmental Protection Agency (Cal/EPA). "Evidence on Developmental and Reproductive Toxicity of Cadmium." October 1996. Accessed online at http://www.oehha.org/prop65/pdf/CD-HID.pdf.

EPA. 2000 Toxics Release Inventory (TRI) Public Data Release Report. Office of Environmental Information, Washington, DC. EPA-260-R-02-003. Accessed online at http://www.epa.gov/tri/tridata/tri00/pdr/index.htm.

EPA. Technology Transfer Network Air Toxics Website, Health Effects Notebook: Cadmium and Compounds. Washington, DC. December 1994. Accessed online at http://www.epa.gov/ttn/atw/hapindex.html.

National Toxicology Program (NTP). Ninth Report on Carcinogens: Cadmium and Cadmium Compounds. U.S. Department of Health and Human Services, Research Triangle Park, NC. (2001).

Chemical Name: ANTIMONY

Summary: Antimony is a metal that is released into the environment during mining operations and from industrial discharges. Exposure to antimony is associated with damage to the heart, lungs, and other organs. There is limited evidence that chronic exposure can cause developmental and reproductive effects.

Uses and Sources: Antimony is used in alloys in the metals industry and to produce fireproofing chemicals, ceramics, ammunition, glassware, pigments, and other items. The primary source of antimony releases to the environment is mining and processing of gold ores. According to EPA's Toxic Release Inventory, reporting industries released almost 30 million pounds of antimony and related compounds into the environment in 2000.

Major Consumer Products: Antimony is used in a variety of products, including storage batteries, pewter, textiles, plastics, paints, ceramics, and enamels.

Routes of Human Exposure: People can be exposed to antimony by breathing contaminated air, drinking contaminated water, or by eating foods that contain this metal. Exposure can also occur through skin contact with contaminated soil or water. Industrial workers can be exposed to antimony by breathing dust or by skin contact.

Health Effects: Chronic exposure to antimony can cause lung and heart disease, increased blood pressure, stomach pain, diarrhea, vomiting, and stomach ulcers. There is limited evidence that antimony can cause developmental and reproductive effects. Animal studies have shown that long-term exposure to antimony can cause heart problems, effects on fertility, and lung cancer. EPA has not determined the potential for antimony to cause cancer in humans. One form of antimony (antimony trioxide) has been identified as a possible human carcinogen by the International Agency for Research on Cancer (IARC).

Detecting Exposure: Antimony is absorbed slowly in the body and can concentrate in liver, lungs, plasma, or red blood cells. It can be measured in the urine, feces, and blood for several days after exposure. CDC's first National Exposure Report (2001) reported a geometric mean urinary antimony level of 0.10 µg/L in persons 6 years of age and older.

Regulations for Protection of Human Health:

Air – Antimony compounds are regulated by EPA as Hazardous Air Pollutants under the Clean Air Act.

Drinking Water - EPA has established an MCL of 0.006 mg/L for antimony in drinking water.

Occupational Exposure – OSHA has established a PEL of 0.5 mg/m^3 for antimony in workplace air.

Sources of Information:

ATSDR. *Toxicological Profile for Antimony and Compounds.* Agency for Toxic Substances and Disease Registry, Atlanta, GA. 1992. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp23.html.

EPA. Integrated Risk Information System (IRIS): Antimony. Office of Research and Development, Washington, DC. Accessed online at http://www.epa.gov/iris/index.html.

EPA. 2000 Toxics Release Inventory (TRI) Public Data Release Report. Office of Environmental Information, Washington, DC. EPA-260-R-02-003. Accessed online at http://www.epa.gov/tri/tridata/tri00/pdr/index.htm.

Klaassen CD, ed. 1996. Casarett and Doull's Toxicology: The Basic Science of Poisons. New York: McGraw-Hill.

Chemical Name: BARIUM

Summary: Barium is a metal commonly found in soils, foods, surface water, and in public drinking water supplies. Barium is also released into the environment from industrial facilities. Exposure to barium can cause a variety of health effects, including damage to the liver, kidney, heart, and spleen. Exposure to barium can also cause difficulty breathing, muscle weakness, and effects on the nervous system.

Uses and Sources: Barium occurs naturally and is commonly found in soils, surface water, and public drinking water supplies. Barium compounds are used in a wide variety of materials including stabilizers for plastics, drilling tools for the oil and gas industry, case hardening steels, paints, bricks, tiles, lubricating oils, and jet fuels. Some barium compounds are used to make insect and rat poisons. Barium sulfate is used by doctors to perform medical tests and taking certain x-rays of the gastrointestinal tract. In the environment, barium typically combines with other elements. It can be taken up into edible plants from the soil. Barium is also discharged into surface water and air by industrial sources. According to EPA's Toxics Release Inventory, reporting industries released over 310 million pounds of barium and barium compounds into the environment in 2000.

Major Consumer Products: Used as contrast material in medical x-ray diagnostics.

Routes of Human Exposure: Barium is released into the environment by natural processes, industrial and manufacturing operations, and combustion of materials such as plastics, paints, oils, and jet fuels. The general population can be exposed by consuming contaminated drinking water or food or by inhaling barium in ambient air.

Health Effects: The health effects of barium depend, in part, on which form of the chemical enters the body. In general, ingestion of barium can cause difficulty breathing, increased blood pressure, changes in heart rhythm, stomach irritation, muscle weakness, changes in nerve reflexes, and damage to the liver, kidney, heart, and spleen. No reproductive or developmental effects have been linked to barium exposure, and EPA has concluded that barium is unlikely to cause cancer in humans by the ingestion route. There is inadequate data to evaluate the potential carcinogenicity of barium by inhalation.

Detecting Exposure: Barium leaves the body primarily in the feces, but can also be found in urine. Most barium leaves the body within a few days, and almost all is gone within 1-2 weeks. Barium remaining in the body is found in the bones (91%), with the remainder stored in soft tissues such as brain, heart, kidneys, and lungs. CDC's first National Exposure Report (2001) reported a geometric mean urinary barium level of 1.6 µg/L in persons 6 years of age and older.

Regulations for Protection of Human Health:

Air – Barium is not regulated in ambient air.

Drinking Water – EPA has established an MCL of 2 mg/L for barium in drinking water.

Occupational Exposure – OSHA has established a PEL of 0.5 mg/m^3 for soluble barium compounds and 15 mg/m^3 for barium sulfate dust in workplace air.

Sources of Information:

ATSDR. *Toxicological Profile for Barium.* Agency for Toxic Substances and Disease Registry, Atlanta, GA. 1992. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp24.html.

EPA. 2000 Toxics Release Inventory (TRI) Public Data Release Report. Office of Environmental Information, Washington, DC. EPA-260-R-02-003. Accessed online at http://www.epa.gov/tri/tridata/tri00/pdr/index.htm.

EPA. Toxicological Review of Barium and Compounds. March 1998. Accessed online at http://www.epa.gov/iris/toxreviews/0010-tr.pdf.

Chemical Name: **BERYLLIUM**

Summary: Beryllium is a hard, grayish metal that occurs in certain rocks, coal and oil, soil, and volcanic dust. It enters the environment through natural processes as well as through mining operations and industrial releases. Inhaled beryllium can remain deposited in the lungs for long periods and can cause lung disease, lung cancer, and effects on the heart. Beryllium can also affect immune system function.

Uses and Sources: Most beryllium ore is used in alloys to make electrical and electronic parts, construction materials, and molds for plastics. Pure beryllium metal is used in nuclear weapons and reactors, aircraft and space vehicles, instruments, and x-ray machines. Beryllium occurs naturally, and can enter water sources from dissolved rocks or soil. Beryllium is also discharged into surface water and air from mining and ore processing facilities and industrial sources. Most beryllium in the atmosphere is from combustion of coal and oil. According to EPA's Toxics Release Inventory, reporting industries released over 940,000 pounds of beryllium and beryllium compounds into the environment in 2000.

Major Consumer Products: No major consumer products.

Routes of Human Exposure: People can be exposed to beryllium by breathing contaminated air, consuming food, or drinking water that contains beryllium. Cities have higher levels of beryllium in air, because it is released from burning coal and fuel oil. Beryllium can be found in drinking water supplies, but is not a common contaminant. It also occurs naturally in some foods. Workers involved with beryllium mining, processing, or other use can be exposed to higher-than-normal levels.

Health Effects: Inhalation exposure to beryllium has been associated with lung cancer in animals, and beryllium is likely to cause cancer in humans. Some forms of beryllium, when inhaled, can cause lung disease and effects on the heart. Animal studies have found gastrointestinal ulcers and effects on the kidneys. Exposure to beryllium can also affect immune system function. There is insufficient information to determine whether beryllium causes reproductive or developmental effects in humans.

Detecting Exposure: Most beryllium that is swallowed leaves the body within a few days through the feces without entering the bloodstream. Inhaled beryllium can be deposited in the lungs and can remain in the body for months to years. Beryllium can be measured in the urine and blood, but these levels may not accurately reflect the amount of exposure. In CDC's first National Exposure Report (2001), beryllium was not detected in urine of study participants (persons ages 6 and older).

Regulations for Protection of Human Health:

Air – Beryllium compounds are regulated by EPA as Hazardous Air Pollutants under the Clean Air Act.

Drinking Water – EPA has established an MCL of 0.004 mg/L for beryllium in drinking water.

Occupational Exposure – OSHA has established a PEL-TWA of 2 $\mu g/m^3$ for beryllium and beryllium compounds in workplace air.

Sources of Information:

ATSDR. *Toxicological Profile for Beryllium (Update).* Agency for Toxic Substances and Disease Registry, Atlanta, GA. September 2000. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp4.html.

EPA. 2000 Toxics Release Inventory (TRI) Public Data Release Report. Office of Environmental Information, Washington, DC. EPA-260-R-02-003. Accessed online at http://www.epa.gov/tri/tridata/tri00/pdr/index.htm.

Chemical Name: **CESIUM**

Summary: Cesium is a soft, malleable, silver-white metal that naturally occurs in rocks, soil, and clay. For most people, ingestion of contaminated food is the most significant source of exposure to cesium. Naturally occurring cesium is not radioactive and has low toxicity to humans. However, radioactive forms of cesium, such as produced by nuclear power plants and nuclear weapon explosions, can cause serious health effects. In humans, radioactive cesium has been shown to cause reduced fertility. Animal studies have indicated potential for neurological, developmental, reproductive, genotoxic, and cancer-causing effects.

Uses and Sources: Because cesium is not mined in the U.S., domestic use is dependent on imports. Only small quantities of cesium are used in the U.S., primarily in research and development and in electronic, photoelectric, and medical applications. Most cesium in the environment is non-radioactive, and comes from natural erosion and weathering of rocks and minerals. Radioactive cesium is released into the environment during operation of nuclear power plants, explosions of nuclear weapons (especially atmospheric testing carried out in the past), and nuclear accidents. Cesium is also released in the ash of hazardous waste incinerators and coal-burning power plants. Some radioactive isotopes of cesium break down very slowly, and can remain in the environment for decades after release.

Major Consumer Products: No major consumer products.

Routes of Human Exposure: Although detailed exposure studies have not been conducted, researchers believe that people are exposed to relatively low levels of cesium through food, drinking water, air, and by skin contact with cesium in soil. Ingestion of cesium-contaminated food items is the most significant source of exposure to both naturally occurring and radioactive cesium. Cesium can travel long distances in the atmosphere, and be deposited onto land, water, and plant surfaces far from the original source. Cesium can be absorbed by vegetation, which is then consumed by animals and humans. Radioactive cesium has been detected in many types of food, including breast milk and pasteurized milk. People working in industries that process or use cesium, including workers in the nuclear power industry, may have above-average levels of exposure.

Health Effects: Radioactive cesium can seriously affect health, particularly following external exposure and eating contaminated food. Radioactive cesium emits beta particles and gamma rays, which can damage living cells. Reduced fertility has been observed in men exposed to radioisotopes of cesium. Animal studies have shown evidence of adverse neurological, developmental, reproductive, genotoxic, and cancer-causing effects following exposure to radioactive cesium. Long-term studies of cancer risk for humans exposed to radioactive cesium have not been conducted, and EPA has not classified cesium for human carcinogenicity. However, animal studies have suggested an increased potential for cancer, particularly at high doses.

Detecting Exposure: Urinalysis is the preferred test for analysis of cesium, although cesium can also be measured in blood, feces, and other tissues. Unlike some radioactive substances, cesium does not accumulate in any one part of the body. CDC's first National Exposure Report (2001) reported a geometric mean urinary cesium level of 4.7 µg/L in persons 6 years of age and older.

Regulations for Protection of Human Health:

Air – EPA does not regulate cesium in ambient air. The Nuclear Regulatory Commission (NRC) limits releases of cesium from commercial nuclear power plants to 2×10^{-10} µCi/mL.

Drinking Water – EPA has not established a drinking water standard or health advisory for cesium.

Occupational Exposure – NIOSH has established a Recommended Exposure Limit (REL) for cesium hydroxide of 2 mg/m^3, as a time-weighted average for up to a 10-hour workday. The ACGIH has recommended a TLV of 2 mg/m^3 for cesium hydroxide over an 8-hour workday. The NRC regulates occupational inhalation exposure to radioactive cesium.

Source of Information:

ATSDR. *Draft Toxicological Profile for Cesium*. Agency for Toxic Substances and Disease Registry, Atlanta, GA. 2001. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp157.html

Chemical Name: COBALT

Summary: Cobalt is a relatively rare, naturally occurring metal that is used in a variety of products and industries. There is one stable (non-radioactive) form of cobalt (Co-59), and many radioactive forms. Cobalt can enter the environment naturally or through human activity. In small quantities, stable cobalt is essential for health, but in higher doses, it can have serious health effects such as lung disease and possibly effects on the heart and other organs. Radioactive cobalt is known to cause cancer, and exposure can damage organ systems throughout the body. The developing fetus is particularly sensitive to the effects of radioactive cobalt.

Uses and Sources: Small amounts of cobalt occur naturally in rocks, soil, water, plants, and animals, usually combined with other elements. Cobalt is not mined in the U.S., but is obtained from imported materials and scrap metal recycling. Cobalt is used in the military and industry; the largest use is in superalloys used for aircraft engines. Cobalt metal is also used to dry paint and porcelain enamel, in colored pigments, and as catalysts in the petroleum industry. Radioactive isotopes of cobalt, generated by nuclear reactions, are used in medical and research applications, consumer products, and for food irradiation. Human activity contributes to environmental cobalt contamination through combustion of fossil fuels, sewage sludge, fertilizers, mining, smelting, and processing of cobalt. Once released into the environment, cobalt is not destroyed, but radioactive isotopes undergo decay. According to EPA's Toxics Release Inventory, reporting industries released more than 17 million pounds of cobalt and cobalt compounds in 2000.

Major Consumer Products: Cobalt is used in artificial replacement joints.

Routes of Human Exposure: For most people, food is the most important source of cobalt exposure. Exposure to cobalt can also occur by breathing contaminated air, drinking water, and contact with soil or other materials containing cobalt. Cobalt is found in tea, coffee, many fruits and vegetables, and in some fish. The general public is not exposed to significant levels of radioactive cobalt. Workers involved in processing or working in industries using cobalt may have higher-than-average exposure to stable cobalt and workers at nuclear facilities, irradiation facilities, and nuclear waste storage sites may be exposed to radioactive forms of cobalt.

Health Effects: Cobalt is a component of vitamin B_{12} and is an essential nutrient in very small quantities. At high levels of exposure, such as in the workplace, cobalt can cause serious health effects in humans. By inhalation, cobalt exposure can cause serious effects on the lungs, cardiovascular system, liver, kidney, and eyes. Stable cobalt has not been shown to cause cancer in humans, but animal studies suggest it may have carcinogenic potential.

Exposure to radioactive forms of cobalt can damage the lungs, gastrointestinal system, bone marrow, nervous system, and reproductive system. Radioactive cobalt is classified by EPA as a known human carcinogen. Animal studies have found that the developing fetus is particularly sensitive to the effects of exposure to radioactive cobalt.

Detecting Exposure: Analysis of urine and feces are the preferred samples for analysis of cobalt. CDC's first National Exposure Report (2001) reported a geometric mean urinary cobalt level of 0.36 µg/L in persons 6 years of age and older.

Regulations for Protection of Human Health:

Air – Cobalt compounds are regulated by EPA as Hazardous Air Pollutants under the Clean Air Act. Emissions of radioactive cobalt into air are regulated by the Department of Energy and the Nuclear Regulatory Commission.

Drinking Water – EPA has not established a drinking water standard or health advisory for stable cobalt. EPA has developed relevant standards for radioactive cobalt in drinking water. The MCL for particle and photon activity is 4 millirems/yr, and the MCL for gross alpha particle activity is 15 picoCuries per liter (pCi/L).

Occupational Exposure – OSHA has established a PEL of 0.1 mg/m^3 for cobalt in workplace air.

Sources of Information:

ATSDR. *Draft Toxicological Profile for Cobalt*. Agency for Toxic Substances and Disease Registry, Atlanta, GA. September 2001. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp33.html.

EPA. 2000 Toxics Release Inventory (TRI) Public Data Release Report. Office of Environmental Information, Washington, DC. EPA-260-R-02-003. Accessed online at http://www.epa.gov/tri/tridata/tri00/pdr/index.htm.

Klaassen CD, ed. 1996. Casarett and Doull's Toxicology: The Basic Science of Poisons. New York: McGraw-Hill.

Chemical Name: MOLYBDENUM

Summary: Molybdenum is a silvery-white metal that is an essential human nutrient in very small doses. There is limited information available regarding human health effects of exposure to excess molybdenum. Animal studies have shown potential for serious effects, including reproductive effects, organ failure, and skeletal abnormalities.

Uses and Sources: Molybdenum is used in some nickel-based alloys, which are heat-and corrosion-resistant. It is also used in electrical applications, as a catalyst in petroleum refining, in nuclear energy production, and for missile and aircraft parts. Molybdenum is recovered as a byproduct of tungsten and copper mining. Molybdenum can be released to the environment from mining/processing operations and through industrial discharges. Molybdenum releases into the environment are not tracked under EPA's Toxics Release Inventory.

Major Consumer Products: No major consumer products.

Routes of Human Exposure: Exposure to molybdenum is highest in persons involved in production or manufacture of molybdenum products. Molybdenum can also be found in ambient air, drinking water, and naturally in certain foods (soy products, grains, and nuts).

Health Effects: Small quantities of molybdenum are required for normal body functions. Molybdenum compounds have relatively low toxicity, although there are limited data for human toxicity. In laboratory animals, molybdenum exposure has caused weight loss, kidney failure, skeletal abnormalities, infertility, and other effects. The relevance of these findings to humans is uncertain. Reproductive effects have also been observed in animals exposed to molybdenum. There is no evidence that molybdenum causes cancer in humans.

Detecting Exposure: Excess molybdenum is rapidly excreted in the urine. CDC's first National Exposure Report (2001) reported a geometric mean urinary molybdenum level of 48.4 µg/L in persons 6 years of age and older.

Regulations for Protection of Human Health:

Air – Molybdenum is not regulated in ambient air.

Drinking Water – EPA has not established an MCL for molybdenum in drinking water. EPA has developed a health advisory for children of 0.08 mg/L and a lifetime health advisory of 0.04 mg/L of molybdenum in drinking water.

Occupational Exposure – OSHA has established a PEL of 5 mg/m^3 for soluble molybdenum compounds and 15 mg/m^3 for insoluble compounds in workplace air.

Sources of Information:

National Academy of Sciences. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. Institute of Medicine. National Academy Press, Washington, DC. (2001).

National Library of Medicine. TOXNET, Hazardous Substances Data Bank (HSDB). Accessed online at http://www.toxnet.nlm.nih. gov/cgi-bin/sis/htmlgen?HSDB.

Chemical Name: **PLATINUM**

Summary: Platinum is a naturally occurring silver-gray, malleable metal. In metallic form, platinum has low toxicity. Platinum dusts and complexes can have potentially serious health effects, including neuromuscular toxicity, kidney toxicity, and cancer.

Uses and Sources: Platinum occurs in nature both as pure metal and combined with other metals. Platinum is used in jewelry, in electrical applications, as a catalyst in industrial and chemistry applications, as an anti-tumor agent, and in prosthetic and biomedical devices. Platinum releases into the environment are not tracked under EPA's Toxics Release Inventory.

Major Consumer Products: Jewelry, prosthetic devices.

Routes of Human Exposure: Potential for exposure to platinum is highest for individuals involved in mining and processing operations and in industrial operations using platinum. Exposure can occur by breathing platinum dusts.

Health Effects: Platinum is relatively non-toxic in the metallic state. Exposure to platinum dust can cause breathing difficulty, and platinum salts can cause neuromuscular toxicity and kidney toxicity. Animal studies have shown that platinum can cross the placenta and reach the fetus. Some platinum complexes may cause cancer, but there is no evidence of increased cancer risk from occupational exposure to platinum compounds.

Detecting Exposure: Animal studies have found that almost all ingested platinum is readily excreted in the urine and feces. In CDC's first National Exposure Report (2001), platinum levels were below the analytical detection limit.

Regulations for Protection of Human Health:

Air – Platinum is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for platinum.

Occupational Exposure – OSHA has established a PEL of 0.002 mg/m^3 for soluble platinum salts in workplace air.

Sources of Information:

Klaassen CD, ed. 1996. Casarett and Doull's Toxicology: The Basic Science of Poisons. New York: McGraw-Hill.

National Library of Medicine. TOXNET, Hazardous Substances Data Bank (HSDB). Available online at Accessed online at http://www.toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

Chemical Name: THALLIUM

Summary: Thallium is a naturally occurring metal that is used in a variety of industrial applications. Little is known about the effects of long-term, low-level exposure to thallium. There is evidence from animal studies that thallium exposure can cause organ damage and may have toxic effects on the reproductive system.

Uses and Sources: Thallium is used in the manufacture of electronic devices, switches, and closures. Thallium is no longer produced in the U.S., but is imported. Thallium occurs naturally in the environment, primarily as salts. In addition to natural sources, thallium is released into the atmosphere from coal-burning power plants, cement factories, and smelters. Thallium in the air can deposit onto soil and surface water, potentially contaminating food and drinking water sources. According to EPA's Toxics Release Inventory, reporting industries released more than 5 million pounds of thallium and thallium compounds into the environment in 2000.

Major Consumer Products: No major consumer products.

Routes of Human Exposure: The primary source of exposure to thallium is consumption of fruits and vegetables that are grown in thallium-contaminated soil (including home-grown produce). Cigarette smoking is also a source of thallium exposure. People who work in power plants, cement factories, and smelters can be exposed to thallium.

Health Effects: Exposure to thallium in large amounts can damage the nervous system, lungs, heart, liver and kidneys. Symptoms of exposure to high doses include vomiting, diarrhea, and possibly death. Little is known about the human health effects of long-term, low-level exposure to thallium. Animal studies have found reproductive effects (damage to testes) after drinking small quantities of thallium-contaminated water. There is insufficient information to determine whether thallium causes cancer in humans.

Detecting Exposure: Thallium is rapidly absorbed and distributed in the body (primarily the kidneys and liver). It leaves the body slowly, and can be found in urine as long as 2 months after exposure. CDC's first National Exposure Report (2001) reported a geometric mean urinary thallium level of 0.19 µg/L in persons 6 years of age and older.

Regulations for Protection of Human Health:

Air – Thallium is not regulated in ambient air.

Drinking Water – EPA has established an MCL of 0.002 mg/L for thallium in drinking water.

Occupational Exposure – OSHA has established a PEL of 0.1 mg/m^3 for thallium in workplace air.

Sources of Information:

ATSDR *Toxicological Profile for Thallium*. Agency for Toxic Substances and Disease Registry, Atlanta, GA. 1992. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp54.html.

EPA. 2000 Toxics Release Inventory (TRI) Public Data Release Report. Office of Environmental Information, Washington, DC. EPA-260-R-02-003. Accessed online at http://www.epa.gov/tri/tridata/tri00/pdr/index.htm.

Chemical Name: **TUNGSTEN**

Summary: Pure tungsten is a pure-white to tin-gray metal. Very little is known about the health effects of tungsten, but there is evidence that tungsten may cause lung damage.

Uses and Sources: Most of the tungsten used in the U.S. is for cemented carbide parts for metalworking, oil and gas drilling, mining, and construction industries. Tungsten is also used in lamp filaments, electrodes, and other electrical and electronics components; in steels and alloys; and as chemicals for catalysts and pigments. Tungsten can be released into the environment through mining operations and through industrial releases to air, water, or land. Tungsten releases into the environment are not tracked under EPA's Toxics Release Inventory.

Major Consumer Products: No major consumer products.

Routes of Human Exposure: Individuals working in tungsten mining and processing or in industries using tungsten, have a higher exposure potential than the general public. The primary route of exposure is by inhalation.

Health Effects: There is very little information available regarding the toxicity of tungsten. Tungsten carbide is toxic by the inhalation route, and chronic exposure by this route can cause lung damage. Symptoms of exposure to tungsten carbide include wheezing, coughing, difficulty breathing, and cardiac failure. EPA has not evaluated the potential carcinogenicity of tungsten.

Detecting Exposure: Tungsten levels can be measured in urine. CDC's first National Exposure Report (2001) reported a geometric mean urinary tungsten level of 0.10 µg/L in persons 6 years of age and older.

Regulations for Protection of Human Health:

Air – Tungsten is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for tungsten.

Occupational Exposure – OSHA has not established a standard for tungsten in workplace air.

Sources of Information:

National Toxicology Program (NTP). Chemical Repository: Tungsten Carbide. August 1991. Radian Corp. Accessed online at http://ntp-db.niehs.nih.gov/NTP_Reports/NTP_Chem_H&S/NTP_Chem1/Radian12070-12-1.txt.

Chemical Name: **URANIUM**

Summary: Uranium is a naturally occurring, radioactive heavy metal. Although uranium is radioactive, the weak radiation cannot penetrate the skin to enter the body. Exposure occurs by ingesting uranium in food or water or by breathing contaminated air. Available studies have shown potential for serious health effects from exposure to uranium. These include kidney disease, heart and lung damage, developmental effects, and possibly cancer.

Uses and Sources: The primary uses of uranium are in nuclear power plants, on helicopters and airplanes, as shielding to protect Army tanks, in nuclear weapons, and in ammunition. Small quantities of uranium are also used to make some ceramic glazes, light bulbs, photographic chemicals, and household products. It is found in low levels in rocks, soil, surface water and groundwater, air, and plants and animals. Uranium can also enter the environment from human activity, including uranium mining and processing; use in nuclear energy and nuclear weapons; phosphate fertilizers; or accidental releases. Uranium releases into the environment are not tracked under EPA's Toxics Release Inventory.

Major Consumer Products: No major consumer products.

Routes of Human Exposure: People can be exposed to uranium from air, water, food, and soil. Root vegetables, such as beets and potatoes, tend to take up more uranium from soil than other foods. People who work in industries where uranium is processed or used can be exposed to higher-than-normal levels of this element.

Health Effects: Kidney disease has been observed in people and animals exposed to large amounts of uranium. Animal studies suggest that the lung and heart can be damaged from exposure to uranium. There is insufficient evidence to determine whether uranium causes cancer in humans. However, it is known that long-term exposure to radioactive substances can cause cancer. It is not known whether uranium causes reproductive effects in humans. Animal studies have shown reduced sperm counts in animals exposed to high levels of uranium. There is evidence that uranium can have adverse effects on the developing fetus. Studies of animals exposed to uranium have found higher levels of fetal death, reduced growth, smaller litter sizes, and birth defects.

Detecting Exposure: When uranium is inhaled, a portion remains in the lungs. Most ingested uranium, from food or water, leaves the body in the urine and feces within a few days. Some uranium can remain in the bones, kidneys, or other soft tissues where it can remain for years.

Regulations for Protection of Human Health:

Air – EPA regulates uranium releases into ambient air from a variety of industrial sources. For more information, contact EPA or visit the EPA Office of Air and Radiation website at http://www.epa.gov/oar/.

Drinking Water – EPA has established an MCL of 30 µg/L for uranium in drinking water.

Occupational Exposure – OSHA has established a PEL of 0.05 mg/m^3 for soluble uranium compounds and 0.25 mg/m^3 for insoluble compounds in workplace air.

Source of Information:

ATSDR. *Toxicological Profile for Uranium (Update).* Agency for Toxic Substances and Disease Registry, Atlanta, GA, 1999. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp150.html

Pesticides and Pest Repellents

ORGANOCHLORINE PESTICIDES

Organochlorine insecticides were, at one time, the most heavily used pesticides in the world. In the U.S., they were widely used from the 1940s to the 1960s for agricultural pest control and for control of insect-borne diseases such as malaria. After years of heavy use, however, evidence began to emerge that these highly persistent pesticides were causing widespread ecological damage and global contamination, and target insects were becoming resistant. EPA subsequently banned the use of almost all organochlorine pesticides in the U.S. Yet DDT and other organochlorine pesticides are still used in other countries and can be transported long distances in the environment. Continued use of organochlorine pesticides in other countries adds to the global threat posed by these chemicals. The Stockholm Convention, when implemented, will require ratifying countries to ban the production and use of aldrin, dieldrin, endrin, chlordane, heptachlor, hexachlorobenzene, and mirex. DDT use is to be phased out eventually, but for the foreseeable future countries that need it will be allowed to use it to control disease-carrying insects such as malarial mosquitoes.

Although their levels are declining, organochlorine pesticides remain ubiquitous in the environment and in the human population years after being banned. They are still found in the food supply, including fish, produce, and even in breast milk. Biomonitoring studies continue to find DDT and other organochlorine pesticides in blood, adipose tissue, and breast milk of people around the world. Body burdens have declined since these chemicals were banned, yet virtually the entire population still carries detectable levels of these toxic compounds.

Effects of Chronic Exposure to Organochlorine Insecticides

Chronic exposure to even low levels of organochlorine pesticides can cause a wide range of serious harmful effects in humans. Organochlorine pesticides are highly toxic to the nervous system, particularly during early stages of development. Prenatal exposure to pesticides such as DDT has been associated with neurological effects such as learning deficits and behavioral changes in infants. Organochlorine pesticides have been linked with many forms of cancer. Animal studies have shown the potential for reproductive and developmental effects and disruption of normal hormone function.

Children may be especially vulnerable to pesticide exposure because they consume larger amounts of food and water relative to their body weight, than adults. Children's developing organ systems are more sensitive, and their bodies have a limited ability to detoxify pesticides.

CDC has added the following organochlorine insecticides to the list of chemicals addressed in the National Exposure Report:

Hexachlorobenzene (HCB)
Beta-hexachlorocyclohexane
Gamma-hexachlorocyclohexane (Lindane)

DDT/DDD/DDE
Chlordane (reported as metabolite oxychlordane)
Trans-nonachlor
Heptachlor epoxide
Mirex
Pentachlorophenol
2,3,5-trichlorophenol
2,4,6-trichlorophenol

This packet includes abbreviated chemical profiles for four of these organochlorine pesticides: DDT, chlordane, lindane, and pentachlorophenol.

Sources of Information:

ATSDR has published detailed toxicological profiles for many of the organochlorine insecticides included in CDC's National Exposure Report. Readers interested in more detailed information are referred to the following publications:

ATSDR. *Toxicological Profile for Hexachlorobenzene*. Agency for Toxic Substances and Disease Registry, Atlanta, GA. 2002. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp90.html.

- *Toxicological Profile for Hexachlorocyclohexanes (HCH)*, 1994. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp43.html

- *Toxicological Profile for DDT, DDE and DDD.* 2002. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp35.html

- *Toxicological Profile for Heptachlor and Heptachlor Epoxide*. 1993. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp12.html

- *Toxicological Profile for Mirex and Chlordecone*. 1995. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp66.html

- *Toxicological Profile for Pentachlorophenol.* 2001. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp51.html

- *Toxicological Profile for Chlorophenols*. 1999. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp107.html

Chemical Name: **DDT**

Summary: The organochlorine pesticide DDT has been banned from use in the U.S. and most other nations because of its potential for human toxicity and severe ecological effects. Although DDT has not been used in the U.S. for decades, it is still found as a contaminant because of its extreme persistence and high mobility in the environment. The most common route of DDT exposure is through the diet, particularly fatty foods such as fish, meat, and dairy products. DDT is known to adversely affect the nervous system, and there is particular concern that DDT and its metabolites may interfere with normal reproduction and development as a result of its endocrine-disrupting properties. EPA has classified DDT and two of its metabolites, DDE and DDD, as probable human carcinogens.

Uses and Sources: DDT is an organochlorine insecticide that was commonly used for control of disease-bearing insects and on a variety of food crops in the U.S., until it was banned in 1972 due to concerns over health and environmental impacts. DDT is still sprayed indoors in some developing countries to control mosquitoes that carry malaria. In the environment and in the body, DDT breaks down into DDE and DDD over time. Because of the large quantities once used, and because these compounds are extremely persistent, they are still found in the environment decades after being banned. Their continued use in some countries further contributes to worldwide environmental contamination and can accumulate to high levels in soil, sediment, plants, animals, fish and humans. About two dozen countries still use DDT for malaria vector control, but the Stockholm Convention will phase out public use of DDT. Under the terms of the treaty, countries can still claim exemptions to use DDT where necessary for public health.

Major Consumer Products: DDT is no longer used in the U.S.

Routes of Human Exposure: For the general public, most exposure to DDT and its metabolites occurs by consumption of contaminated food. Measurable amounts of these compounds are found in many foods, but are highest in meat, fish, poultry, and dairy products. In FDA's most recent Total Diet Study, DDT was the most frequently detected pesticide in food, occurring in 21% of samples. DDT in the mother's body can cross the placenta and enter the fetus. Nursing infants can also be exposed to DDT through breast milk. Because of their extreme persistence, these compounds are likely to be found in the food supply for decades to come. Persons who consume large quantities of freshwater fish for many years can have higher-than-average exposure to DDT, DDE, and DDD. Indigenous people in the Arctic are highly exposed to DDT, because they derive a large portion of their diet from fish and marine mammals. People eating large quantities of Great Lakes fish have also been found to have higher DDT levels than the general population.

Health Effects: As with other organochlorine pesticides, DDT can have serious adverse effects on the nervous system. DDT can adversely affect reproduction and development, probably due to its disruption of normal hormone function. In humans, high levels of DDT have shown endocrine-disrupting effects on lactation, fertility, and fetal

development. Animal studies have also raised concerns that exposure to DDT early in life may cause neurobehavioral problems later in life. Few studies have addressed the effects of long-term, low-level exposure to DDT in humans, but concerns exist because of the potential for DDT to interfere with hormone function, which influences growth, development, and functioning of tissues and systems throughout the body. Animal studies have determined that even low levels of exposure to endocrine-disrupting chemicals by the fetus can lead to severe effects on development of the reproductive system. EPA has classified DDT, DDE, and DDD as probable human carcinogens. There is limited evidence from animal studies that DDT and its metabolites may also affect the liver and impair the immune system.

Detecting Exposure: Most of the DDT, DDD, and DDE entering the body is stored in fatty tissue, where it can persist for decades. They leave the body very slowly, primarily through the urine. Analysis of blood and urine are the most common methods for detecting DDT exposure, although DDT can also be measured in fatty tissues and breast milk. DDE has the shortest biological half-life, followed by DDT then DDD.

Regulations for Protection of Human Health:

Air – DDE is regulated by EPA as a Hazardous Air Pollutant under the Clean Air Act.

Drinking Water – EPA has not established an MCL for DDT/DDE/DDD in drinking water.

Food – EPA has established action levels ranging from 0.05 to 5 ppm for DDT/DDE/DDD in various fruits, vegetables, grains, and meats. FDA has set an action level of 5 ppm for DDT and DDE in all fish.

Occupational Exposure – OSHA has established a PEL of 1 mg/m^3 for DDT in workplace air.

Sources of Information:

ATSDR. *Toxicological Profile for DDT, DDE and DDD.* Agency for Toxic Substances and Disease Registry, Atlanta, GA. 2002. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp35.html

FDA. Food and Drug Administration Pesticide Program: Residue Monitoring 2000. Center for Food Safety and Applied Nutrition. 2002. Accessed online at http://www.cfsan.fda.gov/~dms/pes00rep.html.

Chemical Name: CHLORDANE

Summary: Chlordane is an organochlorine pesticide, commonly used in the past to kill insects on crops and for termite control. Chlordane does not readily break down in the environment, and it bioaccumulates in fatty tissues of animals and fish. The most common route of exposure to chlordane is consumption of contaminated foods, although people living in homes where chlordane was used to kill termites can also be exposed. Chlordane exposure has been linked to toxic effects on the nervous system, digestive system, and liver. EPA has classified chlordane as a probable human carcinogen.

Uses and Sources: Chlordane was commonly used in the U.S. to control termites around buildings and to treat agricultural crops, home lawns, and gardens for insects. EPA canceled all uses of the pesticide in 1988 due to health concerns, but chlordane is still produced in the U.S. for export. According to EPA's Toxics Release Inventory, reporting industries released almost 10,000 pounds of chlordane into the environment in 2000.

Major Consumer Products: None.

Routes of Human Exposure: Chlordane is a highly persistent compound, and can travel long distances in the atmosphere. These characteristics, combined with the widespread past use of chlordane in the U.S. and current use in other countries, have resulted in people's continued exposure to this pesticide worldwide. Chlordane is extremely persistent in soil and aquatic systems and accumulates in edible crops, fish, and animals. Although dietary exposures to chlordane have decreased since its use on crops was banned in 1978, this remains the most common source of exposure. Chlordane residues are also stored in fatty tissues of the body and have been found in breast milk. People living in homes treated with chlordane for termite control can also be exposed, as chlordane gradually vaporizes from the treated soil around the foundations of homes into the air of the house. Disturbing the soil can also result in dermal contact and absorption through the skin. Chlordane, if not properly disposed at waste sites, can migrate into surrounding water, air, and soil.

Health Effects: Long-term exposures to low levels of chlordane have caused effects on the nervous system and are likely to cause gastrointestinal symptoms. Chlordane exposure has also been linked to toxic effects on the digestive system and the liver, and may impair immune function. Animal studies demonstrate that the liver is a particular target organ of chlordane toxicity. Although high levels of chlordane exposure in animal studies have shown reproductive effects, no data link the chemical to reproductive or developmental effects in humans. Chlordane has been shown to cause cancer in laboratory animals and has been implicated in some cases of human cancer. EPA has classified chlordane as a probable human carcinogen.

Detecting Exposure: Chlordane and its metabolites can be measured in blood, serum, adipose (fat) tissue, breast milk, and body tissues. Technical chlordane is a composite of numerous organochlorines, so analytical tests measure the presence of these components

(principally *cis*- and *trans*-chlordane, *cis*- and *trans*-nonachlor) and their metabolites (principally oxychlordane).

Regulations for Protection of Human Health:

Air – Chlordane is regulated by EPA as a Hazardous Air Pollutant under the Clean Air Act.

Drinking water – EPA has established an MCL of 0.002 mg/L for chlordane in drinking water.

Food – EPA has established a tolerance of 1 ppm for residues of chlordane in fruits and vegetables. FDA has set an action of level of 0.3 ppm for chlordane residues in rendered animal fat and edible portions of fish.

Occupational exposure – OSHA has established a PEL of 0.5 mg/m^3 for chlordane in workplace air.

Sources of Information:

ATSDR. *Toxicological Profile for Chlordane*. Agency for Toxic Substances and Disease Registry, Atlanta, GA. 1994. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp31.html

Chemical Name: LINDANE (gamma-hexachlorocyclohexane)

Summary: Lindane is an organochlorine pesticide, with a very limited number of products registered for use in the U.S. A few products containing lindane are still used to treat seeds prior to planting and as a pharmaceutical agent for control of head lice and scabies. Because of its persistence and widespread use in the past, lindane still contaminates the environment, including the human food supply. Dietary exposure is the most common route of lindane exposure for most people, but workers involved in manufacturing, packaging, and application of this pesticide can be more highly exposed. Lindane exposure can cause a wide range of adverse health effects in humans, including neurological effects, liver toxicity, and reproductive and developmental effects. There is evidence, although inconclusive, that lindane may cause cancer in humans.

Uses and Sources: Lindane is a restricted use pesticide used to treat seeds (barley, corn, oats, rye, sorghum, and wheat) before planting. Each year, approximately 233,000 pounds of lindane are used for seed treatment in the U.S. EPA has recently imposed additional restrictions on re-registration of lindane-containing seed treatment products, including requirements for new tolerances to reduce residues in raw agricultural products grown from treated seeds. Lindane is also approved by the FDA for medicinal use in control of head lice and scabies in humans. Most uses of lindane were canceled by 1983 (i.e., use on food crops, ornamental plants, livestock, and home uses) but because of its past use and extreme persistence, lindane is still found in the environment. Lindane has no residential uses.

Major Consumer Products: Prescription shampoos and other treatments for control of scabies and lice.

Routes of Human Exposure: The primary route of exposure to lindane is through the food supply. Lindane is highly persistent in the environment and can remain in soil and sediment for extended periods, accumulating in plants, fish, and animals consumed by humans. When used for lice and scabies treatment, lindane is absorbed through the skin. This exposure route may be more significant for children, who are more likely to be treated for these pests than adults. Workers involved in lindane manufacture and pesticide application can experience higher-than-average exposures to lindane.

Health Effects: A variety of adverse health effects have been associated with exposure to lindane. Studies of occupational and accidental exposures have shown that exposure to high doses can cause symptoms such as vomiting, nausea, diarrhea, muscle weakness, and seizures, as well as neurological effects, blood disorders, liver toxicity, and immune deficiencies. Studies have also shown possible associations between lindane exposure in pregnant women and increased risk of spontaneous abortion and premature delivery. Animal studies have suggested that lindane exposure may adversely affect fetal development. Farmers using lindane were found to have higher risk for non-Hodgkin's lymphoma. Animal studies suggest that lindane may cause liver cancer.

Detecting Exposure: Lindane is rapidly broken down and excreted from the body. Lindane and its metabolites can be measured in serum, urine, and other body fluids and tissues.

Regulations for Protection of Human Health:

Air – Hexachlorocyclohexane (all isomers, including lindane) is regulated by EPA as a Hazardous Air Pollutant under the Clean Air Act.

Drinking water – EPA has established an MCL of 0.0002 mg/L for lindane in drinking water.

Food – EPA has established tolerances ranging from 0.01 to 7 ppm for lindane residues in a variety of foods and agricultural commodities.

Occupational Exposure – OSHA has established a PEL of 0.5 mg/m^3 for lindane in workplace air.

Sources of Information:

ATSDR. *Toxicological Profile for Alpha-, Beta-, Gamma- and Delta-Hexachlorocyclohexane*. Agency for Toxic Substances and Disease Registry, Atlanta, GA. 1999. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp43.html

EPA. Lindane Reregistration Eligibility Decision (RED) Document. Office of Prevention, Pesticides and Toxic Substances, Washington, DC. 2002. Accessed online at http://www.epa.gov/oppsrrd1/REDs/lindane_red.pdf

Chemical Name: PENTACHLOROPHENOL

Summary: Pentachlorophenol (PCP) is a synthetic organochlorine chemical once commonly used for wood treatment and pest eradication. Inhalation and dermal absorption are the major routes of exposure, as the amount of PCP found in food and water has declined since the chemical became a restricted-use pesticide. Chronic, low level exposure to PCP can damage the nervous system, kidneys, liver, and blood. EPA has classified PCP as a probable human carcinogen. Because of its toxicity, exposure to PCP should be avoided, particularly in children.

Uses and Sources: PCP is primarily used as a wood preservative, commonly used on utility poles, fence posts, and other wood products. In the past, PCP was one of the most widely used pesticides in the U.S. (including uses as an insecticide [for termites], fungicide, herbicide, disinfectant, and other applications), but it was reclassified by EPA as a restricted use pesticide in 1984. PCP is no longer registered for uses in and around the home. According to EPA's Toxics Release Inventory, reporting industries released almost 4,000 pounds of PCP into the environment in 2000. As a result of the manufacturing process, commercial grade PCP is contaminated with highly toxic polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans.

Major Consumer Products: PCP is a restricted use pesticide and is not available for use by the general public.

Routes of Human Exposure: Soil, air, and water are all contaminated with PCP. Runoff from wood treatment facilities, improperly disposed wastes, and evaporation from treated wood release significant amounts of PCP into the local environment.

Small amounts of PCP have been widely detected in human urine, blood, and tissues. Exposures come from the inhalation of contaminated indoor or outdoor air, ingestion of contaminated food or water, or direct dermal contact with treated wood. Future indoor air contamination will be limited because of the restriction of PCP-treated wood in residential and workplace buildings. General population exposure usually comes from the air, water, and soil surrounding hazardous waste sites or wood-treatment facilities. Worker exposure can be very significant as the workplace air can contain large amounts of PCP.

Health Effects: PCP passes very quickly through the body with most of the absorbed chemical eliminated in the urine before being metabolized. The fraction that is not eliminated is distributed to the liver, kidneys, brain, lungs, blood, and fat tissues.

Brief exposures to large amounts of PCP caused by unintended releases in the workplace can cause harm to the liver, kidneys, blood, lungs, nervous system, immune system, and gastrointestinal tract. Long-term exposure to low levels of the chemical, commonly found in normal working conditions in wood-treatment facilities, can cause damage to the endocrine, immune, and respiratory systems. Animal studies of chronic exposure also indicate that PCP can adversely affect the blood, liver, and kidneys. The EPA has

classified PCP as a probable human carcinogen based on the results of increased cancer incidence in exposed animals and weaker evidence in humans. Evidence exists that young children are more susceptible to PCP exposure than adults.

Detecting Exposure: PCP can be measured in blood, urine, and adipose tissue but most is excreted in the urine. PCP is not metabolized to a significant extent and can be measured directly in urine. However, PCP in body fluids is not a specific biomarker for PCP exposure, because other compounds such as lindane and hexachlorobenzene may also be metabolized into PCP.

Regulations for Protection of Human Health:

Air – PCP is regulated by EPA as a Hazardous Air Pollutant under the Clean Air Act.

Drinking water – EPA has established an MCL of 0.001 mg/L for PCP in drinking water.

Occupational exposure – OSHA has established a PEL of 0.5 mg/m^3 for PCP in workplace air.

Source of Information:

ATSDR. *Toxicological Profile for PCP*. Agency for Toxic Substances and Disease Registry, Atlanta, GA. 2001. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp51.html

NON-PERSISTENT PESTICIDES

The first CDC National Exposure Report included analyses of the class of non-persistent pesticides called *organophosphate insecticides*. This year, CDC has expanded their analysis to also include the closely related class of carbamate pesticides. These chemicals are called "non-persistent pesticides" because they break down fairly rapidly in the environment (within days or weeks), reducing their potential to accumulate in the tissues of plants, animals, or humans. Organophosphate and carbamate pesticides are widely used in agriculture, in the home, and in the garden. These pesticides are effective in killing insects because they interfere with the normal transmission of nerve impulses. More specifically, they cause important chemical transmitters to build up at the nerve endings and ultimately cause death from over-stimulation of the body by the nervous system. Organophosphate and carbamate insecticides affect the nervous systems of humans through a similar mechanism. At high doses, these insecticides cause loss of coordination, inability to control muscle movement, difficulty breathing, convulsions, and even death.

Because organophosphate and carbamate insecticides affect the body through similar mechanisms, their health effects are considered to be "additive." This means that the toxicity of the combination of two or more chemicals is equal to the sum of the toxicity of each individual chemical.

Exposure to organophosphate pesticides is assessed by measuring levels of several different breakdown products. The CDC study measures levels of the following six metabolites:

- dimethylphosphate (DMP);
- diethylphosphate (DEP);
- dimethylphosphorothioate (DMTP);
- diethylphosphorothioate (DEPT);
- dimethylphosphorodithioate (DMDPT); and
- diethylphosphorodithioate (DEDTP).

The degradation of most organophosphate pesticides produces only these six metabolites. Detection of DMP and DEP has been found to be directly attributable to pesticide exposure. Detection of DMDPT and DEDTP is difficult since they are rapidly degraded and are less directly associated with pesticide exposure. DMPT and DEPT can be produced from other non-pesticide sources, limiting their usefulness as markers for pesticide absorption.

Effects of Chronic Exposure to Organophosphate and Carbamate Insecticides

Recent studies have found that home environments throughout the U.S. are commonly contaminated with pesticides, including non-persistent pesticides. Pesticide residues from applications around the home, as well as residues on food and in drinking water can lead to long-term, low-level exposure to these chemicals. Chronic exposures to organophosphates and carbamates can cause a wide range of health effects, depending on the amount of pesticides one

is exposed to. Effects on the reproductive and hormonal systems of the body can result from exposure to some pesticides. Home pesticide use has been found to be a risk factor for development of Parkinson's disease. Children may be especially vulnerable to the adverse health effects of organophosphate and carbamate pesticides. Children consume larger quantities of water and food, pound for pound, than adults. Therefore, children are exposed to higher concentrations of pesticides relative to their body weight. Children may also have higher exposure to pesticides because they are in close contact with floors, lawns, and other surfaces where pesticides are often applied and because of their tendency to put their hands in their mouths. Furthermore, children's developing organ systems are more sensitive, and their bodies have a limited ability to detoxify pesticides.

There is evidence that exposure to some organophosphate insecticides can cause developmental abnormalities in the fetus and developing child. One study of the effects of chronic low-level exposure of children to pesticides found that they had reduced stamina, poor coordination, and impaired memory when compared to children who were not exposed. Evidence from rodent studies has shown that low-level chronic exposure may affect growth and nervous system function and development. Animal studies have shown that exposure by the fetus to organophosphate insecticides can cause permanent chemical changes in the brain, behavioral problems such as hyperactivity, poor coordination, and slowed growth. There is also evidence that fetal exposure to organophosphates may lead to increased risk of childhood brain tumors, leukemia, and other cancers.

Human and animal studies have suggested that long-term, low-level exposure to carbamates can also cause adverse health effects, including nervous system and neuromuscular effects, and persistent neurobehavioral changes. There is limited evidence that some carbamates may cause adverse developmental and reproductive effects, immune effects, genetic mutations, and possibly cancer.

Sources of Information

Eskenazi, B. et al. Exposures of children to organophosphate pesticides and their potential adverse health effects. *Environmental Health Perspectives* 107 (Suppl. 3):409-419.(1999).

EXTOXNET's Toxicology Information Briefs: Cholinesterase Inhibition. Sept., 1993. Accessed online at http://ace.orst.edu/info/extoxnet/tibs/cholines.htm.

Klaassen CD, ed. 1996. Casarett and Doull's Toxicology: The Basic Science of Poisons. New York: McGraw-Hill.

Physicians for Social Responsibility. "Drinking water fact sheet #5, Pesticides: What health care providers should know."

CARBAMATE PESTICIDES

Summary: Carbamates are a class of pesticides widely used in agricultural crop production for control of insects, fungus, and weeds. They are also found in some pesticide formulations for home and garden use. They are not highly persistent in the environment, but can be highly toxic to humans. Carbamates, like the organophosphate pesticides, are cholinesterase inhibitors and can cause serious neurological effects. There is also limited evidence that chronic exposure to carbamates may cause reproductive and developmental damage, and possibly cancer. Because of their common use on food crops, people are exposed to low levels of carbamates through the diet. Exposure can also occur during or following use of these products in and around the home, or by consumption of contamination drinking water.

Uses and Sources: Over 30 carbamate pesticides are currently registered by the U.S.EPA for pest control in homes, gardens and agriculture. Most carbamates belong to the class called N-methylcarbamates and are used as insecticides. Common pesticides in this group include carbaryl (Sevin), aldicarb (Temik), carbofuran, fenoxycarb, propoxur (Baygon), and methomyl. Other forms of carbamates include thiocarbamates (primarily herbicides) and dithiocarbamates (primarily fungicides). A number of carbamates are restricted use pesticides, which means that they can be purchased and used only by certified applicators. The carbamate pesticides metam-sodium and ethyl dipropylthiocarbamate (EPTC) are among the most heavily used agricultural pesticides in the U.S. Carbaryl is one of the most commonly used carbamate insecticides for home and garden applications. For these heavily used pesticides, the table below provides estimates of the quantities used (from EPA surveys), as well as environmental releases of each pesticide by industrial sources (based on EPA's Toxics Release Inventory for 2000).

Pesticide	**Estimate Annual Use** (pounds/yr)	**Industrial Releases** (pounds/yr)
Metam-sodium	53 – 58 million (Agriculture)	9, 417
EPTC	7 – 10 million (Agriculture)	11,010
Carbaryl	1 – 3 million (Home and garden)	8,679

Major Consumer Products: Carbamates are found in certain pesticide products used in and around the home for control of insects on lawns, gardens, and around building foundations. Examples include propoxur (Baygon) and methiocarb.

Routes of Human Exposure: For most people, the primary route of exposure to carbamate pesticides is by consumption of fruits, vegetables, and other agricultural products that have been treated with carbamate pesticides. In FDA's most recent analysis of baby foods (fruit juices, fruits, and grains), carbaryl was a frequently detected pesticide, found in 18% of all samples–second only to DDT. People can also be exposed to carbaryl during use of carbamate-containing products in and around the home and by contact with treated lawns and other surfaces. Well water can also be contaminated with carbamate pesticides, particularly in agricultural regions. Agricultural workers and

pesticide applicators are particularly at risk of exposure to carbamates, primarily by inhalation and skin absorption.

Health Effects: Carbamate pesticides appear to exert their toxicity in a manner similar to the organophosphates, (i.e., by inhibiting cholinesterase enzymes). This inhibition causes overstimulation of the nervous system, but in the case of carbamates, the inhibition is reversible. Because they act in similar ways in the body, persons exposed to both carbamates and organophosphates can be at increased risk of adverse health effects. Carbamates vary widely in their toxicity, but some can be highly toxic, including aldicarb, methiocarb, methomyl and propoxur. The symptoms of acute carbamate poisoning are similar to those for organophosphates and can include malaise, muscle weakness, dizziness, tremors, sweating, headache nausea, vomiting. More severe cases of carbamate poisoning can lead to coma and death. Acute poisoning by carbamates is generally of shorter duration than organophosphates because carbamates are more rapidly metabolized by the body.

There is evidence from human and animal studies, although limited, that long-term, low-level exposure to carbamates can cause adverse health effects, including nervous system and neuromuscular effects, and persistent neurobehavioral changes. There is limited evidence that some carbamates may cause adverse developmental and reproductive effects, immune effects, genetic mutations, and possibly cancer.

Detecting Exposure: Carbamates are quickly metabolized and excreted from the body in the urine and feces. The CDC National Exposure Report included measurements of three metabolites of carbamate pesticides. 1-Naphthol is the metabolite of carbaryl; 2-Isopropoxyphenol of propoxur; and carbofuranphenol of carbofuran.

Regulations for Protection of Human Health:

Air – Carbaryl and propoxur are regulated by EPA as Hazardous Air Pollutants under the Clean Air Act.

Drinking water – EPA has developed MCLs for the following carbamate pesticides:
- Carbofuran: 0.04 mg/L
- Oxamyl: 0.2 mg/L

Food – EPA has established tolerances for specific carbamate pesticides on a wide variety of food products. Chemical-specific and crop-specific tolerances can be found at http://www.epa.gov/pesticides/food/viewtols.htm.

Occupational exposure – OSHA has established PELs for the following carbamate pesticides:
- Carbaryl: 5 mg/m^3
- Carbofuran: 0.1 mg/m^3
- Methomyl: 2.5 mg/m^3
- Propoxur: 0.5 mg/m^3

Sources of Information:

Krieger, RI and J Doull. Handbook of pesticide toxicology, 2nd ed. Chapter 52: Carbamate Insecticides. Academic Press, San Diego, London. 2001.

U.S. EPA. Pesticide Industry Sales and Usage: 1996 and 1997 Market Estimates. Office of Prevention, Pesticides and Toxic Substances. Washington, DC. November 1999. Accessed online at http://www.epa.gov/oppbead1/pestsales/.

U.S. FDA. Food and Drug Administration Pesticide Program: Residue Monitoring 2000. Center for Food Safety and Applied Nutrition. May 2002. Accessed online at http://www.cfsan.fda.gov/~dms/pes00rep.html.

ORGANOPHOSPHATE PESTICIDES

Chemical Name: MALATHION

Summary: Malathion is one of the most widely used organophosphate pesticides. It is used in large quantities in both rural and urban areas nationwide. This pesticide is applied to a wide variety of foods and feed crops. It is also used in head lice treatments and for mosquito-borne disease control, including recent control of West Nile encephalitis in the U.S. Severe malathion poisoning can result in respiratory failure and death, while symptoms of chronic exposure include headache, weakness, memory loss, fatigue, poor coordination, tremors, and disorientation.

Uses and Sources: According to the EPA, approximately 16.7 million pounds of malathion active ingredient are used annually nationwide. It is used agriculturally on approximately 100 food crops, including oranges, broccoli, corn, strawberries, and spinach. In addition, it is used on a variety of feed crops, and other non-food crops, such as Christmas trees. Malathion has registered residential uses on lawns and gardens. It is also used for public health mosquito control and in government programs such as the USDA Boll Weevil Eradication Project. Most of the annual usage of malathion is applied to cotton in the boll weevil project (11.2 million pounds).

The national use breakdown for malathion is roughly as follows:

- USDA *(including boll weevil, medfly, grasshopper programs)* 59-61%
- General agriculture *(food and non-food crops)* 16-20%
- Public health *(mosquito control, including for West Nile virus)* 8-15%
- Home and garden 10%

Major Consumer Products: There are many registered malathion products. Some common ones used on gardens are Prentox lawn sprays and Riverdale floral sprays.

Routes of Human Exposure: The most likely routes of exposure to malathion for the general public are through food (pesticide residues), drinking water, and application of malathion in residential and public areas. The risk of exposure is likely to be higher for people who use malathion at home and for toddlers who come into contact with residues after such applications. Exposure can occur through inhalation, absorption through the skin, or accidental ingestion. In the 2000 Total Diet Study conducted by FDA, malathion was one of the most commonly detected pesticides, with residues reported in 18% of all food samples and 12% of baby food samples.

Health Effects: Malathion is a less potent toxicant than many of the other organophosphate insecticides. However, exposure to malathion at high doses can overstimulate the nervous system (through cholinesterase inhibition), potentially causing permanent damage to the nervous and respiratory systems. Severe malathion poisoning can result in respiratory failure and death. Symptoms of chronic exposure to malathion may include headache, weakness, memory loss, fatigue, poor coordination, tremors and disorientation. Behavioral effects such as personality changes, depression, anxiety, or

irritability can also occur from long-term exposure. Animal studies have not found malathion to cause developmental or reproductive toxicity. EPA has classified malathion as having "suggestive evidence of carcinogenicity but not sufficient to assess human carcinogenic potential." Animal studies have shown occurrence of leukemia, as well as tumors of the liver, thyroid gland, testes, and uterus following malathion exposure. However, with the exception of liver tumors, it is not clear whether these tumors are caused by malathion exposure.

Malathion poisoning is most often seen among pesticide workers and in small children who are accidentally exposed. Several studies have shown that farmworkers and their families may be at increased risk of organophosphate toxicity, because they are among the most highly exposed groups. Agricultural workers exposed to malathion over prolonged periods (months) were found to have abnormally low activity levels for several serum enzymes. As with other organophosphate insecticides, children, pregnant women, nursing mothers, and individuals with diseases of the nervous, respiratory, or digestive systems may be at increased risk of health effects from exposure to malathion. Persons who are exposed to other chemicals affecting cholinesterase in the body may also be at increased health risk from exposure to malathion.

Detecting Exposure: Animal studies have shown that malathion is rapidly metabolized and excreted primarily in the urine. It does not appear to accumulate in tissues. The metabolites DMP, DMTP, DMDTP, and malathion dicarboxylic acid are included in the CDC National Exposure Report as breakdown products of malathion.

Regulations for Protection of Human Health:

Air – Malathion is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard for malathion but has developed a health advisory of 0.2 ppm for children and a lifetime health advisory of 0.1 ppm.

Food – EPA has established tolerances ranging from 0.1 to 135 ppm for malathion residues in a variety of foods and agricultural commodities.

Occupational Exposure – OSHA has established a PEL of 15 mg/m^3 for malathion (as total dust) in workplace air.

Sources of Information:

EPA. Human Health Risk Assessment for the Reregistration Eligibility Decision Document: Malathion. 9/22/2000. Accessed online at http://www.epa.gov/pesticides/op/malathion.htm.

EPA. Malathion: The Toxicology Chapter for the RED. April 2000. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/malathion/toxicology.pdf.

Extension Toxicology Network (EXTOXNET). Pesticide Information Profile for Malathion. Accessed online at http://ace.ace.orst.edu/info/extoxnet/pips/malathio.htm

FDA. Food and Drug Administration Pesticide Program: Residue Monitoring 2000. Center for Food Safety and Applied Nutrition. May 2002. Accessed online at http://www.cfsan.fda.gov/~dms/pes00rep.html.

Pesticide Action Network Pesticide Database. Chemical Information about Malathion. San Francisco, CA. 2002. Accessed online at http://www.pesticideinfo.org/PCW/Detail_Chemical.jsp?Rec_Id=PC32924.

Chemical Name: CHLORPYRIFOS

Summary: Chlorpyrifos is an organophosphate insecticide that is highly toxic to the nervous system. Until recently, chlorpyrifos was the most widely used pesticide in U.S. households, where it was commonly involved in poisoning incidents. The EPA recently restricted the residential uses of this pesticide, which is particularly hazardous to children.

Uses and Sources: According to the EPA, approximately 20,960,000 pounds of chlorpyrifos active ingredient are used annually nationwide. Approximately 8,027,000 acres, within the U.S. are treated each year with the insecticide. It is used primarily on agricultural products, such as Brussels sprouts, cranberries, apples, broccoli, and cauliflower. The greatest usage is for corn crops. Non-agricultural uses of chlorpyrifos include termite control and turf applications.

Last year, EPA and manufacturers of chlorpyrifos (also known as Dursban) agreed to phase out most residential uses of this pesticide (home, lawn, and garden), use in schools and parks; and most uses for termite control. EPA will also restrict the use of chlorpyrifos on certain food crops commonly consumed by children (apples, grapes, and tomatoes). Some residential uses and most agricultural uses, however, will not be affected by this decision.

Major Consumer Products: EPA is phasing out most consumer and residential uses of chlorpyrifos. The only products currently registered for consumer use are baits made with child resistant packaging, such as Black Jack ant baits and Raid roach baits.

Routes of Human Exposure: Exposure to chlorpyrifos can occur through use of pesticide products in and around the home or by consuming foods treated with this pesticide. Agricultural workers may also be exposed to chlorpyrifos by breathing vapors or dusts during pesticide application or by pesticide contact with the skin. The potential for exposures to occur in and around the home will be significantly reduced by restrictions on use of chlorpyrifos.

Health Effects: Chlorpyrifos is moderately toxic by ingestion, skin absorption, and inhalation routes of exposure. Chlorpyrifos, like other organophosphate pesticides, has its main effect on the nervous system by acting as a cholinesterase inhibitor. Neurological effects of chlorpyrifos exposure can appear weeks or months after the exposure. Acute exposure to chlorpyrifos can cause symptoms such as dizziness, fatigue, weakness, and nausea. Higher doses can cause breathing difficulty, paralysis, seizures, loss of consciousness and death.

There is very limited data available on effects of chronic, low-level exposures by humans to chlorpyrifos. Chronic exposures in animals have shown effects on the liver, weight loss, and effects on the adrenal glands. Pregnant rats have been found to be especially sensitive to the cholinesterase inhibition effects of chlorpyrifos. Acute exposures by pregnant animals caused adverse effects on the fetus, including bone deformities, lower fetal weight, and delayed or impaired brain development. Chlorpyrifos has also caused

reproductive effects in rats, but only at doses that were toxic to the parents. Chlorpyrifos has shown no evidence of carcinogenicity in animal studies.

Detecting Exposure: Chlorpyrifos is rapidly eliminated from the human body, with most of the pesticide eliminated in the urine within days of exposure. Metabolites of chlorpyrifos exposure that were addressed in the CDC National Exposure Report are DEP and DEPT.

Regulations for Protection of Human Health:

Air – Chlorpyrifos is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard for chlorpyrifos. EPA has issued a health advisory recommending that levels of chlorpyrifos in drinking water should not exceed 20 ppb, based on lifetime exposure. EPA has also issued a health advisory, recommending that levels of chlorpyrifos in drinking water should not exceed 30 ppb for children exposed up to 10 days.

Food – The EPA has set tolerances for allowable residues of chlorpyrifos or on raw agricultural commodities for the pesticide chlorpyrifos. The tolerances vary by crop or food commodity, and range from 13 ppm for alfalfa hay to 0.01 ppm for eggs, figs, whole milk, and sugarcane.

Occupational Exposure – OSHA has not established a standard for chlorpyrifos in workplace air.

Sources of Information:

ATSDR. *Toxicological Profile for Chlorpyrifos*. U.S. Department of Health and Human Services. September 1997. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp84.html

EPA. Chlorpyrifos Revised Risk Assessment. Office of Pesticide Programs. August 2000. Accessed online at http://www.epa.gov/pesticides/op/chlorpyrifos.htm.

EPA. Toxicology Chapter for Chlorpyrifos. April 2000. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/chlorpyrifos/rev_tox.pdf.

EPA. Reregistration Eligibility Decision for Chlorpyrifos. Office of Pesticide Programs. February 2002. Accessed online at http://www.epa.gov/oppsrrd1/REDs/factsheets/chlorpyrifos_fs.htm.

Chemical Name: DIAZINON

Summary: Diazinon is an organophosphate insecticide that is used broadly throughout the U.S. on a wide variety of agricultural crops and livestock. It also controls insects in and around the home and is the most widely used pesticide by homeowners on lawns and gardens. Diazinon is often associated with acute insecticide poisonings, with most incidents occurring in the home or workplace, where pesticides are handled or applied. Longer-term exposure to low levels of diazinon may lead to subtle effects on behavior, memory, and physical coordination. To protect the health of children, EPA recently announced that diazinon will be phased out for all indoor uses and for use on lawns and gardens over the next three years.

Uses and Sources: Diazinon is used on agricultural crops, livestock, and to control soil insects, household insects (such as flies, fleas, and cockroaches), and other pests. According to the EPA's data from 1987 through 1996, roughly 13 million pounds of diazinon active ingredient are used annually nationwide; California, Florida, and Texas have the highest usage rates. Diazinon has been registered to use for indoor and outdoor domestic dwellings for crack and crevice treatment; in pet collars; as domestic and commercial lawn and garden treatment; and for over 75 food crops, such as apples, blueberries, carrots, onions, potatoes, peaches, peas, bananas, and sugarcane. On December 5, 2000, the EPA announced a plan to phase out the use of diazinon for indoor uses by March 2001 and to phase-out the lawn, garden, and turf use by December 2003. At this time, however, it is still legal to purchase and use diazinon.

The national breakdown for diazinon is roughly as follows:

- Homeowner use (outdoor) 39%
- Agricultural uses (food and non-food) 25%
- Professional lawn care companies 19%
- Pest control operators (indoor and outdoor) 11%
- Homeowner use (indoor) and veterinary use 6%

Major Consumer Products: EPA is phasing out consumer uses of products containing diazinon.

Routes of Human Exposure: The impending EPA-mandated phase-out of diazinon in homes is expected to significantly reduce potential exposures to children. However, until the phase-out is complete, people can still use products containing diazinon and may thus come into contact through use in and around the home. Children can be exposed to pesticide residues in the air, on indoor surfaces, and on treated lawns. In addition, this pesticide is still registered for use in agriculture. Workers involved in handling, mixing, and applying diazinon have the highest potential exposure to this pesticide. This use can result in potential human exposure to diazinon residues in certain foods and drinking water. Diazinon has been found in surface and ground water samples, indicating that it can enter drinking water supplies.

Health Effects: Mild symptoms of acute diazinon exposure include headache, dizziness, weakness, and vision difficulty. More severe symptoms include nausea and vomiting, slow pulse, difficulty breathing, and coma. Death has occurred from exposures to high levels of diazinon. Some of these symptoms can occur in people who have been exposed to lower levels of diazinon for long periods. There is also evidence that longer-term exposure to low levels of diazinon by adults or by the fetus may lead to subtle effects on behavior, memory, and physical coordination.

The primary effect of chronic exposure to diazinon is cholinesterase inhibition. Developmental studies in rats and rabbits have shown effects on the fetus only at doses that caused maternal toxicity. Reproductive effects seen in animal studies include decreased mating and fertility, longer gestation, and at highest doses, reduced litter size. Diazinon is classified by EPA as not likely to be carcinogenic in humans and is not believed to be mutagenic.

Detecting Exposure: Diazinon is rapidly metabolized and eliminated from the body through urine and feces. Diazinon has not been shown to accumulate in the body, and almost all of the pesticide is eliminated from the body within 12 days. Because diazinon is rapidly eliminated, analysis of metabolites in urine or feces is useful only in detecting recent exposures. Metabolites of diazinon addressed in the CDC National Exposure Report include DEP and DEPT.

Regulations for Protection of Human Health:

Air – Diazinon is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard for diazinon , but has developed a health advisory of 0.02 ppm for children and a lifetime health advisory of 0.0006 ppm.

Food – EPA has established tolerances ranging from 0.1 to 40 ppm for diazinon residues in a variety of foods and agricultural commodities.

Occupational Exposure – OSHA has not established a standard for diazinon in workplace air.

Sources of Information:

ATSDR. *Toxicological Profile for Diazinon (Update)*. August 1996. U.S. Department of Health and Human Services. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp86.html.

EPA. Human Health Risk Assessment for Diazinon. December 2000. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/diazinon.htm.

EPA. Diazinon: Toxicology Chapter for the RED as revised 3/30/00. Accessed online at http://www.epa.gov/pesticides/op/diazinon/toxicology.pdf.

Extension Toxicology Network (EXTOXNET). Pesticide Information Profile for Diazinon. June 1996. Accessed online at http://ace.ace.orst.edu/info/extoxnet/pips/diazinon.htm.

Chemical Name: **AZINPHOS-METHYL**

Summary: Azinphos-methyl is a highly persistent organophosphate pesticide used to control pests on a wide variety of fruit and vegetable crops and grains. There have been few studies of this pesticide's toxicity to humans, but animal studies suggest that exposure may affect reproduction and the development of the fetus.

Uses and Sources: According to EPA, approximately 2,192,000 pounds of azinphos-methyl are applied annually to 1,813,000 acres of food and other crops nationwide. Azinphos-methyl is classified by EPA as a restricted use pesticide because of its toxicity to humans and animals and is not registered for use in residential settings. EPA has initiated a phase-out of azinphos-methyl for agricultural uses due to potential health risks for agricultural workers. The use of azinphos-methyl on ornamental trees, Christmas trees, forest trees, and shade trees has also been cancelled.

Major Consumer Products: There are no consumer or residential uses for azinphos-methyl.

Routes of Human Exposure: The main route of exposure to azinphos-methyl for the general public is consumption of contaminated foods. In August 1999, EPA announced voluntary measures to reduce dietary and worker risks from this azinphos-methyl. These measures were taken under the Food Quality Protection Act due to unacceptable health risks to children (from dietary and drinking water exposures) and workers. Workers can be exposed to azinphos-methyl while handling or applying this chemical and when re-entering a treated site. Voluntary measures will include reducing use on certain fruits; canceling or limiting uses on cotton, sugarcane, and certain trees; limiting production in the U.S.; and implementing new worker protections. These measures are expected to reduce exposures to certain populations. However, because of its continued widespread use, people can still be exposed to this pesticide through food consumption, drinking water, and occupational routes.

Health Effects: Azinphos-methyl is an organophosphate pesticide that blocks the function of cholinesterase enzymes in the nervous system. This cholinesterase inhibition can last for several weeks. Symptoms of exposure at high doses include blurred vision, tremors, nausea, stomach pain, vomiting, breathing difficulties, unconsciousness, and even death. This pesticide is highly toxic by inhalation and skin absorption. Chronic exposure to azinphos-methyl can cause cholinesterase inhibition with flu-like symptoms. Limited animal studies have shown that azinphos-methyl may affect fetal development, but only at doses that were also toxic to the mother. There is also limited evidence from animal studies that azinphos-methyl can affect reproduction. It is not yet known whether these effects would occur in humans at low levels of exposure. EPA has classified azinphos-methyl as not likely to be carcinogenic to humans.

Detecting Azinphos-methyl Exposure: Animal studies suggest that azinphos-methyl is broken down rapidly and eliminated in feces and urine within days of exposure. Azinphos-methyl does not appear to accumulate in tissues. Measurement of azinphos-

methyl in the urine or feces provides evidence of only recent exposure to this pesticide. Metabolites of azinphos-methyl addressed in the CDC National Exposure Report are DMP, DMTP, and DMDTP.

Regulations for Protection of Human Health:

Air – Azinphos-methyl is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for azinphos-methyl.

Food – There are no established food tolerances for azinphos-methyl.

Occupational Exposure – OSHA has established a PEL of 0.2 mg/m^3 for azinphos-methyl in workplace air.

Sources of Information:

EPA. Revised Human Health Risk Assessment: Azinphos-methyl. May 1999. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/azm.htm.

EPA. Preliminary Human Health Assessment for Azinphos methyl Reregistration Eligibility Decision Document. May 1998. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/azinphos/azinphos.pdf.

EPA. Interim Reregistration Eligibility Decision for Azinphos-methyl. Office of Pesticide Programs. 2001. Accessed online at http://www.epa.gov/oppsrrd1/REDs/factsheets/azm_fs.htm.

Extension Toxicology Network (EXTOXNET). Pesticide Information Profile for Azinphos-methyl. June 1996. Accessed online at http://ace.orst.edu/info/extoxnet/pips/azinopho.htm.

Chemical Name: CHLORETHOXYPHOS

Summary: Chlorethoxyfos is a highly toxic organophosphate insecticide used largely in Midwestern corn and feed crop production. Toxic effects observed in long-term animal studies include tremors, weight loss, and decreased weight gain.

Uses and Sources: Chlorethoxyfos is a restricted use pesticide registered for agricultural use on corn and feed crops to control rootworms, wireworms, cutworms, and other pests. It is primarily used in Illinois, Indiana, and Ohio. Approximately 8,500 to 17,800 pounds of chlorethoxyfos are used annually on 37,000 to 122,000 acres in the U.S.

Major Consumer Products: Chlorethoxyfos was registered in 1995 as an insecticide. There are no allowable non-food or residential uses for this product.

Routes of Human Exposure: Workers are most likely to be exposed to this pesticide through handling and applying it. Exposure can occur through dermal absorption, inhalation, or ingestion. According to EPA, field studies have found no residues of chlorethoxyfos in raw agricultural commodities tested. These findings would suggest low potential for human exposure by food consumption. Drinking water is also potentially contaminated with this pesticide. However, EPA modeling studies indicate that exposure to chlorethoxyfos by this route is not expected to pose a health threat.

Health Effects: Chlorethoxyfos is highly toxic by the oral, dermal, and inhalation routes of exposure. The primary effect of exposure to chlorethoxyfos is cholinesterase inhibition. In humans, this can cause symptoms such as nausea, dizziness, confusion, and at high doses, respiratory failure and death. Toxic effects observed in long-term animal studies include tremors, cholinesterase inhibition, decrease in body weight or decreased weight gain. Chlorethoxyfos does not appear to cause delayed neurotoxicity. Animal studies have shown developmental effects (lower numbers of live fetuses per litter), but only at doses that were also toxic to the mother. There is also evidence from animal studies suggesting that females may be more sensitive to the effects of chlorethoxyfos than males. Chlorethoxyfos has not shown mutagenicity in laboratory studies. Based on lack of evidence in animal studies, EPA categorizes chlorethoxyfos as not classifiable as to human carcinogenicity.

Detecting Exposure: Chlorethoxyfos appears to be rapidly metabolized and excreted from the body. Metabolites of chlorethoxyfos addressed in the CDC National Exposure Report are DEP and DEPT.

Regulations for Protection of Human Health:

Air – Chlorethoxyfos is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for chlorethoxyfos.

Food – EPA has established a maximum allowable residue of 0.01 ppb in corn commodities treated with chlorethoxyfos.

Occupational Exposure – OSHA has not established a standard for chlorethoxyfos in workplace air.

Sources of Information:

EPA. Report on FQPA Tolerance Reassessment Progress and Interim Risk Management Decision for Chlorethoxyfos. Office of Pesticide Programs. June 2000. Accessed online at http://www.epa.gov/REDs/chlorethtred.pdf.

EPA. Human Health Risk Assessment: Chlorethoxyfos. August 1999. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/chlorethoxyfos.htm.

Chemical Name: CHLORPYRIFOS-METHYL

Summary: Chlorpyrifos-methyl is an organophosphate insecticide registered for use solely for the control of insects on various grains. Symptoms of exposure to are similar to those of other organophosphate insecticides, and include effects on the liver, kidney and adrenal glands.

Uses and Sources: Chlorpyrifos-methyl is applied to certain stored grains, such as wheat, barley, oats, rice, and sorghum, and to treat empty grain bins. The EPA reports that approximately 80,000 pounds of chlorpyrifos-methyl active ingredient are applied each year within the U.S. Registrations of chlorpyrifos-methyl were recently voluntarily cancelled by manufacturers. These cancellations will be phased-in to allow users time to transition to other products.

Major Consumer Products: There are no allowable consumer or residential uses.

Routes of Human Exposure: Chlorpyrifos-methyl is one of the most commonly found pesticides in the food supply. The FDA reported detectable residues of this pesticide in 18% of all food samples analyzed in the 2000 Total Diet Study and in 10% of all baby foods tested (fruit juices, fruits, and grains). The potential for drinking water contamination with chlorpyrifos-methyl appears to be low, because it is used inside storage facilities. Workers involved in mixing, loading, and applying chlorpyrifos-methyl can be exposed to higher levels of this pesticide than the general population.

Health Effects: Chlorpyrifos-methyl is known to be toxic by ingestion, skin absorption, and eye contact. There is insufficient information to evaluate the toxicity of chlorpyrifos-methyl by the inhalation route. The clinical symptoms of exposure to chlorpyrifos-methyl are similar to other organophosphate insecticides, and include cholinesterase inhibition, weight loss, and effects on the liver, kidney, and adrenal glands. Chlorpyrifos-methyl has been classified by EPA as not likely to be a human carcinogen. There is inadequate information available to determine whether this pesticide has effects on the reproductive system or on the developing fetus. It is also uncertain whether chlorpyrifos-methyl exposure can have delayed effects on the nervous system.

Detecting Exposure: No information was found regarding uptake, metabolism, retention, or excretion rates for metabolites of chlorpyrifos-methyl. DMP, DMTP, and 3,5,6-trichloro-2-pyridinol are metabolites of chlorpyrifos-methyl.

Regulations for Protection of Human Health:

Air – Chlorpyrifos-methyl is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for chlorpyrifos-methyl.

Food – EPA has established tolerances for residues of chlorpyrifos-methyl in raw agricultural commodities. The tolerances vary by crop or food commodity and range from 6.0 ppm for grains (barley, oats, sorghum and wheat) to 0.1 ppm for eggs.

Occupational Exposure – OSHA has not established a standard for chlorpyrifos-methyl in workplace air.

Sources of Information:

EPA. Human Health Risk Assessment: Chlorpyrifos-Methyl. Office of Pesticide Programs. April 2000. Accessed online at http://www.epa.gov/pesticides/op/chlorpyrifos-methyl.htm.

EPA. Toxicology Section of the RED Chapter, Chlorpyrifos-methyl. April 2000. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/chlorpyrifos-methyl/rev_toxicology.pdf.

EPA. Reregistration Eligibility Decision for Chlorpyrifos-methyl. Office of Pesticide Programs. October 2000. Accessed online at http://www.epa.gov/oppsrrd1/REDs/factsheets/cpm_fs.htm.

Chemical Name: COUMAPHOS

Summary: Coumaphos is an organophosphate pesticide that is used primarily to treat cattle and other livestock for fleas, ticks, and mites. There is limited evidence from animal studies that high doses of coumaphos can cause reproductive effects, including fewer pregnancies.

Uses and Sources: Coumaphos is used on beef and dairy cattle, sheep, swine, horses, and in beehives, to manage fleas, ticks, and mites. According to the EPA, approximately 71,000 pounds of coumaphos active ingredient are used nationwide, treating an average of 6.1 million animals annually. Its largest market is allocated to cattle, accounting for 83% of coumaphos used yearly, and most usage is in Texas, Arizona, and Colorado.

The national use breakdown for coumaphos is roughly as follows:

- General agricultural 91%
- USDA (including tick eradication and import programs) 9%

Major Consumer Products: There are no registered uses of coumaphos in or around residential areas.

Routes of Human Exposure: Workers involved in handling, mixing, and applying coumaphos have the highest exposure potential. People can also be exposed to coumaphos through consumption of certain meat and dairy products that may contain pesticide residues. Drinking water can also be contaminated by coumaphos and may be another source of exposure to this pesticide.

Health Effects: Coumaphos is highly toxic by ingestion and inhalation routes of exposure and moderately toxic by skin absorption. As with other organophosphates, coumaphos causes cholinesterase inhibition in humans. In high doses, exposure can cause nausea, dizziness, confusion, and at very high doses, respiratory paralysis and death. According to EPA, coumaphos is not likely to be carcinogenic in humans and there is no evidence of mutagenicity. Once in the mother's bloodstream, coumaphos can cross the placenta and reach the fetus. There is limited evidence from animal studies that high doses of coumaphos can cause reproductive effects, including fewer pregnancies, smaller litters, and fewer surviving offspring. Developmental studies in animals have found no evidence that the fetus is more sensitive to coumaphos than adult animals. Effects on the fetus and offspring have been observed only at or above exposure levels that were toxic to the mother.

Detecting Exposure: Coumaphos is rapidly broken down in the body and eliminated in urine and feces. There is no evidence that coumaphos accumulates in the body. Animal studies showed that 70% of an oral dose was eliminated within 7 days. Metabolites of coumaphos addressed in the CDC National Exposure Report are DEP and DEPT.

Regulations for Protection of Human Health:

Air – Coumaphos is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for coumaphos.

Food – EPA has established tolerances ranging from 0.1 to 1 ppm for coumaphos residues in a variety of foods and agricultural commodities.

Occupational Exposure – OSHA has not established a standard for coumaphos in workplace air.

Sources of Information:

EPA. Reregistration Eligibility Decision Addendum and FQPA Tolerance Reassessment Progress Report: Coumaphos. September 2000. Office of Pesticide Programs. Accessed online at http://www.epa.gov/REDs/0018tred.pdf.

EPA. Risk Assessment for Coumaphos. January 2000. EPA Office of Pesticide Programs. Updates issued in June, August, and October 2000. Accessed online at http://www.epa.gov/pesticides/op/coumaphos.htm.

Extension Toxicology Network (EXTOXNET), Pesticide Information Profile for Coumaphos. June 1996. Accessed online at http://ace.ace.orst.edu/info/extoxnet/pips/coumapho.htm.

Chemical Name: DICHLORVOS

Summary: Dichlorvos is used as an insecticide primarily in food storage areas, greenhouses, and barns. Dichlorvos is classified by EPA as a probable human carcinogen, and may weaken the immune system. There is limited evidence linking dichlorvos exposure to sperm abnormalities in mice.

Uses and Sources: Dichlorvos is registered for the control of aphids, flies, mosquitoes, gnats, cockroaches, fleas, and other pests in agricultural, commercial, industrial, and residential areas. It can be used in the home for crack and crevice treatment or for pest control on pets. Dichlorvos is also used to treat parasitic worm infections in dogs, livestock, and humans.

Major Consumer Products: The EPA has recommended that the use of dichlorvos be reduced, though over 100 registered products exist. Alco pet products come in strips, sprays, and collars to ward off insects (especially fleas) from household pets. Household insecticides containing dichlorvos are less commonly used, but Summit products are examples of these.

Routes of Human Exposure: The most likely routes of exposure to dichlorvos are breathing contaminated air following application and exposure through the skin while handling and applying dichlorvos. Dichlorvos can also be present as a residue on fruits, vegetables, and grains.

Health Effects: Dichlorvos is highly toxic to humans by inhalation, skin absorption, and ingestion. Acute exposure can cause a wide range of effects in humans, depending on the amount taken into the body. These effects include perspiration, nausea, vomiting, diarrhea, muscle weakness, fatigue, convulsions, breathing difficulties, coma, and possibly death. There is limited information on the chronic effects of dichlorvos in humans, but it is known that chronic low-level exposures can cause cholinesterase inhibition without clinical signs of neurological toxicity. Dichlorvos exposure does not appear to cause delayed effects on the nervous system. Dichlorvos is classified by EPA as a probable human carcinogen.

Studies of chronic exposure in animals have shown decreased cholinesterase activity and potential to weaken the immune system. There is limited evidence of reproductive effects (sperm abnormalities) in mice exposed to dichlorvos. There is no evidence from animal studies that dichlorvos causes developmental effects.

Detecting Exposure: Metabolites of dichlorvos can be measured in the urine, although the breakdown products are rapidly excreted from the body (within a day or two after exposure). In the CDC National Exposure Report, DMP is reported as the metabolic breakdown product of dichlorvos. Two other organophosphates, naled and trichlorphon, are converted in the body to dichlorvos. Exposure to these compounds would have to be ruled out to confirm dichlorvos exposure.

Regulations for Protection of Human Health:

Air – Dichlorvos is regulated by EPA as a Hazardous Air Pollutant under the Clean Air Act.

Drinking Water – EPA has not established a drinking water standard or health advisory for dichlorvos.

Food – EPA has established tolerances ranging from 0.02 to 2 ppm for dichlorvos residues in a variety of foods and agricultural commodities.

Occupational Exposure – OSHA has established a PEL of 1 mg/m^3 for dichlorvos in workplace air.

Sources of Information:

ATSDR. *Toxicological Profile for Dichlorvos*. September 1997. U.S. Department of Health and Human Services. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp88.html.

EPA. Human Health Risk Assessment: Dichlorvos (DDVP). Office of Pesticide Programs. August 2000. Accessed online at http://www.epa.gov/pesticides/op/ddvp/hedrisk.pdf.

Pesticide Action Network Pesticide Database. Chemical Information about Dichlorvos. San Francisco, CA. 2002. Accessed online at http://www.pesticideinfo.org/PCW/Detail_Chemical.jsp?Rec_Id=PC33362.

Chemical Name: DICROTOPHOS

Summary: Dicrotophos is an organophosphate pesticide used largely on cotton to control aphids, spider mites, grasshoppers, boll weevils, and other insects. As a restricted use pesticide, dicrotophos can only be purchased and used by certified applicators. Dicrotophos is suspected to be toxic to the fetus, and animal studies have provided limited evidence of reproductive effects at high levels of exposure.

Uses and Sources: Dicrotophos is registered for use on cotton and ornamental or shade trees for control of aphids, spider mites, grasshoppers, boll weevils, and other insects. According to the EPA, approximately 313,000 pounds are applied per year on 1,145,000 acres within the U.S. Dicrotophos is used from California to North Carolina, with the heaviest use in the Mississippi Valley region.

Major Consumer Products: There are no allowable residential uses of the insecticide.

Routes of Human Exposure: Exposure to dicrotophos can occur through ingestion, dermal contact, and inhalation of the product. Since there are no residential uses of this pesticide, the general public is unlikely to come into direct contact. Exposure could potentially occur through consumption of contaminated drinking water. Dicrotophos has been found to contaminate surface water (due to runoff from treated areas), which could serve as a drinking water supply. Workers can be exposed to dicrotophos while handling, mixing, or applying the pesticide.

Health Effects: Dicrotophos is moderately to highly toxic by oral and dermal routes of exposure. The primary health effect of dicrotophos is cholinesterase inhibition. Symptoms of acute exposure include headache, nausea, vertigo, weakness, loss of muscle coordination, diarrhea, convulsions, coma, shock and other effects. Animal studies have provided limited evidence of reproductive effects (reduced litter size) at high levels of exposure. There is no evidence from animal or human studies of developmental effects from exposure to dicrotophos. Dicrotophos is suspected to be mutagenic. According to EPA, there is suggestive evidence that dicrotophos may be carcinogenic, but there is insufficient information to assess its potential for carcinogenicity in humans.

Detecting Exposure: Dicrotophos is rapidly metabolized and excreted from the body. There is no evidence that dicrotophos accumulates in the body. DMP is the metabolite used to assess dicrotophos exposure in the CDC National Exposure Report.

Regulations for Protection of Human Health:

Air – Dicrotophos is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for dicrotophos.

Food – Dicrotophos is not used on food products. EPA has established a tolerance of 0.05 ppm for dicrotophos residues on cotton.

Occupational Exposure – OSHA has not established a standard for dicrotophos in workplace air.

Sources of Information:

EPA. Human Health Risk Assessment for Dicrotophos. June 2000. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/dicrotophos.htm.

Extension Toxicology Network (EXTOXNET). Pesticide Information Profile for Dicrotophos. Accessed online at http://ace.ace.orst.edu/info/extoxnet/pips/dicrotop.htm.

Chemical Name: DIMETHOATE

Summary: Dimethoate is an organophosphate insecticide and miticide used on food and non-food crops and non-grass animal feed. There is limited evidence of reproductive effects from dimethoate exposure, including decreased reproduction, decreases in number of live births, and decreased growth rates of surviving animals.

Uses and Sources: According to EPA, approximately 2.6 million pounds of dimethoate active ingredient are applied annually to an average of 5 million acres in the U.S. Dimethoate is used to kill aphids, citrus thirps, grasshoppers, beetles, fruit worms, moths and other pests found on a wide variety of fields and orchard agricultural crops and ornamental plants. It is applied in the largest amounts to wheat, alfalfa, and cotton crops, and is also used on various fruits, nuts, vegetables, and grains, including apples, tomatoes, citrus fruits, potatoes, and melons.

National use breakdown for dimethoate is roughly as follows:

- Major field crops 47%
- Orchard crops 35%
- Vegetable crops 17.2%
- Non-food crops Not available

Major Consumer Products: There are no residential products containing dimethoate.

Routes of Human Exposure: Exposure to dimethoate in and around the home is unlikely, since the manufacturer has recently withdrawn residential and public area uses of this pesticide. Dimethoate is used on a wide variety of food crops, introducing the potential for the general public to be exposed by eating foods with pesticide residues. (Dimethoate is used on certain leafy vegetables, cereal grains, citrus fruit, legumes, and many other food crops.) Dimethoate can also enter surface water and groundwater, with the potential to contaminate drinking water supplies. Workers are potentially exposed to dimethoate while handling, mixing, and applying dimethoate.

Health Effects: Exposure to dimethoate can cause cholinesterase inhibition, resulting in overstimulation of the nervous system. Symptoms of acute exposure include nausea, dizziness, tremors, confusion, and at high doses, respiratory paralysis and death. It is moderately toxic by ingestion and slightly toxic by dermal absorption. There is limited evidence of reproductive and developmental effects from dimethoate exposure. Reproductive toxicity studies in animals have shown decreased reproduction, decreases in number of live births, and decreased growth rates of surviving animals. Developmental studies in animals have shown reduced fetal weight at high doses of dimethoate. Dimethoate has shown some evidence of mutagenicity. EPA has classified dimethoate as a possible human carcinogen.

Detecting Exposure: Based on animal and human studies, dimethoate appears to be metabolized and eliminated rapidly from the body. In human volunteers, most of the

administered dimethoate was eliminated within 24 hours. The metabolites of dimethoate included in the CDC Exposure Report are DMP, DMTP, and DMDTP.

Regulations for Protection of Human Health:

Air – Dimethoate is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for dimethoate.

Food – EPA has established tolerances ranging from 0.002 to 5 ppm for dimethoate residues in a variety of foods and agricultural products.

Occupational Exposure – OSHA has not established a standard for dimethoate in workplace air.

Sources of Information:

EPA. Human Health Risk Assessment for Dimethoate. December 1999. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/dimethoate.htm.

Extension Toxicology Network (EXTOXNET). Pesticide Information Profile for Dimethoate. Accessed online at http://ace.ace.orst.edu/info/extoxnet/pips/dimethoa.htm.

Pesticide Action Network Pesticide Database. Chemical Information about Dimethoate. San Francisco, CA. 2002. Accessed online at http://www.pesticideinfo.org/PCW/Detail_Chemical.jsp?Rec_Id=PC33349.

Chemical Name: DISULFOTON

Summary: Disulfoton is an organophosphate insecticide used to control pests on various grains, vegetables, cotton, and other crops. Chronic exposure to disulfoton has been found to cause damage to the eyes and nearsightedness in animals. It may also affect organs such as the lungs, stomach, and lymph nodes.

Uses and Sources: Disulfoton is used in agricultural settings to control pests, such as aphids, leafhoppers, beet flies, spider mites, and coffee leaf miners on various crops, including coffee, cotton, wheat, peanuts, barley, corns, oats, potatoes, beans, and alfalfa. It also has residential uses such as treatment of Christmas trees, vegetable and rose gardens, ornamentals, and non-bearing fruit trees. According to the EPA, approximately 1.7 million pounds of disulfoton active ingredient are used in the U.S. each year. The largest use of disulfoton is for cotton and wheat. California uses the largest amount.

Major Consumer Products: Non-crop disulfoton applications are used on ornamental, flower, and vegetable gardens. However, the EPA has recently enforced restrictions on the amounts, frequency, and types of residential treatments to reduce residential exposures. Green Light and Dragon products are both disulfoton insecticides applied to rose and ornamental gardens.

Routes of Human Exposure: Most exposure to disulfoton occurs by ingesting residues in food and water; inhalation or skin contact during pesticide handling and application; or through contact with residues on treated plants.

Health Effects: Disulfoton is acutely toxic by ingestion, skin absorption, and inhalation. Disulfoton and its metabolites inhibit the activity of cholinesterase enzymes in the nervous system. Neurological effects such as tremors, convulsions, coma and possibly death are more likely to be observed following acute exposures to disulfoton. Other symptoms of acute exposure include vomiting, diarrhea, and difficulty breathing. There are few studies available on the chronic effects of disulfoton exposure on humans. However, animal studies have found that chronic exposure to low levels of disulfoton also causes cholinesterase inhibition. In animal studies, chronic exposure was found to cause damage to the eyes and nearsightedness. Chronic exposure to disulfoton may also affect the lungs, stomach, pancreas, spleen and lymph nodes, based on results of animal studies. Disulfoton has been found to have developmental effects in some animal studies, but at doses that are also toxic to the mother. Animals born to exposed mothers have shown effects including underdeveloped bones, effects on the liver and kidneys, and underdeveloped testes. Some animal studies have also shown reproductive effects, including lower pregnancy rates and smaller litters. The relevance of these studies to effects on humans is unclear. Disulfoton has been found to be mutagenic in some studies, but there is no evidence for carcinogenicity in animals or humans.

Detecting Exposure: Disulfoton is rapidly metabolized and excreted, and does not appear to accumulate in the body. It can be measured in blood, urine, feces, liver, kidney, or body fat and is detectable in urine for up to one week after a person was last exposed.

Metabolites of disulfoton addressed in the CDC National Exposure Report are DEP, DEPT, and DEDTP.

Regulations for Protection of Human Health:

Air – Disulfoton is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard for disulfoton, but has developed a health advisory of 0.01 ppm for children and a lifetime health advisory of 0.0003 ppm.

Food – EPA has established tolerances ranging from 0.1 to 5 ppm for disulfoton residues in a variety of foods and agricultural commodities.

Occupational Exposure – OSHA has not established a standard for disulfoton in workplace air.

Sources of Information:

ATSDR. *Toxicological Profile for Disulfoton*. August 1995. U.S. Department of Health and Human Services. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp65.html.

EPA. Human Health Risk Assessment: Disulfoton. Office of Pesticide Programs, February 2000. Accessed online at http://www.epa.gov/pesticides/op/disulfoton/hedrra.pdf.

Extension Toxicology Network (EXTOXNET). Pesticide Information Profile for Disulfoton. Accessed online at http://ace.ace.orst.edu/info/extoxnet/pips/disulfot.htm.

Pesticide Action Network Pesticide Database. Chemical Information about Disulfoton. San Francisco, CA. 2002. Accessed online at http://www.pesticideinfo.org/PCW/Detail_Chemical.jsp?Rec_Id=PC33305.

Chemical Name: ETHION

Summary: Ethion is an organophosphate insecticide used primarily on citrus fruits in Florida. People can be exposed by consuming raw fruit and fruit juices. There is limited information available regarding the potential toxic effects of chronic, low-level exposure to ethion. Developmental effects, including delayed development of the bones, have been observed in animal studies. Starting in 2003, EPA will begin prohibiting the sale and distribution of ethion for domestic products.

Uses and Sources: Ethion is an insecticide used to kill aphids, mites, and other insects on citrus fruits and is also applied on livestock to control biting flies, other insects, and parasites. It is used most heavily in Florida (99%) and Texas. Based on data from 1987 through 1997, the EPA has estimated that approximately 883,000 to 1,270,000 pounds of ethion active ingredient are used annually, nationwide. Between 287,000 and 413,000 acres are treated each year with this insecticide. Ethion is used on various food crops such as grapefruit, lemons, oranges, limes, tangelos, and tangerines. Limes, tangelos, grapefruit, and oranges are the crops applied with the highest percentage of ethion.

Major Consumer Products: There are no residential uses of ethion; all products with residential uses were voluntary cancelled by the manufacturer in 1998.

Routes of Human Exposure: Ethion can be absorbed into the body by inhalation of aerosols, through the skin, or by ingestion. People can be exposed by consuming raw fruit and fruit juices or by consuming meat and milk. Exposure can also occur by drinking water contaminated by ethion, although this is not believed to be a significant route of exposure. Ethion is rarely found in drinking water in the U.S. Ethion can appear in breast milk, based on results of animal studies. Workers can be exposed to ethion while handling, mixing, and applying ethion, or by re-entering treated areas.

Health Effects: Ethion is toxic to humans by the oral route, inhalation route, and dermal route. As an organophosphate pesticide, ethion's toxicity results from inhibition of cholinesterase enzymes in the nervous system. Symptoms of acute exposure include nausea, vomiting, heavy sweating, dizziness, confusion, and at high exposures, respiratory paralysis, and death. There is limited information available regarding the potential toxic effects of chronic, low-level exposure to ethion. In the small number of studies conducted, no signs of toxicity have been observed in humans or animals exposed to low levels of ethion. Developmental effects, including delayed development of the bones, have been observed in animal fetuses following maternal exposure to high levels of ethion. It is not yet known whether ethion interferes with hormonal mechanisms in the body. There is currently no evidence of reproductive effects from ethion exposure. EPA has classified ethion as non-carcinogenic in humans, based on lack of effects in animal studies.

Detecting Exposure: Ethion is readily metabolized in the body and excreted primarily into the urine. There is no evidence that ethion accumulates in the body. Metabolites of ethion included in the CDC National Exposure Report are DEP, DEPT, and DEDTP.

Regulations for Protection of Public Health:

Air – Ethion is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for ethion.

Food – EPA has established tolerances ranging from 0.2 to 10 ppm for ethion residues in a variety of foods and agricultural commodities.

Occupational Exposures – OSHA does not regulate levels of ethion in workplace air. The National Institute for Occupational Safety and Health (NIOSH) recommends that levels of ethion in workplace air not exceed 0.4 mg/m^3, as an average over 10 hours.

Sources of Information:

ATSDR. *Toxicological Profile for Ethion*. September 2000. U.S. Department of Health and Human Services. Accessed online at http://www.atsdr.cdc.gov/toxprofiles/tp152.html.

EPA. Human Health Risk Assessment for Ethion. July 1999. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/ethion.htm.

Chemical Name: FENITROTHION

Summary: Fenitrothion is an organophosphate pesticide used in ant and roach baits and to control various other pests. Chronic exposure can cause vision problems, memory loss, nausea, weight loss, muscle weakness, and tremors in humans. Reproductive effects, including decreased fertility, decreased viability of the fetus, and reduced lactation, have also been observed in animal studies. Fenitrothion can also be toxic to the immune system.

Uses and Sources: Fenitrothion is used in ant and roach baits for homes and commercial establishments and on non-food crops. Fenitrothion is also used for mosquito control. There are no domestic food or feed uses for fenitrothion, although it can be found in wheat gluten imported from Australia. EPA estimates that approximately 250 million pounds of fenitrothion are applied annually in the U.S.

Major Consumer Products: Only four products of fenitrothion are registered with the EPA. Tat products include an ant trap and a roach bait, and Sumithion produced concentrates for roach baits.

Routes of Human Exposure: People can be exposed to fenitrothion in the home after mosquito control treatments, consumption of food products containing imported wheat gluten, use of insect baits, or by drinking water contaminated with fenitrothion. Workers can be exposed while handling, mixing, or applying fenitrothion, or by re-entering areas after treatment.

Health Effects: Fenitrothion is toxic by inhalation, skin absorption, and ingestion and can cause effects on the nervous system through cholinesterase inhibition. Acute human exposure to fenitrothion causes symptoms similar to other organophosphate insecticides, including vomiting, diarrhea, tremors, and muscle weakness. Chronic exposure can cause effects on vision, fatigue, memory loss, nausea, weight loss, muscle weakness, and tremors.

Animal studies have shown evidence of developmental effects, including fetal bone abnormalities, but only at doses of fenitrothion that were also toxic to the mother. Reproductive effects have also been observed in animal studies, including decreased fertility, decreased viability of the fetus, and reduced lactation. Fenitrothion can also be toxic to the immune system. EPA has classified fenitrothion as non-carcinogenic in humans.

Detecting Exposure: Metabolism studies in animals have shown that fenitrothion is rapidly metabolized and excreted in the urine and feces. Metabolites of fenitrothion addressed in the CDC National Exposure Report are DMP and DMTP.

Regulations for Protection of Human Health:

Air – Fenitrothion is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for fenitrothion.

Food – The EPA has established a tolerance of 15 ppm for fenitrothion in wheat gluten imported from Australia.

Occupational Exposure – OSHA has not established a standard for fenitrothion in workplace air.

Sources of Information:

EPA. Human Health Risk Assessment for Fenitrothion. March 2000. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/fenitrothion.htm.

EXTOXNET. Pesticide Information Profile for Fenitrothion. September 1995. Accessed online at http://ace.orst.edu/info/extoxnet/pips/fenitrot.htm.

Pesticide Action Network Pesticide Database. Chemical Information about Fenitrothion. San Francisco, CA. 2002. Accessed online at http://www.pesticideinfo.org/PCW/Detail_Chemical.jsp?Rec_Id=PC36373.

Chemical Name: FENTHION

Summary: Fenthion is an organophosphate insecticide used only in Arkansas, Florida, and Missouri. Although it has been used to protect livestock from lice, flies, and ticks, all uses except mosquito control are now being phased out. Animal research has produced limited evidence that fenthion is a developmental toxicant. It may also cause reproductive effects, including fewer implanted embryos, reduced fertility, and decreased growth in newborns.

Uses and Sources: Fenthion was first registered in 1965 to be used for mosquito and insect control for swamp areas, standing water, recreation areas, alfalfa, pasture grass, forests, restaurants, and homes. It was also used for lice control on livestock, control of insects on flowers, and for bird control. Fenthion is used on cattle and swine to protect against lice, flies and ticks. These uses were voluntarily cancelled by the manufacturer in 2000 and will be phased out over the next two years. According to EPA data, approximately 343,100 pounds of fenthion are used annually; approximately one-third of this is for mosquito control. However, in 1998, the EPA issued a Registration Standard that ordered the termination of fenthion for several of those uses.

Major Consumer Products: There are no registered residential uses of products containing fenthion.

Routes of Human Exposure: Fenthion can enter fat and meat of cattle treated with this pesticide, posing a possible risk to people consuming meat products. This risk is removed, however, by cancellation of uses on livestock. People can also come into contact with fenthion in areas where it is used for mosquito control. Outdoor applications of fenthion for mosquito control can also result in potential contamination of drinking water sources. Workers can be exposed to fenthion while handling, mixing and applying fenthion.

Health Effects: Fenthion is a strong cholinesterase inhibitor in humans and is toxic by ingestion, skin absorption, and inhalation. Acute effects of fenthion exposure include nausea, dizziness, confusion, and, at high doses, respiratory paralysis and death. Signs of chronic toxicity observed in exposed animals include damage to the eyes and to the male reproductive system. Animal studies have shown limited evidence that fenthion is a developmental toxicant. There is also evidence from animal studies that fenthion can cause reproductive effects, including fewer implanted embryos, reduced fertility, fewer and smaller litters, and decreased growth in newborn animals. EPA has indicated that fenthion is not likely to cause cancer in humans.

Detecting Exposure: Based on animal studies, fenthion is quickly absorbed into the body and eliminated through the urine and feces. There is evidence that fenthion may be stored in the body for a period of time before it is eliminated. Metabolites of fenthion included in the CDC National Exposure Report are DMT and DMTP.

Regulations for Protection of Human Health:

Air – Fenthion is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for fenthion.

Food Residues – EPA has established tolerances ranging from 0.01 to 0.1 ppm for fenthion residues in a variety of foods and agricultural commodities.

Occupational Exposure – OSHA has not established a standard for fenthion in workplace air.

Sources of Information:

EPA. Interim Reregistration Eligibility Decision for Fenthion. January 2001. Office of Pesticide Programs. Accessed online at http://www.epa.gov/REDs/0290ired.pdf.

EPA. Human Health Risk Assessment for Fenthion. October 1999. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/fenthion.htm.

Extension Toxicology Network (EXTOXNET). Pesticide Information Profile for Fenthion. June 1996. Accessed online at http://ace.ace.orst.edu/info/extoxnet/pips/fenthion.htm.

Pesticide Action Network Pesticide Database. Chemical Information about Fenthion. San Francisco, CA. 2002. Accessed online at http://www.pesticideinfo.org/PCW/Detail_Chemical.jsp?Rec_Id=PC33351.

Chemical Name: METHIDATHION

Summary: Methidathion is an organophosphate insecticide-miticide used on various crops throughout the U.S., especially tree fruits and nuts. It is believed that chronic exposure to methidathion can cause liver damage and reproductive effects.

Uses and Sources: Methidathion is used to control a range of sucking, leaf eating, and scale insects. According to EPA data from 1987 through 1997, approximately 241,000 pounds of methidathion active ingredient were used annually on 138,000 acres throughout the U.S. It is used on a variety of crops, including nuts, citrus and other fruit crops, artichokes, olives, cotton, alfalfa, safflower, and several other food and feed products. Almond, orange, plum, prune, and walnut crops receive the largest applications of methidathion annually.

Major Consumer Products: Products containing methidathion have very limited use except on crops. Supracide is an insecticide-miticide that is registered for use on ornamental shrubs and flowers but not inside the house.

Routes of Human Exposure: People can be exposed to this pesticide by consuming produce (primarily through citrus) that has been treated; by consuming contaminated drinking water; or through the workplace. Methidathion is not registered for use in the home, schools, or parks, reducing the potential for exposure by the general public. Workers can be exposed to methidathion while mixing and applying the product or by re-entering sprayed areas.

Health Effects: Methidathion is a cholinesterase inhibitor and at high doses can cause nausea, dizziness, confusion, respiratory paralysis, and death. It is toxic by inhalation, dermal absorption, and ingestion. Toxic effects observed in chronic animal studies include liver abnormalities and chronic hepatitis. There is also evidence that exposure can cause reproductive effects in animals. There is no evidence of developmental effects from exposure to methidathion. EPA has classified methidathion as a possible human carcinogen.

Detecting Exposure: Methidathion is readily metabolized and excreted from the body. Metabolites of this pesticide included in the CDC National Exposure Report are DMP, DMTP, and DMDTP.

Regulations for Protection of Human Health:

Air – Methidathion is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for methidathion.

Food – EPA has established tolerances ranging from 0.05 to 12 ppm for methidathion residues in a variety of foods and agricultural commodities.

Occupational Exposure – OSHA has not established a standard for methidathion in workplace air.

Sources of Information:

EPA. Human Health Risk Assessment for Methidathion. December 1999. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/methidathion.htm.

Extension Toxicology Network (EXTOXNET). Pesticide Information Profile for Methidathion. June 1996. Accessed online at http://ace.orst.edu/info/extoxnet/pips/methidat.htm.

Pesticide Action Network Pesticide Database. Chemical Information about Methidathion. San Francisco, CA. 2002. Accessed online at http://www.pesticideinfo.org/PCW/Detail_Chemical.jsp?Rec_Id=PC32869.

Chemical Name: METHYL PARATHION

Summary: Methyl parathion is an organophosphate insecticide that is registered for use on agricultural crops only. Its use is generally limited to Southern and Midwestern states that produce cotton, vegetables, and soybeans, although illegal use in homes and businesses has been increasingly documented in several states. Low-level, long-term exposure to methyl parathion can cause effects on the central nervous system, decreased heart rate and blood pressure, and weakening of the immune system, as documented in animal studies. Methyl parathion may also be an endocrine disruptor.

Uses and Sources: Methyl parathion is a restricted use pesticide, meaning that only trained people are allowed to mix, load, and spray it. It is registered for use on cotton, certain food and feed crops, ornamental plants, forest trees, rights-of-way, and grazing lands. Uses on most fruits and vegetables have been canceled. It is used in nearly every state, but most heavily in southern states and in California. Approximately 4 million pounds of methyl parathion are used on an annual basis within the U.S. on 26 crops spanning 5 million acres. Uses include: cotton, alfalfa, cabbage, oats, pecans, and potatoes; ornamental plants and forest trees; rights-of-way, and grazing lands. More than two-thirds of the annual use of methyl parathion is applied to cotton and corn crops. There are no registered uses for homeowners, and its use on most fruits and vegetables have been canceled.

Major Consumer Products: Uses on several food and non-food products have been canceled, including apples, broccoli, carrots, cauliflower, celery, grapes, lettuce, pears, plums, tomatoes, daisies, flowering plants, and non-agricultural land.

There are no allowable residential uses of methyl parathion. In recent years, however, a pattern of illegal misuse has emerged in which unscrupulous pesticide applicators use methyl parathion in people's homes and businesses. Thousands of homes and businesses have been contaminated in Mississippi, Louisiana, Tennessee, Arkansas, Illinois, Ohio, Michigan, and Texas since 1994, due to this illegal and dangerous practice.

Routes of Human Exposure: There is potential for people living near agricultural areas to be exposed to spray drift from its use in fields, and illegal use indoors can result in dangerous exposures. Workers can also be exposed to methyl parathion while mixing and applying the pesticide. Methyl parathion can also contaminate surface water and ground water that serve as sources of drinking water.

Health Effects: The primary effect of methyl parathion exposure is cholinesterase inhibition, with associated effects on the central nervous system. Methyl parathion is acutely toxic by oral, dermal, and inhalation routes of exposure and can cause loss of consciousness, dizziness, confusion, headaches, difficulty breathing, blurred vision, sweating, and even death. Changes in mental state may last several months after exposure to high levels of methyl parathion. Animal studies have shown that low-level, long-term exposure to methyl parathion can cause effects on the central nervous system, decreased heart rate and blood pressure, and weakening of the immune system. Developmental

effects have been observed in animal studies, but only at doses that were also toxic to the mother. There is also limited evidence that methyl parathion may cause reproductive effects. Animal studies have been linked to reproductive effects, including reduced survival of newborns, reduced birth weight, stunted growth, and bone formation. EPA has classified methyl parathion as non-carcinogenic to humans. There is evidence that methyl parathion may be an endocrine disruptor.

Detecting Exposure: Animal studies indicate that methyl parathion is rapidly metabolized and excreted in the urine. Metabolites of methyl parathion addressed in the CDC National Exposure Report include DMP, DMTP, and paranitrophenol.

Regulations for Protection of Human Health:

Air – Methyl parathion is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard for methyl parathion, but has developed a health advisory of 0.3 ppm for children and a lifetime health advisory of 0.002 ppm.

Food – EPA has established tolerances ranging from 0.1 to 5 ppm for methyl parathion residues in a variety of foods and agricultural commodities.

Occupational Exposure – OSHA has not established a standard for methyl parathion in workplace air.

Sources of Information:

EPA. Human Health Risk Assessment for Methyl Parathion. August 1999. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/methyl_parathion.htm.

EPA. Revised Toxicology Chapter for the Methyl Parathion RED. August 1999. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/methyl_parathion/mphed_att3.pdf.

ATSDR. ToxFAQs. Accessed online at http://atsdr1.atsdr.cdc.gov/tfacts48.html.

Extension Toxicology Network (EXTOXNET). Pesticide Information Profile for Methyl Parathion. Accessed online at http://ace.ace.orst.edu/info/extoxnet/pips/methylpa.htm.

Chemical Name: NALED

Summary: Naled is an organophosphate insecticide used to control mosquitoes and black flies. Like other organophosphates, naled can cause nervous system effects. There is also the potential for reproductive effects as a result of chronic exposure.

Uses and Sources: According to EPA estimates, approximately 1 million pounds of naled active ingredient are used annually nationwide. In addition to use against flies and mosquitoes, naled is used on food and feed crops such as almonds, beans, broccoli, Brussels sprouts, cabbage, cauliflower, celery, cottonseed, eggplant, grapes, hops, melons, oranges, peas, spinach, strawberries, tomatoes, and walnuts. It also registered for use in greenhouses and pet collars.

National use breakdown for naled is roughly as follow:

- Mosquito/black fly control 70%
- Agricultural uses 28%
- Pet collar use 2%

Major Consumer Products: Naled residential products include Sergeant's pet collars and Trumpet insecticides, though the number of registrants is very limited.

Routes of Human Exposure: People can be exposed to naled by eating treated food products, contact with treated areas, handling pet flea collars, or through occupational exposure. Naled is rapidly broken down in water, reducing the potential for exposure by the drinking water route. Workers can be exposed to naled while mixing and applying the product or by entering treated areas.

Health Effects: Naled causes cholinesterase inhibition, resulting in overstimulation of the nervous system. Symptoms of acute exposure can include abdominal cramps, nausea, vomiting, anxiety, vertigo, weakness, blurred vision, difficulty breathing, convulsions, coma, and death. Chronic exposure studies in animals have shown effects including diarrhea, vomiting, anemia, and changes in bones of the spine. Animal studies have shown no evidence of developmental effects for naled, although there is evidence for potential reproductive effects. The significance of these findings for humans is uncertain. Although naled is classified as non-carcinogenic in humans, one of its metabolites, the pesticide dichlorvos, is classified as a possible human carcinogen.

Detecting Exposure: In animal studies, naled is rapidly absorbed, distributed, and excreted. One of the breakdown products of naled is dichlorvos, another registered pesticide. The metabolite DMP is included in the CDC National Exposure Report as a breakdown product of naled.

Regulations for Protection of Human Health:

Air – Naled is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for naled.

Food – EPA has established tolerances ranging from 0.5 to 10 ppm for naled residues in a variety of foods and agricultural commodities.

Occupational Exposure – OSHA has not established a standard for naled in workplace air.

Sources of Information:

EPA. Human Health Risk Assessment for Naled. October 1999. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/naled.htm

Extension Toxicology Network (EXTOXNET). Pesticide Information Profile for Naled. June 1996. Accessed online at http://ace.ace.orst.edu/info/extoxnet/pips/naled.htm.

Pesticide Action Network Pesticide Database. Chemical Information about Naled. San Francisco, CA. 2002. Accessed online at http://www.pesticideinfo.org/PCW/Detail_Chemical.jsp?Rec_Id=PC127.

Chemical Name: OXYDEMETON-METHYL

Summary: Oxydemeton-methyl (ODM) is an organophosphate insecticide used on a variety of agricultural crops, especially in California's Central and Coastal Valleys and in Idaho. Animal studies have shown evidence of reproductive toxicity following exposure to ODM, including decreased fertility.

Uses and Sources: Oxydemeton-methyl is registered for use to control biting and sucking insects in agricultural crops. This insecticide is used most prominently in California Central and Coastal Valleys and Idaho. Approximately 154,000 pounds of ODM active ingredient are applied yearly to 231,000 acres in the U.S. It is used on field crops, fruit, vegetables, melons, and nuts. In addition, it is used on Christmas tree plantations, seed orchards, ornamental plants, shrubs, and shade trees. Use on blackberries, raspberries, potatoes, and peas has been cancelled.

Major Consumer Products: There are no registered uses for ODM in or around the home.

Routes of Human Exposure: People can be exposed to ODM by consuming treated foods or through occupational contact with the pesticide. Workers can be exposed to ODM by mixing, loading, and applying ODM or by entering treated areas. ODM breaks down readily in water and thus is not a common drinking water contaminant.

Health Effects: ODM causes cholinesterase inhibition in humans. Symptoms of exposure can include nausea, dizziness, confusion, blurred vision, chest tightness, and at high doses, convulsions, coma, respiratory failure, and even death. ODM is highly toxic by dermal absorption, inhalation, and ingestion routes of exposure. Animal studies have shown evidence of reproductive toxicity following exposure to ODM, including decreased fertility. There is no evidence of developmental effects in animal fetuses following ODM exposure. ODM has been found to be mutagenic, but is classified by EPA as not likely to be carcinogenic in humans.

Detecting Exposure: Animal studies have found ODM to be rapidly absorbed, metabolized, and excreted. Metabolites of ODM addressed in the CDC National Exposure Report are DMP and DMTP.

Regulations for Protection of Human Health:

Air – Oxydemeton-methyl is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for oxydemeton-methyl.

Food – EPA has established tolerances ranging from 0.01 to12 ppm for oxydemeton-methyl residues in a variety of foods and agricultural commodities.

Occupational Exposure – OSHA has not established a standard for oxydemeton-methyl in workplace air.

Sources of Information:

EPA. Human Health Risk Assessment for Oxydemeton-methyl. December 1999. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/odm.htm.

EPA. Oxydemeton-methyl: Revised HED Toxicology Chapter for the Reregistration Eligibility Decision (RED) Document. August 1999. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/oxydemeton-methyl/toxicology.pdf.

Chemical Name: PARATHION (ETHYL PARATHION)

Summary: Parathion has been used for the past 40 years to control agricultural pests, such as mites. It is applied on crops mainly in the Central Plains states. Parathion is one of the most highly toxic organophosphate insecticides and can be fatal even at relatively low doses. Chronic exposure to parathion in animals is associated with effects such as anemia, eye, and nervous system damage.

Uses and Sources: Parathion has been used for the past 40 years as a control for agricultural pests, such as mites. It is applied on crops mainly in the Central Plains, including Colorado, Kansas, Nebraska, New Mexico, North Dakota, Oklahoma, South Dakota, and Texas. It is also used often in Alabama, Arizona, Delaware, Georgia, Montana, and Washington. Parathion is used in the largest amounts on wheat and alfalfa and is also used on barley, corn, cotton, canola, sorghum, soybean, and sunflowers. The EPA reports 600,000 pounds of ethyl parathion are used annually nationwide on 777,000 acres.

Major Consumer Products: Because of its high toxicity, parathion it is classified as a restricted use pesticide. There are no allowable residential uses.

Routes of Human Exposure: People can be exposed to parathion residues in food (including meat, poultry, and eggs), through drinking water, or through occupational contact. Workers involved in mixing, loading, and applying parathion are potentially at high risk of exposure to parathion.

Health Effects: Parathion is one of the most potent organophosphate insecticides and a strong inhibitor of cholinesterase. It can be fatal in low doses by inhalation, dermal absorption, and ingestion routes of exposure. Symptoms of acute exposure to parathion include headache, weakness, blurred vision, vomiting, difficulty breathing, convulsions, coma, and potentially, death. Chronic exposure to parathion can result in reduced cholinesterase activity, nausea, and headaches. Chronic toxicity studies in animals have found effects such as anemia and degeneration of the retina and portions of the nervous system. Parathion has not been found to cause developmental effects or reproductive toxicity in animal studies. EPA has classified parathion as a possible human carcinogen.

Detecting Exposure: Parathion is readily metabolized and excreted, primarily in the urine. It may be stored in fat, but is rapidly broken down once released from fat. Metabolites of parathion measured in the CDC National Exposure Report include DEP, DEPT, paranitrophenol.

Regulations for Protection of Human Health:

Air – Parathion is regulated by EPA as Hazardous Air Pollutant under the Clean Air Act.

Drinking Water – EPA has not established a drinking water standard or health advisory for parathion.

Food – EPA has established tolerances ranging from 0.1 to 5 ppm for parathion residues in a variety of foods and agricultural commodities.

Occupational Exposure – OSHA has established a PEL of 0.1 mg/m^3 for parathion in workplace air.

Sources of Information:

EPA. Human Health Risk Assessment for Ethyl Parathion. September 1999. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/ethyl_parathion.htm.

Extension Toxicology Network (EXTOXNET). Pesticide Information Profile for Parathion. September 1993. Accessed online at http://ace.ace.orst.edu/info/extoxnet/pips/parathio.htm.

Chemical Name: PHORATE

Summary: Phorate is a restricted use organophosphate pesticide used in agricultural regions nationwide. Symptoms of phorate exposure are similar to those of other organophosphate insecticides. Chronic exposure to phorate can cause effects on the nervous system and muscle weakness. There is also limited evidence that phorate may have harmful effects on the reproductive system and on the developing fetus.

Uses and Sources: According to EPA, approximately 3 million pounds of phorate active ingredient are used annually throughout the U.S. It is used primarily on corn (45%), potatoes (24%), and cotton (15%), as well as on peanuts, sugarcane, wheat, and sugar beets.

Major Consumer Products: There are no allowable residential uses for phorate.

Routes of Human Exposure: People can be exposed to phorate residues on food products, in drinking water, or through occupational use of this pesticide. Phorate can contaminate drinking water supplies, although it is not persistent in soil or water. Workers can be exposed to phorate while mixing, loading, and applying the pesticide.

Health Effects: Phorate is highly toxic by ingestion, dermal absorption, and inhalation. The major breakdown products of phorate in the body are more toxic and have greater cholinesterase activity than phorate. Symptoms of acute exposure to phorate are similar to those of other organophosphate pesticides. Chronic exposure can cause cholinesterase inhibition, with associated effects on the nervous system and muscles. There is limited evidence from animal studies that phorate may have effects on the developing fetus and the reproductive system. However, EPA does not consider phorate to be a significant developmental or reproductive toxicant. EPA has classified phorate as not likely to be carcinogenic to humans, based on animal studies.

Detecting Exposure: Phorate appears to be readily metabolized by the body and is excreted primarily in the urine. The metabolites of phorate addressed in the CDC National Exposure Report are DEP, DEPT, and DEDTP.

Regulations for Protection of Human Health:

Air – Phorate is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for phorate.

Food – EPA has established tolerances ranging from 0.02 to 3 ppm for phorate residues in a variety of foods and agricultural commodities.

Occupational Exposure – OSHA has not established a standard for phorate in workplace air.

Sources of Information:

EPA. Human Health Risk Assessment for Phorate. September 1999. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/phorate/phoratehedrevrisk.pdf.

Extension Toxicology Network (EXTOXNET). Pesticide Information Profile for Phorate. Accessed online at http://ace.ace.orst.edu/info/extoxnet/pips/phorate.htm.

Chemical Name: PHOSMET

Summary: Phosmet is an organophosphate insecticide, widely used on fruit and vegetable crops, tree crops, nut trees, cotton, ornamental plants, and in forestry. The general population may be exposed to phosmet by consuming food products with pesticide residues or contaminated drinking water. Like other organophosphate pesticides, phosmet can have serious adverse effects on the nervous system, particularly at higher levels of exposure. Animal studies suggest that phosmet may cause cancer, although studies are insufficient to determine its carcinogenic potential in humans.

Uses and Sources: Phosmet is used for control of insects on various food and non-food crops and on cattle and swine. The EPA estimates that approximately 1,250,000 pounds of phosmet are applied yearly within the U.S. Phosmet is used to control pests such as moths, beetles, lice, flies, and ticks on various fruit and vegetable crops, nut trees, cotton, ornamentals, and forests. EPA recently canceled household uses of phosmet, including use on pets, trees, shrub, and household fruit trees.

Major Consumer Products: Following cancellation of residential phosmet uses, there are no remaining consumer uses of this pesticide.

Routes of Human Exposure: Workers involved in mixing, handling, loading, and applying phosmet are at highest risk of exposure. Cancellation of residential uses of phosmet reduces the potential for significant exposure to this pesticide by the general population. However, people can still be exposed to phosmet by consuming food or drinking water that is contaminated with the pesticide.

Health Effects: The toxic effects of phosmet are related to its ability to inhibit cholinesterase in the body. Phosmet is toxic to humans by ingestion, dermal absorption, and inhalation. Symptoms of exposure are similar to those of other organophosphate insecticides. In test animals, toxic effects associated with phosmet exposure include tremors, muscle weakness, convulsions, and decreased activity. There is evidence from animal studies that phosmet may have reproductive and developmental effects, but only at doses that are also toxic to the mother. Animal studies have suggested that phosmet may cause cancer, but there is insufficient evidence to assess its potential for causing cancer in humans.

Detecting Exposure: Phosmet is rapidly metabolized by the body and eliminated in the urine and feces. Phosmet does not appear to bioaccumulate in the body. Metabolites of phosmet addressed in the CDC Exposure Report are DMP, DMTP, and DMDTP.

Regulations for Protection of Human Health:

Air – Phosmet is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for phosmet.

Food – EPA has established tolerances ranging form 0.1 to 40 ppm for phosmet residues in a variety of foods and agricultural commodities.

Occupational Exposure – OSHA has not established a standard for phosmet in workplace air.

Sources of Information:

EPA. Human Health Risk Assessment for Phosmet. February 2000. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/phosmet.htm.

EPA. Interim Reregistration Eligibility Decision for Phosmet. Office of Prevention, Pesticides, and Toxic Substances, Washington, DC. October 2001. Accessed online at http://www.epa.gov/REDs/phosmet_ired.pdf

Extension Toxicology Network (EXTOXNET). Pesticide Information Profile for Phosmet. Accessed online at http://ace.ace.orst.edu/info/extoxnet/pips/phosmet.htm.

Chemical Name: PIRIMIPHOS-METHYL

Summary: Pirimiphos-methyl is an organophosphate pesticide that is registered for the post-harvest control of various pests on stored grains, as well as to control flies on livestock. Pirimiphos-methyl is acutely toxic at very low doses, but there is insufficient information regarding potential chronic human health effects.

Uses and Sources: According to the EPA, approximately 12,000 pounds of pirimiphos-methyl active ingredient are applied annually throughout the nation. Most pirimiphos-methyl is used to control insects on stored corn, sorghum seed, and grain. It is also used in cattle ear tags and on iris bulbs. There are no residential uses of pirimiphos methyl.

Major Consumer Products: There are no allowable residential uses for this pesticide.

Routes of Human Exposure: People can be exposed to pirimiphos-methyl by consuming food with pesticide residues or through occupational exposure. EPA's risk assessment for pirimiphos-methyl indicates that outdoor uses of this pesticide are not likely to cause contamination of drinking water sources. Workers involved in mixing, loading, and application of pirimiphos-methyl are at higher risk of exposure than the general public.

Health Effects: Pirimiphos-methyl causes cholinesterase inhibition at very low doses, and is toxic by all routes of exposure. Symptoms of exposure observed in animal studies include tremors, loss of coordination, paralysis, and salivation. There is insufficient information regarding potential chronic human health effects or potential carcinogenicity of this pesticide. Limited animal studies have found no evidence for developmental or reproductive effects for pirimiphos-methyl.

Detecting Exposure: Pirimiphos-methyl is rapidly metabolized and excreted and does not appear to accumulate in the body. The metabolites of pirimiphos-methyl addressed in the CDC National Exposure Report are DMP and DMTP.

Regulations for Protection of Human Health:

Air – Pirimiphos-methyl is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for pirimiphos-methyl.

Food – EPA has established tolerances ranging from 0.2 to 8 ppm for pirimiphos-methyl residues in a variety of foods and agricultural commodities.

Occupational Exposure – OSHA has not established a standard for pirimiphos-methyl in workplace air.

Sources of Information:

EPA. Human Health Risk Assessment for Pirimiphos-methyl. Office of Pesticide Programs, Washington, DC. July 1999. Accessed online at http://www.epa.gov/oppsrrd1/op/pirimiphos-methyl/hedrra.pdf.

EPA. Interim Reregistration Eligibility Decision for Pirimiphos-Methyl. Office of Prevention, Pesticides, and Toxic Substances, Washington, DC. June 2001. Accessed online at http://www.epa.gov/oppsrrd1/REDs/pirimiphos-methyl_ired.pdf.

EPA. Pirimiphos-methyl: Toxicology Chapter of the Registration Eligibility Document. January 1998. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/pirimiphos-methyl/pm_06.pdf.

Chemical Name: SULFOTEPP

Summary: Sulfotepp is a restricted use organophosphate insecticide that is being phased out for all uses over the next four years. It is highly toxic to humans. The toxicological information for sulfotepp is limited, but it is believed that chronic exposures to small doses of sulfotepp can produce effects similar to those from acute organophosphate exposure.

Uses and Sources: Sulfotepp is used in greenhouses primarily to control whiteflies on poinsettias. It also controls insects, mites, and thrips on ornamental plants. Sulfotepp is not registered for use on food or feed products. The pesticide registrants agreed to voluntarily cancel uses of sulfotepp, effective September 2002. Existing stockpiles can be used until September 2004. EPA estimates that approximately 3,000 to 5,000 pounds of sulfotepp are used annually within the U.S.

Major Consumer Products: There are no registered uses of sulfotepp in residential settings.

Routes of Human Exposure: Workers have the highest potential risk of exposure to sulfotepp. Exposure can occur during application, by entering greenhouses after treatment, or by handling treated plants. Sulfotepp can enter the body by inhalation, ingestion, or absorption through the skin.

Health Effects: The toxicological information for sulfotepp is limited. Sulfotepp is highly toxic to humans and, like other organophosphate insecticides, inhibits cholinesterase in the body. Acute exposure can cause serious health effects including effects on vision, difficulty breathing, nausea, vomiting, slurred speech, loss of coordination, muscle weakness, respiratory paralysis, coma, and death. Chronic exposure to small doses of sulfotepp can also lead to these effects. No information was found regarding potential carcinogenicity, reproductive or developmental effects of exposure to sulfotepp.

Detecting Exposure: The metabolites of sulfotepp addressed in the CDC National Exposure Report are DEP and DEPT.

Regulations for Protection of Human Health:

Air – Sulfotepp is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for sulfotepp.

Food – Sulfotepp has no registered uses on food or food products.

Occupational Exposure – OSHA has not established a standard for sulfotepp in workplace air.

Sources of Information:

EPA. Reregistration Eligibility Decision for Sulfotepp. September 1999. Office of Pesticide Programs. Accessed online at http://www.epa.gov/REDs/0338red.pdf.

EPA. Human Health Risk Assessment for Sulfotepp. June 1999. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/sulfotepp.htm.

National Library of Medicine, TOXNET, Hazardous Substances Data Bank (HSDB). Dithion (Sulfotepp). Accessed online at http://toxnet.nlm.nih.gov/.

Chemical Name: TEMEPHOS

Summary: Temephos is an organophosphate insecticide used primarily to control mosquito larvae. Temephos has low to moderate toxicity to humans and causes symptoms similar to other organophosphate insecticides.

Uses and Sources: Temephos is used to control mosquito larvae in lakes, ponds, wetlands, and other areas of standing water. According to EPA, approximately 25,000 to 40,000 pounds of temephos are used annually nationwide. Temephos was once applied to citrus fruits, but today there are no registered uses on food or feed products. Usage of temephos has been decreasing in recent years.

Major Consumer Products: There are no registered residential uses for temephos.

Routes of Human Exposure: The general public is not likely to be exposed to significant levels of temephos because it is not used in residential areas and it has no uses on food products. According to EPA, drinking water sources are unlikely to be contaminated with temephos because it is not persistent in water, and it is generally applied to water that is not suitable for human consumption (i.e., shallow, brackish, or stagnant water). Workers have the highest potential for exposure to temephos, while mixing, loading, and applying temephos, or by entering a treated area.

Health Effects: There are limited toxicological data available for temephos. It inhibits cholinesterase enzymes in the body and has low to moderate toxicity. Temephos is toxic by all exposure routes of exposure (i.e., ingestion, inhalation, and skin absorption). Signs of acute exposure observed in animal studies are typical of cholinesterase inhibitors and include difficulty breathing, salivation, muscle spasms, and tremors. At this time, there is insufficient information to determine whether temephos causes reproductive or developmental effects or whether it may cause cancer in humans.

Detecting Exposure: The metabolites of temephos included in the CDC National Exposure Report are DMP and DMTP.

Regulations for Protection of Human Health:

Air – Temephos is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for temephos.

Food – Temephos has no registered uses on food or food products.

Occupational Exposure – OSHA has established a PEL of 15 mg/m^3 for temephos as total dust and 5 mg/m^3 for respirable fraction in workplace air.

Sources of Information:

EPA Human Health Risk Assessment for Temephos. September 1999. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/temephos.htm.

Extension Toxicology Network (EXTOXNET). Pesticide Information Profile for Temephos. Accessed online at http://ace.ace.orst.edu/info/extoxnet/pips/temephos.htm.

Chemical Name: TERBUFOS

Summary: Terbufos is one of the most toxic organophosphate insecticides. It is used on corn and other food crops, introducing the potential for exposure through pesticide residues on food and drinking water contamination. Limited studies found reduced male fertility and decreased pregnancy rates in animals exposed to terbufos.

Uses and Sources: According to the EPA, approximately 7.5 million pounds of terbufos active ingredient are applied annually throughout the country to control pests on corn, sugar beets, and sorghum. It is used on bananas grown in South America and imported into the U.S., but not in domestic banana production. Terbufos has no residential uses.

Major Consumer Products: There are no registered residential uses of terbufos.

Routes of Human Exposure: People can be exposed to terbufos by consuming food with pesticide residues, drinking contaminated water, or through occupational exposure. EPA risk assessments have determined that exposure to contaminated drinking water may pose a significant health risk, particularly to infants and children. Workers can be exposed to terbufos during handling, mixing, and applying the pesticide.

Health Effects: Terbufos is one of the most toxic organophosphate insecticides. It is toxic by all routes of exposure, causing cholinesterase inhibition. Symptoms of acute exposure observed in animal studies include tremors, unsteady gait, salivation, muscle weakness, and loss of balance. More severe effects, including death, can occur from acute exposure to terbufos. There is no evidence of developmental effects from animal studies. Limited studies found reduced male fertility and decreased pregnancy rates in animals exposed to terbufos. EPA has classified terbufos as not likely to cause cancer in humans.

Detecting Exposure: Terbufos is rapidly metabolized in the body and excreted primarily in the urine. There is no evidence that terbufos bioaccumulates in humans or animals. The metabolites of terbufos included in the CDC National Exposure Report are DEP, DEPT, and DEDTP.

Regulations for Protection of Human Health:

Air – Terbufos is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard for terbufos, but has developed a health advisory of 0.005 ppm for children and lifetime health advisory of 0.0009 ppm.

Food – EPA has established tolerances ranging from 0.025 to 0.5 ppm for terbufos residues in a variety of food and agricultural commodities.

Occupational Exposure – OSHA has not established a standard for terbufos in workplace air.

Sources of Information:

EPA. Human Health Risk Assessment for Terbufos. Office of Pesticide Programs, Washington, DC. September 1999. Accessed online at http://www.epa.gov/pesticides/op/terbufos.htm.

EPA. Interim Reregistration Eligibility Decision for Terbufos. Office of Pesticide Programs, Washington, DC. September 2001. Accessed online at http://www.epa.gov/REDs/terbufos_ired.pdf

EPA. Terbufos: Toxicology Chapter for RED. September 1999. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/terbufos/hed_tox_chap8.pdf.

EPA. Final Usage Analysis for Terbufos Reregistration Eligibility Decision. August 31, 1999. Accessed online at http://www.epa.gov/pesticides/op/terbufos/terbufos_usage.pdf.

Chemical Name: TETRACHLORVINPHOS

Summary: Tetrachlorvinphos is an organophosphate insecticide used to treat livestock and household pets for pests. Tetrachlorvinphos has relatively low toxicity, but is considered to be a possible human carcinogen based on animal studies.

Uses and Sources: Approximately 661,000 pounds of tetrachlorvinphos active ingredient are used annually nationwide on over 480,000,000 animals, including cattle, horses, poultry, swine, cats, and dogs. Horses make up the largest percentage of treated animals (16%). There are no registered uses for food or feed crops.

Major Consumer Products: Tetrachlorvinphos is used in aerosols, collars, dips, shampoos, powders, and pump sprays to treat household pets for fleas and ticks. Products containing tetrachlorvinphos are sold under the trade names Rabon and Gardona.

Routes of Human Exposure: People can be exposed to tetrachlorvinphos through the use of household pesticide products. Young children may be at particular risk of exposure to this pesticide while it is being applied or by contacting treated pets. Exposure can also occur through consumption of meat, meat byproducts, milk, poultry, and eggs derived from treated livestock. According to EPA, drinking water is not likely to be contaminated with tetrachlorvinphos since it is used over small areas, and is not used to treat crops. Workers can be exposed to this pesticide while handling, mixing, loading, and applying the product.

Health Effects: Tetrachlorvinphos has relatively low toxicity, based on animal studies. The primary effect of exposure to this pesticide is cholinesterase inhibition. Exposed laboratory animals had reduced body weights, liver and thyroid effects, and increased kidney weights. No developmental or reproductive effects have been found in limited animal studies. Tetrachlorvinphos is considered to be a possible human carcinogen based on animal studies.

Detecting Exposure: Tetrachlorvinphos is rapidly metabolized and excreted in the urine and feces and does not appear to be stored in the body. The metabolite of tetrachlorvinphos reported in the CDC National Exposure Report is DMP.

Regulations for Protection of Human Health:

Air – Tetrachlorvinphos is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for tetrachlorvinphos.

Food – EPA has established tolerances ranging from 0.1 to 1.5 ppm for tetrachlorvinphos residues in a variety of foods and agricultural commodities.

Occupational Exposure – OSHA has not established a standard for tetrachlorvinphos in workplace air.

Source of Information:

EPA. Revised Human Health Risk Assessment for Tetrachlorvinphos. December 1999. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/tcvp.htm.

Chemical Name: TRICHLORFON

Summary: Trichlorfon is an organophosphate insecticide used primarily on ornamental plants, golf courses, lawns, and for ant control. Trichlorfon may cause developmental effects including decreased fetal body weight and delayed bone formation. It has also been found to cause gene mutations in cell systems, and is likely to cause cancer in humans at high doses.

Uses and Sources: According to EPA estimates, approximately 1 million pounds of trichlorfon active ingredient are used annually nationwide. It is used for controlling cockroaches, crickets, silverfish, bedbugs, fleas, cattle grubs, flies, ticks, leaf miners, and leafhoppers. This pesticide is no longer used on domestic food or feed products, but is used overseas to treat cattle. Some imported beef and veal products can therefore be sources of exposure to trichlorfon.

Major Consumer Products: The major registrant of trichlorfon products is the Bayer Corporation. They sell six insecticide products under the trade name Dylox to treat lawns, ornamental gardens, and nurseries.

Routes of Human Exposure: People can be exposed to trichlorfon when applying the pesticide to home lawns or ant mounds, by contacting treated surfaces, by drinking contaminated water, or through occupational exposure.

Health Effects: Trichlorfon causes cholinesterase inhibition and other effects on the human nervous system. It is acutely toxic by ingestion, with low toxicity by inhalation and dermal routes. Signs of acute trichlorfon exposure include vomiting, diarrhea, nausea, and blurred vision. Animals with long-term exposure to trichlorfon had decreased cholinesterase activity and at high doses, decreased body weight, anemia, and spleen enlargement. Trichlorfon has been shown to cause developmental effects in animal studies. Observed effects included decreased fetal body weights and delayed bone formation. Trichlorfon has also been found to cause gene mutations in cell systems. EPA has classified trichlorfon as not likely to cause cancer at low doses, but likely to cause cancer in humans at high doses.

Detecting Exposure: Animal studies have shown that trichlorfon is rapidly metabolized and eliminated. The metabolite of trichlorfon included in the CDC National Exposure Report is DMP.

Regulations for Protection of Human Health:

Air – Trichlorfon is not regulated in ambient air.

Drinking Water – EPA has not established a drinking water standard or health advisory for trichlorfon.

Food – EPA has established a tolerance of 0.1 ppm for trichlorfon residues in meat and meat byproducts.

Occupational Exposure – OSHA has not established a standard for trichlorfon in workplace air.

Sources of Information:

EPA. Revised Preliminary Human Health Risk Assessment for Trichlorfon. April 2000. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/trichlorfon/hedrra.pdf.

EPA. Toxicology Chapter for the Risk Assessment for the Reregistration Eligibility Decision Document for Trichlorfon. August 1999. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/op/trichlorfon.htm.

Pesticide Action Network Pesticide Database. Chemical Information about Trichlorfon. San Francisco, CA. 2002. Accessed online at http://www.pesticideinfo.org/PCW/Detail_Chemical.jsp?Rec_Id=PC33346.

HERBICIDES

Herbicides are chemicals used to kill unwanted plants. They are the most common type of pesticide used. EPA estimates that in 1997, 568 million pounds of herbicides and plant growth regulators were used in the U.S., of which more than 80% were used in agricultural crop production. Home and garden uses and other non-agricultural uses accounted for almost 100 million more pounds of herbicide use. The volume of herbicides used in agricultural production has remained fairly steady over the past 20 years, but home and garden uses have increased almost 50% (from 33 million pounds in 1979 to 49 million pounds in 1997).

Atrazine, metolachlor, glyphosate, 2,4-D, cyanazine, and alachlor are the most heavily used herbicides in the U.S. There are many different classes of herbicides, including triazines (e.g., atrazine, cyanazine), chloroacetanilides (alachlor and metolachlor), amides, benzonitriles, carbamates, thiocarbamates, chlorophenoxy (2,4-D and 2,4,5-T), and others. Chlorophenoxy herbicides are widely used for control of broadleaf weeds in agriculture and for control of vegetation along roadsides, railways, and utility rights-of-way and for maintenance of parks, golf courses, and on home lawns and gardens. The chlorophenoxy herbicides 2,4-D and dicamba are among the most commonly occurring pesticides found by EPA in U.S. public drinking water systems. The chlorophenoxy herbicide 2,4,5-T was removed from the market in 1969 due to concern about contamination with 2,3,7,8-TCDD (dioxin) during manufacture. 2,4,5-T, also known as Agent Orange, was used extensively in South Vietnam and Cambodia as a defoliant. 2,4-D, used in more than 1,000 different pesticide products, is not contaminated with dioxin.

Effects of Chronic Exposure to Herbicides

While the human health effects associated with chronic, low-level herbicide exposures are not yet well understood, a growing body of scientific evidence suggests that environmental exposures to different herbicide classes can have varying health effects. Adverse health effects associated with triazine and chlorophenoxy herbicides are described below.

Triazine Herbicides

The triazine pesticides simazine, cyanazine, and propazine are classified as possible human carcinogens by the EPA. Atrazine was also in the same category until it was recently downgraded by the EPA as not likely to be a human carcinogen. The International Agency for Research on Cancer (IARC) has stated that atrazine "is not classifiable as to its carcinogenicity in humans," but that there is "sufficient evidence in experimental animals for carcinogenicity of atrazine." Recent studies have suggested a possible association between exposure to triazine herbicides in drinking water and increased risk for breast cancer and stomach cancer. There is also limited epidemiological evidence of associations between triazine pesticide exposure and ovarian cancer and non-Hodgkin's lymphoma.

According to EPA, lifetime exposure to atrazine at certain levels has the potential to cause weight loss, cardiovascular damage, retinal and some muscle degeneration, and mammary

tumors. Several recent studies have added to the evidence that atrazine and other triazine herbicides are endocrine disrupters. Long-term human exposure to simazine can cause tremors; damage to testes, kidneys, liver, and thyroid; and gene mutations. An epidemiological study in Iowa found an association between maternal exposure to triazine herbicides in drinking water and intrauterine growth retardation and low birth weight.

Chlorophenoxy herbicides

Chlorophenoxy herbicides are mainly irritants. Chlorophenoxy herbicides do not build up in the body. However, according to EPA, chronic exposure to 2,4-D above the federal drinking water standard has the potential to cause damage to the nervous system, kidneys, and liver. Agricultural use of 2,4-D has also been linked to mortality from several types of cancer, although EPA has classified 2,4-D as having "inadequate evidence for carcinogenicity in humans." Epidemiological studies have also reported an association between exposure to 2,4-D and non-Hodgkin's lymphoma. Chronic exposure to 2,4-D has also been linked to immune system suppression and endocrine disruption.

Sources of Information:

EPA. Office of Pesticide Programs. Revised Preliminary Human Health Risk Assessment for Atrazine. Accessed online at http://www.epa.gov/oppsrrd1/reregistration/atrazine/index.htm

EPA. Office of Ground Water and Drinking Water. National Primary Drinking Water Regulations, Technical Fact Sheet on Atrazine. Accessed online at http://www.epa.gov/OGWDW/dwh/t-soc/atrazine.html

EPA. Office of Ground Water and Drinking Water National Primary Drinking Water Regulations, Technical Fact Sheet on Simazine. Accessed online at http://www.epa.gov/OGWDW/dwh/t-soc/simazine.html.

EPA. Office of Ground Water and Drinking Water. National Primary Drinking Water Regulations, Technical Fact Sheet on 2,4-D. Accessed online at http://www.epa.gov/OGWDW/dwh/t-soc/24-d.html.

IARC. Monographs on the evaluation of carcinogenic risks to humans: atrazine. Volume 73: 59. (1999).

Physicians for Social Responsibility. "Drinking water fact sheet #6, Atrazine: What health care providers should know."

Physicians for Social Responsibility. "Drinking water fact sheet #5, Pesticides: What health care providers should know."

Chemical Name: **ATRAZINE**

Summary: Atrazine is the most commonly used agricultural pesticide in the U.S. As a result of this heavy use, atrazine is a common drinking water contaminant in agricultural regions of the country. In heavily agricultural regions, such as the Midwest corn belt, atrazine in drinking water poses a serious health threat to infants, children, and adults. People are also exposed to atrazine residues in the diet and through home use of atrazine lawn products. EPA has determined that contact with wet, treated turf grass can pose health risks to children as well as adults. There is clear evidence from human and animal studies that atrazine disrupts normal hormonal function and more controversial evidence that atrazine may cause cancer. Although human and animal studies have linked atrazine with increased cancer risk, EPA has classified atrazine as "not likely to be carcinogenic to humans" due to lack of a clear biological mechanism. Animal studies have also shown that atrazine exposure can lead to reduced immune function, liver and kidney damage, and abnormal reproduction and development.

Uses and Sources: Atrazine is a triazine herbicide used primarily for weed control on corn, sorghum, and sugarcane. Atrazine is also sold for weed control on residential lawns, golf courses, and public areas. EPA has estimated that 64-75 million pounds of atrazine (active ingredient) are used annually in the U.S. Because of its heavy use, atrazine is the most commonly detected pesticide in ground water and surface water and thus poses a significant threat to drinking water supplies.

Major Consumer Products: Used in many lawn and garden products including St. Augustine lawn weed killer and herbicides formulated by Drexel.

Routes of Human Exposure: For the general public, the most significant routes of exposure to atrazine are consumption of contaminated drinking water and dietary exposures. In agricultural areas such as the Midwest corn belt, levels of atrazine and its chlorinated metabolites often increase dramatically in drinking water in spring and early summer (following spring application of the pesticide). Use of atrazine products on home lawns can also lead to exposure by skin contact and incidental pesticide ingestion by children. Workers involved in mixing, loading, and applying atrazine can have higher levels of exposure than the general public.

Health Effects: Atrazine has low acute toxicity, but there is evidence from animal studies that short-term, high-dose exposures can cause effects including delayed bone formation in fetuses, delayed puberty in young animals, and decreased weight gain in adult animals. There is clear evidence from both human and animal studies that atrazine disrupts normal hormone function, potentially leading to many other adverse health effects. In animal studies, atrazine reduced immune system activity, caused liver and kidney damage, and interfered with normal reproduction and development.

The evidence regarding potential carcinogenicity of atrazine is highly controversial. Several epidemiological studies and numerous laboratory animal studies have found associations between atrazine exposure and cancer. However, EPA has concluded that

there is insufficient evidence of a biologically plausible mechanism relevant to humans, and has classified atrazine as not likely to be carcinogenic in humans.

Risk assessments conducted by EPA suggest that intermediate and chronic exposure to atrazine and its metabolites in drinking water may pose unacceptable health risks to infants, children, and adults. EPA also concluded that contact with wet, treated turf grass is a potential health risk for children and adults. There is evidence that atrazine shares a common mechanism of toxicity with other triazine herbicides, indicating that low-level exposure to more than one of these pesticides could have the same adverse effect as a higher dose of a single chemical. EPA plans to conduct a cumulative risk assessment for the triazine pesticides in the future.

Detecting Exposure: The metabolite atrazine mercapturate is a biomarker of atrazine exposure and can be measured in urine.

Regulations for Protection of Human Health:

Air – Atrazine is not regulated in ambient air.

Drinking water – EPA has established an MCL of 0.003 mg/L of atrazine in drinking water.

Food – EPA has established tolerances ranging from 0.02 to 15 ppm for atrazine residues on crops and other agricultural products.

Occupational exposure – OSHA has not established a standard for atrazine in workplace air.

Sources of Information:

EPA. Atrazine: Revised Human Health Risk Assessment for the Reregistration Eligibility Decision (RED). April 2002. Accessed online at http://www.epa.gov/oppsrrd1/reregistration/atrazine/index.htm.

Cox, C. Atrazine Toxicology. Journal of Pesticide Reform 21(2):12-20 (2001).

Pesticide Action Network Pesticide Database. Chemical Information about Atrazine. San Francisco, CA. 2002. Accessed online at http://www.pesticideinfo.org/PCW/Detail_Chemical.jsp?Rec_Id=PC33440.

Physicians for Social Responsibility. “Drinking water fact sheet #6, Atrazine: What health care providers should know.”

Chemical Name: **2,4-DICHLOROPHENOXYACETIC ACID (2,4-D)**

Summary: 2,4-D, a chlorinated phenoxy compound, is a systemic herbicide used to control many types of broadleaf weeds. It may be found in emulsion form, in aqueous solutions (salts), and as a dry compound. Because 2,4-D is commonly found in products used around the home, the possibility of everyday exposure is high. 2,4-D's ability to cause cancer is controversial. The available studies have shown evidence supporting both sides of the issue. The product Agent Orange, used extensively throughout Vietnam, was about 50% 2,4-D. However, the controversies associated with the use of Agent Orange were associated with a contaminant (dioxin) in the 2,4,5-T component of the defoliant.

Uses and Sources: 2,4-D is widely used to control weeds in agriculture and in the home and garden. It is also used in pasture and rangeland applications, forest management, and to control aquatic vegetation. EPA estimates that about 29-33 million pounds of 2,4-D are used each year for weed control on agricultural crops (e.g., wheat, corn, orchard crops). More 2,4-D is used in Hawaii on the sugar cane crop than in any other state. It is also the most heavily used pesticide in non-agricultural sectors of the U.S., accounting for an additional 7-9 million pounds sold in the home and garden market and 16-18 million pounds in industry, commercial, and government sectors.

Major Consumer Products: 2,4-D is used in many lawn and garden products, including Weed-B-Gon and weed killers and fertilizers formulated by Scott's and Gordon's.

Routes of Human Exposure: People are exposed to 2,4-D mainly by direct contact when using a weed-control product around the home. Consuming contaminated food or drinking contaminated water are possible routes. Dermal contact is a major route of exposure to 2,4-D, which is rapidly absorbed through the skin.

Health Effects: 2,4-D has low to moderate toxicity to humans but can cause severe irritation to the eyes. Symptoms of acute exposure to 2,4-D include nausea, vomiting, diarrhea, headache, confusion, weight loss, and damage to the liver, kidneys, and nervous system. There is also evidence that 2,4-D can cause muscle weakness, involuntary muscle contractions, numbness, pain in the arms and legs, and loss of coordination.

Animal studies have shown that 2,4-D can pass through the placenta, with detectable levels in the fetus. An epidemiologic study of male farmers suggests that 2,4-D exposure can cause reproductive damage, including reduced sperm counts, abnormal sperm, lower birth rates, and higher rates of birth defects in offspring. Some studies in laboratory animals exposed to high doses of 2,4-D showed evidence of reproductive harm and birth defects, and other studies did not.

Controversy surrounds the carcinogenic potential of 2,4-D. EPA has classified 2,4-D as "not classifiable as to human carcinogenicity." IARC has classified it as "possibly carcinogenic." Laboratory animals exposed to 2,4-D have developed cancer, but more recent studies have not shown an association between 2,4-D exposure and cancer in

laboratory animals. Epidemiology studies have been unclear regarding exposure to 2,4-D and cancer in humans. A major problem with some of these epidemiological studies is that exposure to 2,4-D could not be differentiated from exposure to other compounds, including dioxin. However, evidence does suggest that persons exposed to 2,4-D may be at increased risk for developing non-Hodgkin's lymphoma, and studies that look further at that association are ongoing.

Detecting Exposure: 2,4-D is excreted primarily unchanged through the urine. Available studies indicate that most 2,4-D is excreted within several days of exposure. Evidence does not show that 2,4-D accumulates in the body.

Regulations for Protection of Human Health:

Air – 2,4-D is regulated by EPA as Hazardous Air Pollutant under the Clean Air Act.

Drinking water – EPA has established an MCL of 0.07 mg/L for 2,4-D in drinking water.

Food – EPA has established tolerances for residues of 2,4-D on a wide variety of foods and agricultural products. Tolerances for specific crops can be found by searching EPA's online database at http://www.epa.gov/pesticides/food/viewtols.htm. EPA has set a tolerance of 1 ppm 2,4-D in fish.

Occupational exposure – OSHA has established a PEL of 10 mg/m3 for 2,4-D in workplace air.

Sources of information:

Cox, C. 2,4-D: Toxicology. Journal of Pesticide Reform 19:1-2 (1999).

EPA. Consumer Fact Sheet on 2,4-D. Accessed online at http://www.epa.gov/safewater/dwh/c-soc/24-d.html

EPA. Integrated Risk Information System. 2,4-Dichlorophenoxyacetic acid (2,4-D) (CASRN 94-75-7). Accessed online at http://www.epa.gov/iriswebp/iris/subst/0150.htm - carc.

EPA. Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds. Volume 2. Sources of Dioxin-Like Compounds in the U.S. Draft Final Report. Office of Research and Development, Washington, DC September 2000. Accessed online at http://www.epa.gov/NCEA/pdfs/dioxin/index.htm

Extension Toxicology Network (EXTOXNET). Pesticide Information Profile: 2,4-D. June 1996. Accessed online at http://ace.orst.edu/cgi-bin/mfs/01/pips/24-D.htm

Krieger, RI and J Doull. Handbook of pesticide toxicology, 2nd ed. Chapter 72: Phenoxy Herbicides (2,4-D). Academic Press, San Diego. 2001.

National Pesticide Information Center. 2,4-D Fact Sheet. Accessed online at http://npic.orst.edu. http://ace.orst.edu/info/npic/factsheets/2_4-D.pdf

Chemical Name: **ALACHLOR**

Summary: Alachlor is a restricted use pesticide, commonly used to control weeds on corn and other agricultural crops. For the general population, exposure to alachlor occurs primarily by eating foods treated with this herbicide or by drinking water from contaminated supplies. Animal studies have shown that chronic exposure to alachlor, particularly at high doses, can cause cancer, liver damage, eye lesions, and other effects. These effects have not been observed in people occupationally exposed to alachlor, and EPA has found no evidence that alachlor poses a health threat to the developing fetus or young children.

Uses and Sources: Alachlor, a broad-spectrum herbicide in the acetanilide family, is used for weed control on corn, soybeans, sorghum, peanuts, and beans. Alachlor is produced by Monsanto Company and sold under the trade names "Lasso" and "Alanex." EPA has estimated that approximately 13-16 million pounds of alachlor are used in U.S. agricultural crop production each year (1997 data). In the environment, alachlor and its degradation by-products are highly mobile and pose a significant threat to groundwater supplies. Based on concerns for groundwater quality, EPA has classified alachlor as a restricted use pesticide. Alachlor is not available for purchase or use by the general public, and has no uses in the home, schools, parks, or other public areas.

Major Consumer Products: As a restricted use pesticide, alachlor is not available for purchase or use by the general public.

Routes of Human Exposure: The general public can be exposed to low levels of alachlor by eating foods with residues of this pesticide or by consuming contaminated drinking water. Workers involved in mixing, loading, and applying alachlor have a higher risk of exposure than the general public.

Health Effects: Animal studies have shown evidence that chronic exposure to alachlor, particularly at higher doses, can cause cancer, liver damage, eye lesions, and other adverse effects. However, epidemiological studies of workers exposed to alachlor found no evidence for these effects in humans. Alachlor has been classified by EPA as likely to cause cancer at high doses, but not likely to cause cancer at low doses. EPA has concluded from animal studies that alachlor is not likely to pose a significant health risk to the developing fetus, infants, or children. There is evidence that alachlor may share a common mechanism of toxicity with certain other pesticides (i.e., acetochlor, metolachlor, butachlor and propachlor), indicating that exposures to combinations of these pesticides could increase health risk.

Detecting Exposure: Alachlor mercapturate is a biomarker of alachlor exposure and can be measured in urine.

Regulations for Protection of Human Health:

Air – Alachlor is not regulated in ambient air.

Drinking water – EPA has established an MCL of 0.002 mg/L for alachlor in drinking water.

Food – EPA has established tolerances ranging from 0.02 to 3 ppm for alachlor residues in a variety of foods and agricultural products.

Occupational Exposure – OSHA has not established a standard for alachlor in workplace air.

Sources of Information:

EPA. Registration Eligibility Decision (RED): Alachlor. Office of Prevention, Pesticides and Toxic Substances. December 1998. Accessed online at http://www.epa.gov/oppsrrd1/REDs/0063.pdf.

EPA. Pesticides Industry Sales and Usage. 1996 and 1997 Market Estimates. Office of Prevention, Pesticides and Toxic Substances. (1999). Accessed online at http://www.epa.gov/oppbead1/pestsales/97pestsales/index.htm

FUNGICIDES

Fungicides are used to control or destroy fungi. Many fungi can infect and cause diseases in plants, animals, and people. Examples of disease-causing fungi include rusts, mildews, blights, and molds. In American agriculture, metam sodium, copper hydroxide, chorothalonil, and mancozeb are among the most heavily used fungicides. Fungicide use in the U.S. has declined significantly, from 124 million pounds in 1979 to 81 million pounds in 1997.

The only fungicide chemical tested in the 2003 National Exposure Report is ortho-phenylphenol. Ortho-phenylphenol (or 2-phenylphenol, o-phenylphenol) is used as an active ingredient in fungicides, disinfectants, and some insecticides. EPA classifies it as having high acute toxicity and as a probable human carcinogen; however, the International Agency for Research on Cancer (IARC) has called it unclassifiable (meaning there are not enough data). EPA, in their Toxics Release Inventory (TRI), classifies o-phenylphenol as a developmental toxin, meaning that exposure may harm a developing fetus.

Ortho-phenylphenol is used in disinfectants in animal husbandry, hospitals, restaurants, and other institutional uses; household disinfectants; fungicides on fruits and vegetables to protect against microbial damage; and in some insecticide products. Common consumer uses include household disinfectants, such as Lysol brand disinfectants and insecticides such as Raid Ant and Roach Killer. The general population can be exposed to o-phenylphenol by inhalation, ingestion of contaminated food and drinking water, and by skin contact with household disinfection products. O-phenylphenol can be measured in adipose tissue and blood serum.

Effects of Chronic Exposure to Fungicides

For many pesticides, including fungicides, our understanding of human health effects is based largely on occupational or accidental exposure or laboratory animal studies in which doses can be relatively high. Effects such as weight loss, organ damage, muscle degeneration, neurological damage, and cancer have been documented based on such studies. EPA classifies o-phenylphenol as a probable human carcinogen. However, environmental exposures to pesticides usually involve relatively low concentrations that may occur over long periods of time. While the human health effects associated with chronic, low-level pesticide exposures are not yet well understood, a growing body of scientific evidence suggests that environmental pesticide exposures are associated with neurological and reproductive damage, effects on growth and development, birth defects, endocrine disruption, cancer, and other adverse effects. Fungicides are frequently comprised of a large variety of different compounds, so health effects vary accordingly. Of course, different chemicals may cause different adverse effects to human health, or none at all.

Sources of Information:

Hazard Information on Toxic Chemicals Added to EPCRA Section 313 Under Chemical Expansion. Accessed online at http://www.epa.gov/tri/chemical/hazard_cx.htm.

Hazardous Substances Data Bank (HSDB). Accessed online at http://toxnet.nlm.nih.gov/cgi-bin/sis/

Pesticide Action Network Pesticide Database: ortho-phenylphenol. Accessed online at http://www.pesticideinfo.org/PCW/Detail_Chemical.jsp?Rec_Id=PC33048

PEST REPELLENTS

Flies, gnats, mosquitoes, and ticks, in addition to being annoying by flying around the face and leaving itchy bites, pose health risks to individuals. These range in severity from the swelled bite of the biting fly to the transmission of Lyme's disease or Rocky Mountain spotted fever by certain ticks. In recent years, mosquitoes have also spread an increasing number of West Nile virus cases throughout the U.S. Insect repellents are seen as the primary method of warding off biting insects.

Repellents can be lotions, aerosol sprays, liquids, and sticks. They are applied directly to exposed skin or clothing. Products that contain the synthetic chemical DEET are the most effective and most well-known available on the market—Off! and Muskol, among others, contain DEET. Some other naturally occurring chemical repellents (from botanical sources) are citronella and eucalyptus oil. These can be found in numerous products: Avon's Skin So Soft, Bug Guard, Bite Blocker, and Buzz Away.

The EPA has certain recommendations for the application of insect repellents to ensure best effectiveness of the product and minimal exposure to the chemicals. Applying the repellent to exposed skin and clothing, never applying it to open wounds or cuts, applying it in a very thin layer, and washing treated skin after the outdoor activity are steps that will minimize absorption of the chemical while repelling insects.

The CDC's National Exposure Report examined the body burdens of three chemicals found in insect repellents. DEET is a well-known and long-used chemical that is a very effective repellent; it is profiled in this report. 2-Naphthol was used for many years as an insecticide, pesticide, and repellent. All 13 product registrations containing 2-Naphthol have been cancelled since the mid-1980s. All of these products carried cautions or warnings of either their slight or moderate toxicity; however, the toxicity of these products was caused by the combination of chemicals in these products, rather than 2-Naphthol alone. 2-Naphthol is also a metabolite of 1-Naphthol, a chemical in carbamate insecticides. 2,5-Dichlorophenol is a chemical found in agricultural pesticides and veterinary products. It is also the metabolite of 1,4-Dichlorobenzene, an insecticide popularly used to kill clothes moths and repel other insects. Neither 2-Naphthol or 2,5-Dichlorophenol is profiled in this report.

Sources of Information:

EPA. *How to Use Insect Repellents Safely*. Office of Pesticide Programs. Accessed online at http://www.epa.gov/pesticides/citizens/insectrp.htm.

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Chemical Name: DEET (DIETHYLTOLUAMIDE)

Summary: DEET is the well-known shortened name of the chemical N, N-diethyl-m-toluamide. DEET is a very effective multipurpose insect repellent. It is also distinct in being one of the few pesticides that can be applied directly to bare skin. For the past 40 years, it has been used in the house and for leisure activities to repel insects from people and pets. EPA believes that the normal use of DEET does not present a health concern to the general population. However, reports of skin disorders and toxic encephalopathy in children potentially associated with exposure to DEET emphasize the need for the proper use of pest repellents containing DEET. Following manufacturers' instructions for use of DEET-containing products is recommended to avoid excess exposure. In particular, children should not be exposed to large amounts of DEET. Local health departments are often a good source of information about the use of pest repellents in general and DEET-containing products in particular.

Uses and Sources: DEET is a synthetic chemical that repels, but does not kill, insects by an unknown mechanism. It is used to control biting flies and midges, black flies, chiggers, deer flies, fleas, gnats, horse flies, mosquitoes, sand flies, and ticks. Virtually all human exposure to the chemical comes from direct personal application, as this is DEET's only use.

Major Consumer Products: There are approximately 225 DEET products registered for use in the U.S. as insect repellents. Products that are applied directly to the skin, clothes, and pets are available in liquids, impregnated materials, skin lotions, and aerosols.

Routes of Exposure: Due to its limited use pattern, exposures to DEET come only from personal applications. It is not used on, and therefore, not present in food. In the environment, DEET breaks down slowly in soil but fairly quickly in the atmosphere. Very few studies have examined the environmental fate of DEET, however, its small-scale use translates into negligible environmental concentrations.

Human exposures can come through dermal absorption after a normal application of an insect repellent to exposed skin. The lipophilic nature of the chemical allows it to enter into the bloodstream very quickly through this route. Accidental exposures can also come from DEET sprays that are absorbed through the eyes, liquids that are swallowed, and aerosols that are inhaled. The body quickly metabolizes DEET, with most of the chemical is eliminated in the urine within 12 hours.

Health Effects: DEET is a residentially applied pesticide. It has therefore received significant scientific scrutiny over the past 50 years. Although generally safe under occasional usage, DEET-containing products have been associated with some adverse health effects. Dermal application of insect repellents containing DEET can produce a variety of skin reactions, including localized skin irritation. Dermatitis has been noted in both children and adults. Several cases in children of toxic encephalopathy associated with DEET skin exposure have been reported in the medical literature. DEET has not been found to cause birth defects. EPA has classified DEET as a group D carcinogen (not

classifiable as to human carcinogenicity). DEET is only slightly toxic in the eye and through the oral and dermal routes.

Acute exposures from improper and excessive use have caused neurological and cardiovascular effects. Children are of concern as their greater surface area-to-volume makes them more susceptible to toxic poisoning. EPA is no longer allowing child safety claims on product labels.

Commercial insect repellents contain DEET concentrations that range from 5% to 100% DEET by volume. As absorption of DEET into the bloodstream increases at the higher concentrations, one can avoid potentially high exposures by using the appropriate repellent and following instructions on the labels of DEET-containing products. The American Academy of Pediatrics recommends that repellents used on children contain less than 10% DEET. For casual use in adults, DEET concentrations above 35% are not needed.

Detecting Exposure: The metabolism of DEET in the body is fairly rapid and complete. Elimination usually occurs in the urine within several days. Exposure to DEET and its metabolites can be detected in the urine during this time period.

Regulations for Protection of Human Health:

Air – DEET is not regulated in ambient air.

Drinking water – EPA has not established a drinking water standard for DEET.

Food – FDA has not established limits for DEET residues in food at this time.

Occupational exposure – OSHA has not established a standard for DEET in workplace air.

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