

Persistent Organic Pollutants and Human Health



A Publication of the

World Federation of

Public Health Associations'

Persistent Organic

Pollutants Project

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Persistent Organic Pollutants (POPs) and Human Health

A PUBLICATION OF THE WORLD FEDERATION OF PUBLIC HEALTH
ASSOCIATIONS' PERSISTANT ORGANIC POLLUTANTS PROJECT

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I. Summary

In 1992, the world's governments met in Rio de Janeiro to collectively confront environmental problems that are now global in nature. Among those selected for long term planning and resolution was the problem of global pollution by a group of industrial chemicals known as persistent organic pollutants, or "POPs". POPs are organic chemical compounds that are highly toxic, persist in the environment, bio-accumulate in fatty tissues of living organisms, travel long distances, and naturally migrate toward colder climates. Beginning in 1998, 103 governments began negotiations to establish a global, legally binding agreement to reduce or eliminate the health and environmental threats posed by POPs, with a target completion date of fall, 2000.

The twelve POPs designated as targets for early global action are all chlorine-containing organic compounds. They are aldrin and dieldrin, endrin, chlordane, DDT, heptachlor, mirex, toxaphene, hexachlorobenzene, polychlorinated biphenyls (PCBs), polychlorinated dibenzodioxins, and polychlorinated dibenzofurans.

POPs residues have been found in the fat of fish and animals, as well as in human breast milk, on a global scale. Some of the highest levels have been recorded in the arctic areas of both hemispheres.

Reproductive failures, deformities, malfunctions in fish and wildlife are linked by a growing body of evidence to these persistent pollutants. Often the true extent of the wildlife effects are subtle, and can be triggered at extraordinarily low concentrations. In 1991, the Science Advisory Board to the International Joint Commission on the Great Lakes of the U.S. and Canada, reviewed the literature on the effects of POPs on more than a dozen Great Lakes predator species including eagles, cormorants, trout, mink, turtles and others. Their report found that all these species suffered significant health impacts including some combination of: population decline and reproductive dysfunction; eggshell thinning; metabolic changes; deformities and birth defects; tumors and cancers; behavioral changes; abnormally functioning thyroids and other hormone system dysfunction; immune suppression; feminization of males and masculinization of females.

Humans are generally exposed to POPs through their food supply. A growing body of scientific evidence associates human exposure to individual POPs with cancer, neurobehavioral impairment, immune system biochemical alterations and possibly dysfunction, reproductive dysfunction, shortened period of lactation, and diabetes. The mechanism for many of these effects appears to be through disruption of the human endocrine system, often, during fetal development.

Physicians and public health professionals around the world are seen as opinion leaders and trusted voices in policy debates. As diplomats negotiate an international POPs treaty that will affect the health of people throughout the planet, they will be looking to the medical community for guidance and expertise. The world's public health associations and their individual members can play an important role in facilitating debate on realistic response strategies, policies and mechanisms for eliminating emissions, reducing reliance on POPs, and replacing them with safer alternatives. Health professionals have a special responsibility in this debate as well. Health care institutions are currently a major source of POPs exposure due, in part, to the use of disposable products.

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II. Introduction

The first truly global environmental summit was held in Brazil in 1992. The governments in attendance agreed to work jointly to solve specific global environmental problems that had a potential for radically changing human existence.¹ Among those threats targeted for long-term planning and action was the increasing pollution produced by a group of chemicals known as persistent organic pollutants, or POPs. POPs are carbon-based (organic) chemical compounds and mixtures that are highly toxic, persist in the environment, bioaccumulate in fatty tissues of living organisms, travel long distances in air and water, and tend to migrate from warmer to colder regions of the world.²

POPs, the product and by-product of human activities, are of relatively recent origin. Prior to mid-twentieth century, pollutants with these harmful properties were virtually non-existent in the environment or in food. The production and generation of POPs began in earnest in the years following World War II with the increased production of chemicals and their by-products. They have since become ubiquitous pollutants, found in environments worldwide.³

The twelve POPs designated as targets for early global action are all chlorine-containing organic compounds. They are aldrin and dieldrin, endrin, chlordane, DDT, heptachlor, mirex, toxaphene, hexachlorobenzene, polychlorinated biphenyls (PCBs), polychlorinated dibenzodioxins, and polychlorinated dibenzofurans. All of these compounds have been banned or have been subject to intense restriction in many countries, yet due to their persistence, effects at low levels, and global reach, they remain serious threats.⁴

National boundaries pose no barrier to POPs. Even some countries that have worked diligently to restrict and eliminate some of their domestic POPs sources continue to endure health and environmental injury caused by POPs that originate far away.⁵ It has become clear that no government, acting alone, has the power to enact measures that will protect the health of its population or its national envi-

ronment from POPs. It is now agreed that a remedy to the POPs problem requires enactment of global, intergovernmental measures.

Some regional international agreements on POPs have been negotiated. Most recently, under the auspices of the UN Economic Commission for Europe (UN/ECE), thirty-two European and North American countries signed the Aarhus Protocol to the ECE's Convention on Long-Range Transboundary Air Pollution (LRTAP). The objective of the Aarhus Protocol is to control, reduce, or eliminate discharges, emissions and losses of persistent organic pollutants. The agreement will ban nine POPs outright and reduce emissions of seven others.⁶

On a global level, the United Nations Environmental Program (UNEP) began a process in the early 1990s of examining the risks posed by POPs, as well as strategies to phase out the most hazardous of these. In June 1996, the Intergovernmental Forum on Chemical Safety (IFCS) submitted a final report to UNEP and the World Health Assembly (WHA), the governing body of the World Health Organization. The final report recommended immediate international action to protect human health and the environment from POPs.⁷

A February 1997 decision of the UNEP Governing Council called on the governments of

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the world to enter negotiations to establish a global, legally binding agreement to reduce or eliminate the health and environmental threats posed by POPs.⁸ In May 1997, delegates of the World Health Assembly unanimously agreed.

The first meeting of an Intergovernmental Negotiating Committee (INC) on POPs took place in June 1998 in Montreal, Canada, with representatives of 92 countries in attendance. The INC aims to reach a binding, international agreement by the year 2000. The mandate of the INC is to develop a global, legally binding program of action to:

- Reduce and eliminate releases and sources of the twelve well-known and well characterized POPs identified by IFCS and UNEP;
- Develop criteria and a procedure for identifying additional persistent organic pollutants beyond the initial twelve, which would then become candidates for future global action under the agreement;
- Address socio-economic factors that may arise in the implementation of global action on POPs;
- Consider how measures to restrict or eliminate POPs might affect food production, vector control, or otherwise harm human health or well-being;
- Consider the need for capacity-building as well as financing concerns and opportunities in various countries and regions, and also consider possible trade impacts.⁹

Progress was made on all fronts, with delegates overwhelmingly supporting the completion of an international, legally binding instrument on POPs by the year 2000 through an open and transparent process with input from the full range of stakeholders.

In January, 1999, the second INC meeting occurred in Nairobi, Kenya, with 103 countries present. The delegates made progress in defining the issues needing clarification in the next year. A draft document was prepared and reviewed for consultation with home governments.

INC3 was held in September, 1999, in Geneva, Switzerland. The key decisions of this meeting were an agreement to eliminate production and use of the pesticides aldrin, endrin and toxaphene without exemptions, and an agreement to phase out chlordane, dieldrin, heptachlor, mirex and hexachlorobenzene, with consideration of limited country-specific exemptions. Unresolved at this meeting were the elimination and phase-out of PCBs, dioxins, and furans, and the continued use of DDT, the discussions of which generated significant controversy. Technical and financial assistance for POPs phase outs and related activities were also on the agenda, as well as discussions of criteria for adding new chemicals to the POPs list for future action. These issues were deferred for ongoing discussion after INC3 and during INC4.¹⁰

The fourth intergovernmental meeting in the POPs process (INC4) was completed in March, 2000, in Bonn, Germany. Although it appears that some progress was made in keeping critical language regarding elimination and precaution in the draft Treaty text, ongoing contention over several key issues including the fate of chemical stockpiles, who will fund elimination and prevention efforts in developing countries, and the relationship of the POPs treaty to other multilateral agreements. The World Trade Organization¹¹ made it clear that a strong NGO presence continues to be critical as this process advances.

Notes

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6. The 1998 Aarhus Protocol on Persistent Organic Pollutants (POPs), The 1979 Geneva Convention on Long-range Transboundary Air Pollution (LTRAP), United Nations Economic Commission for Europe (UN/ECE), Geneva, Switzerland, 1998.
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III. The Global POPs Problem



POPs differ in a number of ways from most conventional pollutants. Other pollutants tend to remain close to their sources, and often can be effectively controlled through measures that reduce inputs to levels that then dilute and are assimilated without harm. POPs, on the other hand, tend to travel long distances, and upon entering ecosystems, tend not to dilute but rather to build up through the food chain, accumulating in the tissues of mammals. This process is called bioaccumulation. POPs not only bioaccumulate, they also increase in intensity as they move up the food chain. This process is called biomagnification.

The twelve POPs are semi-volatile and evaporate relatively slowly. Persistent substances with this property tend to enter the air, travel long distances on air currents, and then return to earth. They may repeat this process many times as they “jump” north. The colder the climate, however, the less POPs tend to evaporate, resulting in their accumulation in the polar regions, thousands of kilometers away from their original sources. This means that any release to the environment represents a potential global threat.

Air emissions are becoming the major source of POPs. A recent report from the Canadian Chemical Producers Association found that from 1992 to 1994, total Canadian water emissions of POPs by member companies were halved, but the proportion of POPs in air emissions almost doubled.¹² In the U.S., according to the U.S. Toxic Release Inventory¹³, 73 percent of total releases of POPs from manufacturing in the Great Lakes basin were emitted into the air. This number will undoubtedly increase when emissions from electric power utilities and municipal incinerators are also reflected in the totals.

This is a reflection of the fact that abatement strategies and programs that may have proven useful for controlling conventional pollution are often of little use as solutions to POPs pollution. This lesson applies not only to the ma-

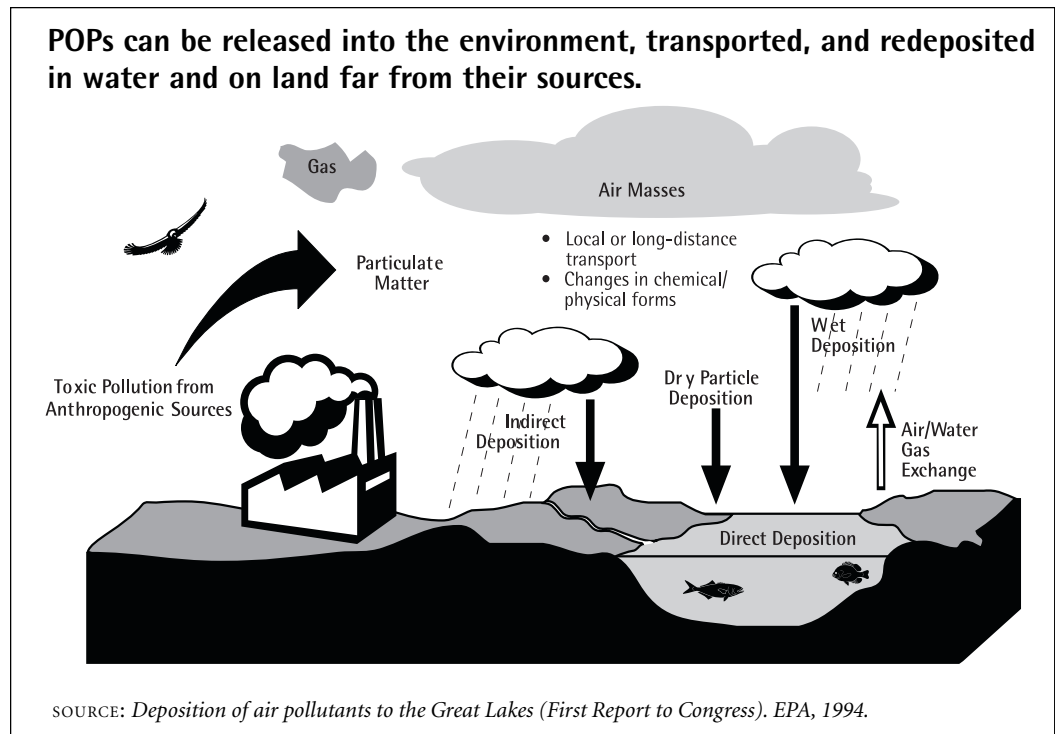
rine and other aquatic ecosystems, but to terrestrial ecosystems as well.

Effects of POPs in the global environment

The effects of POPs in the global environment have been widespread, affecting broadly disparate regions from the Great Lakes of North America to the arctic regions in both hemispheres. Moreover, effects that were once seen primarily in wildlife species are now being observed in some human populations.

Effects on Wildlife. One of the difficulties of studying the effects of individual POPs in wildlife populations is that the effects are rarely isolated or singular. In 1991, the Science Advisory Board (SAB) to the International Joint Commission reviewed the literature on the effects of POPs exposures on more than a dozen Great Lakes predator species including eagles, cormorants, trout, mink turtles and others. Their report found that all these species suffered significant health effects including some combination of: population decline and reproductive effects; eggshell thinning; metabolic changes; deformities and birth defects; tumors and cancers; behavioral changes; abnormally functioning thyroids and other hormone system dysfunction; immune suppression; feminization of males and masculinization of females.¹⁴

Air emissions are becoming the major source of POPs.



In addition, the SAB report found that POPs were associated with a variety of disruptions in the endocrine systems of animals in wildlife, including birds, fish, shellfish, turtles, and mammals.¹⁵ Two years later, the IJC wrote in its 1993 *Biennial Report*: “Research has shown persistent chemicals . . . to be strongly implicated in the disruption of endocrine systems, including estrogenic effects, in laboratory animals and in wildlife. . . .¹⁶ These disruptions in the endocrine system seem to have the greatest affect on the embryo, fetus or perinatal organism, as opposed to an adult¹⁷ Then

again, in 1996, the IJC reported that “(R)eproductive failures, deformities and physiological malfunctions in Great Lakes fish and wildlife is [sic] linked by a growing body of evidence to various pesticides, PCBs, dioxins, furans and similar substances.”¹⁸

The first report on organochlorine contamination of Arctic marine mammals was in

1970, when the pesticides dieldrin and DDT, as well as PCBs, were detected in the blubber of ringed seals from Baffin Island. A few years later, more detailed reports on DDT-related compounds and PCBs in ringed seals and beluga whales were published. Levels of POPs have since been discovered in polar bears,¹⁹ caribou, mink, and terrestrial birds in the Arctic.²⁰ A 1988 study found metabolites of the man-made insecticide chlordane in penguins in Antarctica, thousands of miles from the chemical’s sources.²¹ The Arctic and sub-Arctic regions have some of the lowest population densities of the world, and have therefore been considered pristine because of their remoteness. In fact, however, the region has become a sink for persistent contaminants, which have been detected in Arctic air, surface seawater, suspended sediments, snow, fish, marine mammals, sea birds, and terrestrial plants and animals.²² Many of these contaminants are POPs pesticides and industrial chemicals, which, while banned or restricted for use in most northern industrialized countries, continue to be used in developing nations.²³



Effects in Humans. Evidence of the effects of POPs on wildlife prompted research into whether these chemicals were also affecting humans. Humans are generally exposed to POPs through their food. Foods rich in animal fat, such as meats, fish, and dairy products are the most important means of exposure. A recent study in the U.S. reported findings of detectable levels of several POPs in common fast foods like hamburgers, pizza, and ice cream.²⁴ Workers and residents of communities near POPs sources can also be exposed through inhalation and dermal contact. In the Arctic, high levels of some POPs have been found in fish, seals, and whales, all significant foods in the diets of many northern indigenous peoples.²⁵ Chemical contamination of these traditional foods provides a critical path of contamination to these populations.

The health effects of POPs are generally subtle, and can be triggered at extraordinarily low concentrations. The latency period for POPs may be very complex. Not only can there be many years between exposure and outcome in the exposed individual, but in some cases there is a trans-generational leap from exposure to outcome; that is, exposure in the parent is observed by effects in the offspring.^{26, 27, 28} Without precise information concerning exposures, the relationship between exposure and effect is often difficult to characterize. This creates a significant barrier not only to diagnoses of health outcomes related to POPs exposures, but significantly contributes to the medical invisibility of this potent public health problem.

A growing body of scientific evidence associating exposure to specific POPs with several critical health outcomes in humans now exists. Some of these outcomes include:

- cancer;
- neurobehavioral impairment including learning disorders, reduced performance on standard tests, and attention deficits;

- immune system biochemical alterations;
- reproductive deficits;
- a shortened period of lactation in nursing mothers;
- diabetes.^{29, 30}

One of the mechanisms for many of these effects appears to be through disruption of the human endocrine system. In a 1993 study, published by the U.S. National Institute For Environmental Health Sciences in its journal, *Environmental Health Perspectives*, dieldrin, DDT, heptachlor, mirex, toxaphene, dioxin, and PCBs were included in a list of chemicals with reported reproductive and endocrine-disrupting effects.³¹ These chemicals are able to directly or indirectly influence cell development, carbohydrate and lipid metabolism, protein synthesis, reproductive system growth and function, and even ion and water concentration in the body. This mechanism also accounts for the extraordinary sensitivity of humans to these pollutants during fetal growth.

The literature documents three distinct types of human exposure to POPs. High-dose acute exposure typically results from accidents involving electrical capacitors or other PCB-containing equipment, or high-dose food contamination, such as occurred in Japan in 1968 and Taiwan in 1979.^{32, 33} Mid-level chronic exposure is predominantly characteristic of occupational exposure, and, in some cases, close proximity to environmental storage sites or high consumption of a PCB-contaminated dietary source, such as fish or other marine animals. Chronic, low-dose exposure is the characteristic of population-wide exposure to the existing global background levels of industrial POPs such as PCBs, dioxins and furans, and pesticides such as DDT and its metabolites, with variations due to diet, geography, and level of industrial pollution. The adverse health effects of acute exposures have been well-documented in studies of accidental and



Many of these contaminants are POPs pesticides and industrial chemicals, which, while banned or restricted for use in most northern industrialized countries, continue to be used in developing nations.

occupational exposures to humans,^{34, 35, 36} but low-level and population-wide effects are more difficult to study. People are exposed to multiple POPs during their lifetime, and most people today carry detectable background levels of a number of POPs in their bodies.^{37, 38}

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IV. History, Description, and Toxicity of the Twelve POPs

A. Aldrin and Dieldrin

History and Description

Aldrin and dieldrin are common names for two closely related chemicals that have been widely used for controlling soil insects and certain insect vectors of disease. Aldrin, which readily breaks down to dieldrin in living systems, is used to control soil pests (namely termites) on corn and potato crops. Dieldrin is also an insecticide used on fruit, soil and seed, and has been used to control tsetse flies and other vectors of tropical diseases. Because the chemicals are intended for use on insects in soil, aldrin and dieldrin readily bind to sediment and are rarely leached to groundwater. Dieldrin, for example, persists in soils with a half-life of five years. Both may be volatilized from sediment and redistributed by air currents, contaminating areas far from their sources.

Aldrin and dieldrin have been banned in most developed countries. However, aldrin is still used as a termiticide in Malaysia, Thailand, Venezuela, Zimbabwe and other parts of Africa. Where they are still used, the sources of greatest aldrin and dieldrin human exposure come from occupational use and application, consumption of food grown in treated soil, and dermal contact or direct inhalation of the toxins in houses treated for termites. However, aldrin and dieldrin have also been identified in organisms in Arctic waters and in sediments in the Great Lakes basin, suggesting long-range transport from southern agricultural regions.³⁹

Populations around the world are exposed to aldrin and dieldrin through their diet, especially fish, poultry, beef and dairy products. Aldrin bioconcentrates in mollusks and in fish, and high levels of dieldrin have been found

concentrated in fish, sculpins, snails, and lake trout.⁴⁰ Because aldrin is metabolized to dieldrin in the body, dieldrin residues that show up in tissues are likely an additive of aldrin and direct dieldrin exposures. A study performed in Nigeria found that aldrin/dieldrin residue levels in animal products range on average over twice the maximum limit defined by the UN Food and Agriculture Organization.⁴¹

In a study of breast-fed infants in Australia, 88 percent of the offspring were found to exceed the World Health Organization's Average Daily Intake (ADI) allowance.⁴² Dieldrin has been isolated in the amniotic fluid in tissues of developing human fetuses, confirming its capacity for placental transfer.⁴³ The half-life of the residues in humans is approximately nine to twelve months, and the rates of excretion of dieldrin are roughly equal to the average daily intake for most people.⁴⁴

Health Effects of Aldrin and Dieldrin

Aldrin and dieldrin, though highly toxic, are also very species-specific. Consistent with other organochlorine pollutants, most studies on animals reveal liver damage to be the primary effect. These pesticides also cause convulsions, hypersensitivity, tremors, convulsions, neuronal degradation, transient hypothermia and anorexia in wildlife populations.⁴⁵ They produce adverse enzymatic and hormonal changes in fish that lead to impaired reproductive ability.⁴⁶ Ingestion of aldrin-treated rice is believed to be the cause of death of many waterfowl and shorebirds along the Texas Gulf Coast of the United States.⁴⁷

In humans acutely exposed, aldrin and dieldrin show similar adverse effects. Among

In a study of breast-fed infants in Australia, 88 percent of the offspring were found to exceed the World Health Organization's Average Daily Intake (ADI) allowance for Dieldrin .

these are headache, irritability, dizziness, loss of appetite, nausea, muscle twitching, convulsions, loss of consciousness, and possible death.⁴⁸ These conditions may persist for a few weeks following exposure, but have not been shown to be permanent. The lethal dose of aldrin for an adult male is estimated to be about 5 grams. Dieldrin is 40 to 50 times as toxic as DDT.⁴⁹

Carcinogenic Potential — A number of studies have shown that mice chronically exposed to aldrin or dieldrin developed either benign or malignant tumors in the liver.⁵⁰ At the same time similar tests performed on rats revealed no significant effects on the liver. A statistical increase in liver and biliary tract cancers was observed among a group of workers occupationally exposed to aldrin, dieldrin, and endrin, although the study was limited by lack of quantitative exposure information.⁵¹ The U.S. EPA classifies aldrin and dieldrin as probable human carcinogens.

Neurobehavioral Effects — Neurological symptoms have been elicited in animals after acute and intermediate-length exposure, and severe seizures resulted from the ingestion of unknown quantities of dieldrin by two children.⁵² Workers involved in the manufacture or application of aldrin and/or dieldrin have been reported to suffer headaches, dizziness, nausea and vomiting, anorexia, muscle twitching, myoclonic jerking, and in the most severe cases abnormal EEG readings.⁵³ In addition,

two studies of workers with occupational exposure to aldrin and/or dieldrin showed unexpectedly high rates of psychological illness. One of the studies also showed abnormal electromyograms (EMGs), suggesting peripheral neuropathy.⁵⁴ An association has also been documented between dieldrin levels in the brain and Parkinson's disease.⁵⁵

Reproductive Effects — Dieldrin is recognized as having some estrogenic properties in animals, but fetotoxicity has only been observed at levels at which maternal toxicity is apparent.^{56,57} Observations of offspring of aldrin treated female rats revealed an increase in the effective time for testes descent. In mice and hamsters, large, single dosages of aldrin or dieldrin in mid-gestation have resulted in physical deformities in the fetus, including foot webbing, cleft palate, and open-eye. Increased mortality has also been reported.⁵⁸

Other Effects — Extensive renal damage has been reported in rats exposed to high dosages.⁵⁹ Animals chronically exposed to aldrin and/or dieldrin exhibit hepatic effects characteristic of halogenated hydrocarbon pesticides, including increased liver weight and/or size, hepatocyte enlargement, increase in cellular smooth endoplasmic reticulum and microsomal enzyme levels and activity, and an increase in vacuolization.⁶⁰

B. Endrin

History and Description

Endrin is a rodenticide used to control mice and voles, and an insecticide used on cotton, rice, and maize. Closely related to aldrin and dieldrin, endrin is the most toxic of the three, and its metabolites are more toxic than endrin itself. In soils, endrin is extremely persistent, with a half-life as long as twelve years. In cases of extreme poisoning endrin residues have been found in adipose tissues, but the majority of endrin is metabolized within 24 hours and removed from the body by waste products.⁶¹ Endrin has been detected in rainwater from Cree Lake in northern Saskatchewan, and has been reported in a freshwater lake in the Canadian Arctic.⁶² It has been found to bioaccumulate in species from algae, pouch snail, flathead minnow, rainbow trout, Virginia oyster, and sheepshead minnow.⁶³

Health Effects of Endrin

In animals, endrin has a very high acute toxicity. Mice, rats and dogs administered endrin



developed hepatic abnormalities, including diffuse degeneration and cell vacuolization.⁶⁴ It has enlarged kidneys in dogs and livers in rats.⁶⁵ Fetuses from hamsters and mice treated with endrin demonstrated developmental abnormalities.⁶⁶ Studies performed on rats found that endrin increased reactive oxygen species in liver and brain tissue.⁶⁷

Neurobehavioral Effects — The nervous system is the chief target of acute endrin poisoning. During occupational exposure, twitching muscles, mental confusion, and seizures have occurred.⁶⁸ Approximately 1,500 people were exposed to endrin-contaminated flour in Qatar and Saudi Arabia, resulting in 26 deaths.⁶⁹ Endrin may also contribute to improper bone formation, although no human data exist for this effect.⁷⁰

Reproductive Effects — Increased mortality of offspring in the second and third generations of rats dosed with endrin may suggest that the reproductive system is targeted by the pesticide.⁷¹ Studies on the effects of endrin on pregnant mice have shown that the toxin produces oxidative stress in fetal and placental tissues.⁷²

C. Chlordane

History and Description

Chlordane is an insecticide used in fire ant control, on lawns, and on a variety of crops. It continues to be used in a number of countries. Chlordane is very persistent in the environment, surviving in soils for more than 20 years.⁷³ It can be transported in the atmosphere for long distances and is now present in the Arctic food web.⁷⁴ Chlordane has been measured in freshwater and marine biota including flathead minnow, algae, snail, and the sheephead minnow.⁷⁵

When U.S. production of chlordane was suspended in 1976, it was in response to data from human monitoring studies showing that 90 percent of all Americans had residues of chlo-

rdane metabolites in their tissue, and that these metabolites may be transferred from mother to the fetus across the placenta and from mother to child via mother's milk. The chief route of transfer into the human body was found to be via the diet, through meat and dairy foods.⁷⁶ In 1997, the world's last producer of chlordane, the U.S.-based Velsicol Chemical Corporation, announced that it would permanently cease production.⁷⁷

Women in the Arctic were found to consume greater than the WHO average daily intake level of chlordane in 50 percent of the cases.⁷⁸ Inuit mothers' milk contains 10 times higher levels of chlordane than seen in southern Canadian residents.⁷⁹ Likewise, in Australia, levels of chlordane intake for infants via breast milk exceeded the WHO ADI standards in 48 percent of the cases surveyed.⁸⁰

Health Effects of Chlordane

Inhalation of chlordane has produced liver lesions in rats and hepatomegaly in monkeys.⁸¹ Rats exposed to airborne chlordane have also experienced abnormal respiratory movements, convulsions, and thyroid abnormalities.⁸² Other animal studies have demonstrated damage to the liver and the central nervous system.⁸³

A dose-dependent relationship seems to exist between bronchitis, sinusitis, and migraines and chlordane in the air of houses treated for termites. A study of 261 people exposed to varying levels in indoor air revealed a dose-related increase in respiratory illnesses.⁸⁴ In a study of workers in occupations associated with chlordane manufacture, people experienced cerebrovascular disease at rates twice that expected.⁸⁵

Carcinogenic potential — Chlordane is thought to be a cancer and tumor promoter. It causes benign and malignant liver tumors in mice and rats. The U.S. EPA has classified chlordane as a probable carcinogen,⁸⁶ while the International Agency for Research on Cancer (IARC) classifies it as a possible human carcinogen.

Chlordane is very persistent in the environment, surviving in soils for more than 20 years.

Neurological Effects — A study of individuals living in an apartment complex sprayed for termites showed abnormal balance, choice reaction time, verbal recall, cognitive function, and slowing of motor speed in 216 adults.⁸⁷

Reproductive Effects — Chlordane may mimic sex steroids or change their levels in exposed individuals. A study of the effect of chronic low-level chlordane exposure of male and female rats revealed altered hormone function at levels of 100 and 500 ng/g dosed groups.⁸⁸

D. DDT

History and Description

1,1,1-trichloro-2,2-bis-(p-chlorophenyl) ethane (DDT) is an organochlorine-based pesticide that has been used as an insecticide in agriculture and to combat insect vectors of diseases such as malaria and typhus.

DDT is one of the earliest and most well known pesticides, and one of the most widely used. This has led to equally widespread contamination of water and soil resources and resultant serious health effects in humans and animals. Although banned

in many countries, DDT continues to be used for residual indoor spraying in a significant number of countries and for agriculture in some areas as well.

Because of its effectiveness at killing insects with few acute effects on people, DDT has been a mainstay of many countries' fights against malaria, a disease that is a growing threat to health in much of the world. For this reason, the World Health Organization (WHO), while supporting an ultimate phase-out, continues to endorse the limited use of DDT in government-authorized public health campaigns and for indoor residual application.⁸⁹ A WHO action plan to balance these two priorities is currently under development.

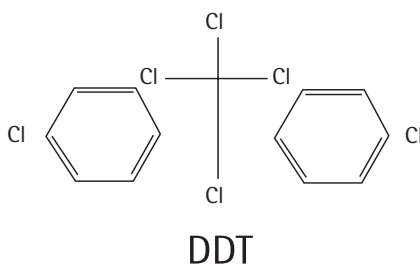
Long-range atmospheric transport of DDT into the northern countries, including the Arc-

tic, is well documented. DDT has been detected in Arctic air, soil, snow and ice, and virtually all levels of the Arctic food chain.⁹¹ Many studies indicate that bottom sediments in lakes and rivers act as reservoirs for DDT and its metabolites.⁹² Despite a twenty-year ban in the U.S., it is still found concentrated in soils and freshwater sediments. Aquatic vertebrates such as fathead minnow and rainbow trout have also been found to contain DDT.⁹³

For most populations, the primary route of exposure to DDT and its metabolites is through food.⁹⁴ DDT is readily metabolized into a stable and equally toxic compound, DDE.⁹⁰ DDT and DDE are fat-soluble and store well in the adipose tissues of humans and animals. They break down in the body very slowly, and are released primarily through urine and breast milk. DDT and its metabolites have been found in virtually every breast milk sample tested, including samples taken in tropical areas of Mexico.⁹⁵ In many countries where the pesticide is still in use, levels exceed FAO/WHO ADI standards; concentrations of DDE are four-to five-fold higher in mothers' milk from Inuit in northern Quebec than populations from southern Canada.⁹⁶

Health Effects of DDT

Adverse health effects of DDT in animals include reproductive and developmental failure, possible immune system effects, and the widespread deaths of wild birds after DDT spraying. As is the case with many organochlorine insecticides, a major target of acute DDT exposure is the nervous system. Long-term administration of DDT has brought about neurological, hepatic, renal, and immunologic effects in animals. Liver damage has been a main effect observed in rats and dogs.⁹⁷ In rats, DDT exposure leads to tremors, decreased thyroid function,⁹⁸ and impaired neurological development.⁹⁹ In a study that chronically exposed rats to DDT through food, immunosuppression was observed in the form of reduced mast



Chemical Structure of DDT

cell population and inhibited anaphylactic shock reaction.¹⁰⁰

The U.S. Occupational Safety and Health Administration has set a permissible exposure limit for adult workers of 1 milligram of DDT per cubic meter of air over an eight hour period.¹⁰¹ Human studies have shown that a single dose of 6 to 10 milligrams of DDT produces excessive sweating, headache and nausea in adults.¹⁰²

Carcinogenic Potential — Based on observation of tumors (generally of the liver) in mice and rats, the U.S. EPA classifies DDT as a probable human carcinogen.¹⁰³ DDT is structurally similar to other probable carcinogens, including its metabolite DDE. Conflicting studies have appeared in the last several years as to a positive correlation between DDT/DDE and breast cancer and the issue is subject to continuing research.^{104,105}

Reproductive Effects — A rise in reproductive abnormalities in both humans and wildlife over the last 20-40 years has caused some scientists to look for environmental factors that may be influencing reproductive capacity. DDT and its metabolites are considered to be environmental estrogens,¹⁰⁶ with estrogenic effects reported in animals.¹⁰⁷ Research has shown that DDT prevents androgen from binding to its receptor thereby blocking androgen from guiding normal sexual development in male rats and resulting in abnormalities.¹⁰⁸ DDT has been shown to have anti-androgenic effects on the sexual development of the fetus and breast-feeding infant.¹⁰⁹ Evidence of the estrogenic properties of DDT and DDE have also been observed in alligators in which hatchlings from DDE-painted eggs are sexually indeterminate; possessing both male and female reproductive characteristics.¹¹⁰ Chronic ingestion of DDT-containing food by adult mallard ducks resulted in eggshells of offspring that were significantly thinner and lighter than those found

in control animals.¹¹¹ Exposure to DDE and the resultant physical feminization of male Florida panthers may be partially responsible for the drastic plunge in the reproduction rate of this species in recent years.¹¹²

In a study in India, a group of men who worked with DDT was found to have decreased fertility, and a significant increase in still births, neonatal deaths and congenital defects among their children. Israeli men with unexplained fertility problems were also found to have high blood levels of DDT.¹¹³

Neurodevelopmental Effects - Experimental studies involving controlled doses of DDT were performed on humans in the 1940s and 1950s. They found that DDT induced nausea, headaches, diarrhea, irritation of the mucous membranes, tremors and convulsions, malaise, moist skin, and hypersensitivity to contact.¹¹⁴ In a study detailing long-term occupational exposure, twenty workers exposed for fourteen years were found to have blood serum levels of DDT that were ten times those of the general population, and showed observable nervous system abnormalities.¹¹⁵

The maternal body burden of DDT and its metabolites is stored at the highest concentrations in breast milk and the placenta, creating a hazard for the developmentally vulnerable offspring. Low doses fed to neonatal mice were associated with a permanent hyperactive condition, as well as tremors and paralysis.¹¹⁶ Fertilized eggs of killifish exposed to DDT in water suffered a delayed rate of physiological development.¹¹⁷

E. Heptachlor

History and Description

Heptachlor is a termiticide and an insecticide used on seed grain and crops. It has also been used extensively for fire ant control, and is present as an impurity in the pesticide chlordane. Heptachlor is metabolized in animals to heptachlor epoxide. The use of heptachlor has

A rise in reproductive abnormalities in both humans and wildlife over the last 20-40 years has caused some scientists to look for environmental factors that may be influencing reproductive capacity.

been banned in Cyprus, Ecuador, the European Union, Portugal, Singapore, Sweden, Switzerland and Turkey. Its use is severely restricted in Argentina, Israel, Austria, Canada, Denmark, Finland, Japan, New Zealand, the Philippines, the U.S., and some countries of the former Soviet Union.¹¹⁸ In recent years, however, heptachlor (and the closely related chlordane) has been used for major road building projects in Africa, for protection of residential structures in Northeastern Australia and Asia, and for crop protection in South America.¹¹⁹ The world's last producer of heptachlor, the U.S.-based Velsicol Chemical Corporation, announced in 1997 that it would permanently cease production.

Heptachlor is subject to long-range transport as indicated by its presence in precipitation samples from Lake Erie.¹²⁰ It has been found in mosquito, fish, soft clam, oyster, and fathead minnow.¹²¹

Human exposure to heptachlor is mainly through ingestion of food with residues of the compound and through inhalation in homes sprayed with heptachlor as an insecticide.¹²² In a 1996 study of breast-fed infants in Australia, 100 percent of the infants sampled were found to exceed the WHO ADI for heptachlor.

Health Effects of Heptachlor

Heptachlor is highly toxic in humans, and causes hyperexcitation of the central nervous system and liver damage. Retrospective studies on people employed as heptachlor sprayers have shown significant increases in death from cerebrovascular disease. Heptachlor has been found to have significant effects on progesterone and estrogen levels in laboratory rats.¹²³ Other animal studies show nervous system disruption and liver damage.¹²⁴

Laboratory animals poisoned with heptachlor exhibited lethargy, convulsions, incoordination, tremors, stomach cramps, pain and coma.¹²⁵ Because heptachlor is stored for extended periods in fatty tissue, intense activity

can mobilize the compound and lead to a re-appearance of toxic symptoms long after exposure has ceased.¹²⁶

Carcinogenic Potential — The U.S. EPA has classified heptachlor as a probable carcinogen.¹²⁷ It has been found to inhibit breast epithelial cell communication; and at high concentration it is a possible breast tumor promoter.¹²⁸ In rats, chronic exposure to heptachlor lead to an increased incidence of liver carcinomas.¹²⁹

F. Hexachlorobenzene

History and Description

Hexachlorobenzene (HCB) has been used as both a pesticide and an industrial chemical in recent years. While intentional production has declined, HCB is also still produced as a byproduct during the manufacture of several chlorinated chemicals, and has been detected in the flue gas and the fly ash of municipal incinerators.

Long-range atmospheric transport of HCB to the Arctic and other remote areas is a well-recognized phenomenon. The substance has been detected in Arctic air, snow, seawater, vegetation and biota.¹³⁰ It has also been observed in other remote areas such as the North Pacific Ocean and in the rainfall of two remote islands on Lake Superior.¹³¹ HCB has been measured in freshwater and marine biota, including grass shrimp, sheephead minnows, and pinfish. Concentrations of HCB have been observed in fish-eating birds and predatory bird species. It has also been detected in the eggs of the peregrine falcon.¹³²

HCB also accumulates in human body tissues and breast milk. In 1986, HCB was found in 98 out of 100 human adipose samples from people throughout the U.S.¹³³ Breast-fed Australian infants were found to have HCB dietary intakes exceeding the WHO ADI standards in 27 percent of the cases.¹³⁴ Levels of HCB in Inuit mothers' milk are five to nine times higher than levels seen in southern Canadian mothers' milk.

Health Effects of HCB

Acute high dose exposure to HCB is associated with porphyria cutanea tarda due to its liver toxicity.¹³⁵ In Turkey, people exposed to HCB-contaminated flour developed this condition, and although most recovered after exposure ceased, some continued to experience porphyria through several years of follow-up.¹³⁶

HCB is also associated with enlarged thyroid glands, scarring, and arthritis exhibited in offspring of accidentally exposed women.¹³⁷ Children born to mothers known to have ingested HCB-tainted food during pregnancy experienced acute illnesses and rashes. These children were additionally exposed through breast milk.¹³⁸ Follow up studies reported porphyria cutanea tarda, reduced growth, and arthritic symptoms in children directly exposed to contaminated bread or mothers' milk. There was also a 37 percent prevalence of enlarged thyroids.¹³⁹ Finally, HCB has been shown to alter a white blood cell function following occupational exposure, although the clinical meaning of this finding is not clear.¹⁴⁰

Carcinogenic Potential — HCB has shown to be carcinogenic in rodents,¹⁴¹ and the U.S. EPA classifies it as a known animal carcinogen. HCB has also been classified by the EPA and the International Agency for Research on Cancer(IARC) as a probable human carcinogen.^{142,143}

Neurodevelopmental and Other Effects - In animals, HCB demonstrates acute neurologic toxicity. Symptoms include tremors, paralysis, incoordination, weakness, and convulsions.¹⁴⁴ Maternal HCB exposure has led to newborn death in rats. Mortality was due to lung damage and related to cumulative exposure through milk.¹⁴⁵ Also in rats, maternal exposure leads to teratogenic effects including cleft palate, changes in rib development, kidney malformations, and decreased body weight. In dogs, HCB has been shown to promote changes in the liver and central nervous systems, while in rats it has caused damage to the

liver and spleen.¹⁴⁶ HCB has been shown to alter steroid production of adrenal cortex cells following low doses in rats.¹⁴⁷

G. Mirex

History and Description

Mirex is a bait insecticide used against a number of insect pests. It has been used heavily in South America and South Africa. Secondary use of mirex as a fire retardant in plastics, paints, and electrical goods is currently heavily restricted or banned in most countries. Mirex is highly resistant to biodegradation and has a half-life of up to ten years in sediment. In the presence of sunlight, mirex breaks down to a far more potent toxin, photomirex.¹⁴⁸ Mirex is known to be one of the most stable and persistent pesticides. Mirex has been detected in Arctic freshwater and terrestrial organisms and in core sediment samples in Lake Ontario. It has also been found in lake trout captured in Lake Ontario, and in fathead minnows and beluga whale oil from the St. Lawrence River.¹⁴⁹

Mirex levels in human milk are above average for communities consuming high amounts of fish and sea bird eggs.¹⁵⁰ Levels in the milk of Inuit from Nunavik, northern Quebec, are 10 times higher than those in southern Canadian residents. Even higher concentrations of Mirex are seen in omental fat tissue from Greenland Inuit.¹⁵¹

Health Effects of Mirex

There have been few studies on human exposures, and little data exists for human health effects of mirex. Animal studies have shown several adverse reactions to mirex doses administered through diet. In rats, mirex exhibits toxic effects on fetuses, including cataract formation, and it causes liver hypertrophy following long-term, low-dose



exposure in rats.¹⁵² Mirex is also associated with suppression of the immune system.¹⁵³ In addition to the severe effects of mirex on test animals, a reduction in germination and emergence of several plant species has been observed, indicating that mirex is highly toxic to a wide variety of systems.¹⁵⁴

Carcinogenic Potential — Due to evidence of its carcinogenicity in animals, the International Agency for Research on Cancer has classified mirex as a possible human carcinogen. Long term administration of 50 and 100 ppm of mirex in the diets of male and female rats was associated with liver lesions and hepatocellular carcinoma.¹⁵⁵ A two year study by the National Institute of Health revealed clear evidence of carcinogenic activity for male and female rats indicated by increased incidences of a variety of abnormal cellular conditions.¹⁵⁶ Structural changes, cell aberrations and cell death were apparent in the livers of chickens fed mirex at 10 ppm and above for 12-16 weeks. Liver enlargement was observed in rats fed mirex at 1-100 ppm for 2-4 weeks.¹⁵⁷ Mirex administered to fish results in kidney lesions and gill damage.¹⁵⁸

Reproductive Effects — Mirex has been identified as an endocrine disrupter. Mirex administered in doses of 0.4-50 mg in female rats inhibited PMS-induced ovulation by affecting mechanisms controlling the release of hormones essential to ovulation.¹⁵⁹ Female rats fed 25 ppm mirex prior to mating and through gestation and lactation had significantly smaller litters, decreased survival of pups, and a 33% incidence of cataracts in pups compared to 0% in controls. Pregnant female rats fed 0-25 ppm mirex experienced significant increase in fetal mortality, decreased pup survival to 8 days and increased incidence of cataracts and other lens changes.¹⁶⁰ Pregnant female rats administered 6 mg/kg-day of mirex on days 7-16 of gestation had by day 21 fetuses with external abnormalities including edema and ectopic gonads.¹⁶¹ Other external abnormali-

ties of fetuses of mirex-fed mothers include scolioses, runts, short tail at 6 mg/kg-day, cleft palate and heart defects at 12 mg/kg-day.¹⁶²

H. Toxaphene

History and Description

Toxaphene is an insecticide and ascaricide, especially against maggots and on cotton. While the production of toxaphene was effectively halted 15 to 20 years ago, the use of small quantities is still permitted. It is persistent and transported through the air. The half-life of toxaphene in the soil may be as long as twelve years, and it is known to bioconcentrate in organisms.

Toxaphene has been detected in Arctic air, sea water, vegetation, and biota.¹⁶³ Aquatic mammals lack hepatic enzymes that would help metabolize toxaphene congeners. As a result, toxaphene can accumulate in very high levels in their adipose tissues. Concentrations of toxaphene have been found in algae, snail, fathead minnow, brook trout, rainbow trout, Virginia oyster, and Atlantic salmon.¹⁶⁴ Blubber samples from Beluga whales inhabiting the north coast of Alaska have been found to contain toxaphene at unhealthy concentrations, even exceeding concentrations of DDT and PCBs in the whales.¹⁶⁵ People are most often exposed to toxaphene through their diet, especially if it includes fish from contaminated sources. Toxaphene has been measured in oils and fats, root vegetables, meats and grains. The insecticide was reported to be one of the most frequently occurring residues in total dietary foods between the years 1982-1984, exceeding levels of DDT in the same samples.¹⁶⁶

In the Canadian Arctic, toxaphene was found in indigenous people as a result of their heavy reliance on fish and game for food, their position at the highest trophic level in the Arctic food chain, and the high lipid content of their diet.¹⁶⁷ A study of women living in the Arctic and eating a traditional diet primarily of fish and blubber from marine mammals showed

that they consumed twenty times the tolerable daily intake of toxaphene.^{168,169} Fifty percent of one indigenous population exceeded the WHO prescribed ADI for toxaphene.¹⁷⁰

Health Effects of Toxaphene

Studies of the effects of toxaphene on rats lasting thirteen weeks revealed altered enzyme activities in the liver, an increased liver/body weight ratio, and dose dependant changes in the kidney, thyroid and liver. Short-term exposure has led to hepatomegaly, inhibition of hepatobiliary function, and induction of microsomal enzymes.¹⁷¹ Chronic exposure to toxaphene has caused disruptive and often toxic effects on the hepatic, renal, and immunological and neurological systems in animals. In addition, toxaphene causes immuno-suppressive and behavioral developmental abnormalities. In some species, intermediate duration oral exposure to toxaphene has lead to degenerative changes in the liver, including cytoplasmic vacuolization, cell hypertrophy, and necrosis.¹⁷²

Acute poisoning from ingestion or prolonged inhalation of toxaphene has been reported to cause damage to the lungs, nervous system, liver and kidneys in humans.^{173,174} In at least six cases, ingestion of high doses of toxaphene has been fatal. Inhaled toxaphene has been reported to cause reversible respiratory failure.¹⁷⁵ The literature is sparse detailing the chronic low level effects of toxaphene exposure to humans, although inference from animal studies suggests that humans are at risk for adverse health effects from limited daily exposures

Carcinogenic Potential — Toxaphene has been associated with cancer in mammals, although few studies have been done on human exposures. Mice were treated with 0, 7, 20, and 50 parts per million toxaphene through their diet for eighteen months and observed at six months post-treatment. An increased incidence of hepatocellular carcinoma was observed in both sexes.¹⁷⁶

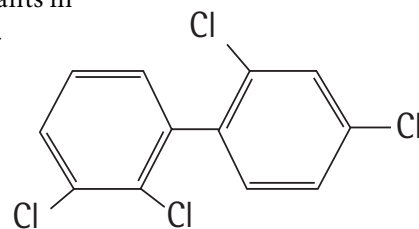
In a third study, the National Cancer Institute treated both rats and mice with toxaphene over an eighty week period and observed the animals for 30 days post-treatment. Both male and female rats displayed statistically significant dose-related increases of thyroid tumors and mice showed statistically significant increases in liver cancer.¹⁷⁷ Toxaphene is known to be genotoxic in mammalian cell systems and a cell replication inhibitor. The International Agency for Research on Cancer has classified toxaphene as a possible human carcinogen.

Reproductive Effects - Toxaphene has been reported to display some estrogenic effects, and has also been observed to be mildly anti-estrogenic in some analyses.^{178,179} It has exhibited estrogenic activity in exposed alligators.¹⁸⁰ Trout exposed to toxaphene for 90 days at 0.039 micrograms/L, the lowest concentration tested, experienced a 46% reduction in weight and females had significantly reduced egg viability.¹⁸¹ At long-term exposure levels of 0.5 micrograms/L, egg viability was reduced to zero. A similar effect has been observed in female ring-necked pheasants, who suffered reductions in egg laying and hatch ability at toxaphene levels of 300 mg/kg in their diet.¹⁸²

I. PCBs

History and Description

Polychlorinated biphenyls, or PCBs, are a group of highly toxic chlorinated industrial chemicals used as coolants and lubricants in electrical transformers and other electrical equipment, weatherproofers, dielectrics, and to prolong residual activity of pesticides. PCBs are usually released to the environment in the form of an impure mixture in which other chemicals are also present.¹⁸³ PCBs are fire-resistant, have a low volatility, and are relatively stable and persistent, making them well-suited for industrial use but also problematic in the environment.



A polychlorinated biphenyl (PCB)

Chemical Structure of PCBs

PCBs had been in use for more than 25 years when, in the late 1960s, attention focused on PCB poisoning of birds and people.¹⁸⁴ By the late 1970's, evidence of the extreme persistence and adverse health effects of PCBs had resulted in bans on their manufacture in most industrialized countries. Although they are no longer manufactured or imported into the U.S. and Canada, there remain sizable quantities in storage in both countries. In addition, PCB fluids are still permitted in closed electrical and hydraulic systems and are present in many older transformers, fluorescent lighting fixtures, and other electrical devices and appliances. These are vulnerable to release into the



environment, as older components can leak. Other sources of PCB contamination come from improper disposal or incineration of PCBs and PCB-contaminated hazardous waste sites.^{185,186}

Once PCBs are released into the environment, they may be carried by wind long distances before they settle in soil or water. They readily absorb into particulate matter and become incorporated into suspended particulates and bottom sediments when released into aquatic systems.¹⁸⁷ There is evidence of PCBs being transported thousands of kilometers in the atmosphere;¹⁸⁸ some have even been found in snow and seawater in the Antarctic.

In general, PCBs are not soluble in water and tend to adhere to organic particles in any material or solution in which they are deposited. They have been found in the Arctic regions in green algae, fungus, oysters, grass shrimp, Atlantic salmon, and mink.¹⁸⁹ Dolphins in supposedly uncontaminated waters have been known to have PCB levels up to 833 ppm - a level that requires special precautions reserved for materials that are considered to be toxic waste.¹⁹⁰

Human tissues are especially to sequester PCBs and resist breakdown and release of the contaminant through normal physiological pathways. In 1985-87, PCBs were measured in the blood of Inuits from the community of Broughton Island in Canada. Results showed that blood PCBs exceeded tolerable levels, set by Health Canada, in 63 percent of the females and males under 15 years of age, in 39 percent of females aged 15-44, in 6 percent of males 15 and older, and in 29 percent of women 45 and older.¹⁹¹ PCBs tend to accumulate in high fat regions of the body, such as breast milk and adipose tissue. A study in the late 1980s showed that PCB levels in the milk of Inuit women from the east coast of Hudson Bay in northern Quebec were approximately five times higher than in women of southern Canada.¹⁹² The U.S. Food and Drug Administration requires that dairy, poultry, seafood and infant products not contain PCBs in more than 0.2-3 ppm. Nonetheless, average human exposure may exceed the EPA and FDA's regulatory guidelines in many foods.^{193, 194, 195}

Health Effects of PCBs

PCBs have a long and documented history of adverse effects in wildlife.¹⁹⁶ They have been associated with poor reproductive success and impaired immune function of captive harbor seals in the Arctic.¹⁹⁷ After a major flood in the Saginaw River basin in Michigan in 1986 allowed PCB contaminants to spread through the ecosystem, the following year's hatch rate of Caspian terns in the area dropped by more than 70 percent. Hatching chicks showed developmental deformities, and none survived more than five days. Hatch ability of this Caspian tern colony did not show recovery after three more breeding seasons.¹⁹⁸

Acute effects of PCB exposure in humans were documented following ingestion of contaminated rice oil in Japan in 1968 and Taiwan in 1979¹⁹⁹. Long term studies of the more than 2,000 people who were exposed during these events revealed increased mortality due to PCB

intake.²⁰⁰ A positive association was established between PCB dosing and acute liver damage, with liver disease being the cause of death in a significant number of exposed people.²⁰¹ Acute exposure to PCBs has also caused chloracne, a chemically induced acneform eruption.²⁰²

Carcinogenic Potential — Based on data from animals, including findings that some liver cancers in rodents are directly linked to PCBs as tumor promoters^{203, 204} the U.S. EPA classifies PCBs as a probable human carcinogen. The literature on PCBs and cancer in humans has been mixed, including studies which have found possible associations between breast cancer cells and the estrogenic properties of PCBs, studies reporting that PCBs inhibit crucial processes in cellular communication that normally prevents carcinogenic precursors in breast epithelial cells, studies which have found associations between PCB exposure and cancer in occupational settings²⁰⁵, and studies which have claimed no association between PCBs and cancer.^{206, 207} Some of this inconsistency is unexplained, some has been attributed to study design, some to difficulties and differences in measuring exposure. Carcinogenesis is a complex multistage process, however, and different PCB congeners may have different health effects citation. It is not surprising, therefore, that unanimous agreement among scientists regarding PCBs and cancer has been difficult to achieve.

Neurodevelopmental Effects — Human fetal exposures of PCBs are associated with neural and developmental changes,²⁰⁸ lower psychomotor scores,²⁰⁹ short-term memory and spatial learning effects,²¹⁰ and long-term effects on intellectual function.²¹¹ Neurological dysfunction has been associated with perinatal PCB exposure in several Dutch studies.^{212, 213, 214}

In the U.S. a study of 313 children of women who ate two or more fish from the PCB-contaminated Great Lakes a month prior to pregnancy revealed behavioral disorders in the in-

fant's reflexes at birth compared with controls.²¹⁵ Follow-up studies on the same children at four years of age found that they continued to suffer from poor short term memory and attention problems.²¹⁶ IQ and achievement tests administered to the same sample at eleven years of age indicated that the most highly exposed children had poorer verbal comprehension, shorter attention span, and were three times as likely to have lower IQ than controls.²¹⁷ These findings are corroborated by similar observations in Taiwanese children whose mothers were exposed to PCBs through contaminated rice oil.²¹⁸ Other observations in Japan included shorter gestation periods, lower birth weights, and deficits in post-natal growth.

A particularly significant result of the study of the Great Lakes children is that although a greater amount of PCBs are transferred to the infant during lactation, no correlation was seen with this exposure. The fetus appears highly vulnerable to PCB toxicity at levels that appear to have less effect on infants and no detectable clinical effects in adults.

Non-human species including rats, mice, monkeys and quail have also shown clear neural changes resulting from PCB exposure. Rhesus monkeys display impaired or abnormal neuromotor function at PCB doses as low as 0.5 parts per million given three times daily over a twelve month period.²¹⁹

Reproductive Effects — PCB-induced reproductive impairment has been documented in seals and porpoises, and other reproductive abnormalities such as embryo toxicity have been seen in mink. The hormone-like effect of PCBs can also cause egg shell thinning in birds by interfering with calcium accumulation.²²⁰

The hormone-disrupting effects of PCBs are profoundly evident in a study of turtle sex-determination.²²¹ Sex of the red-eared slider turtle is determined largely by the temperature at which the egg is incubated; males re-

sult from cooler temperatures, females warmer. In experiments in which eggs were painted with PCBs during the period of sexual differentiation, the temperatures that normally produced male offspring were counteracted by the estrogenic properties of PCBs, resulting in female offspring. The PCB levels that altered turtle sex in the study are comparable to average human levels of PCBs in human breast milk in industrialized nations.

Immune Effects – Perinatal exposure to PCBs may impair immune responses to infection as suggested by a 20-fold higher incidence of infectious diseases and ear infections in a study of Inuit infants with high PCB exposure compared to individuals in a lesser-exposed population.²²²

J. Dibenzo-Dioxins and Furans

History and Description

Polychlorinated dibenzo-para-dioxins (dioxins) and polychlorinated dibenzo-furans (furans) are two structurally similar families of compounds that include 75 and 135 congeners, respectively. At least twenty are considered highly toxic. The overall toxicity of a dioxin containing mixture is assumed to be the Toxic Equivalent (TEQ) of a stated amount of pure 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), the most potent, hazardous and well-studied dioxin.²²³ Dioxins and furans have similar effects on human health, and will be referred to collectively as dioxins.

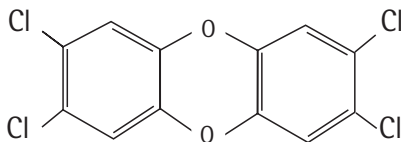
Dioxins are not commercially produced, but are by-products of combustion and industrial processes, including the manufacture of chlorinated chemicals, the incineration of hospital, hazardous, and municipal waste, and the bleaching of paper products.²²⁴ Dioxins are stable, persistent compounds that are believed to have a half-life of seven to twelve years in the human body.²²⁵

There is substantial evidence to indicate that populations of wildlife species high on the food chain are suffering health damage due to reproductive and developmental impairment due to background exposures to dioxins and related compounds. In the Great Lakes, exposure to dioxin-like compounds has been linked to large-scale hormonal, reproductive, and developmental impairment among numerous species of predator birds, fish and wildlife; these impacts are primarily transgenerational, affecting the offspring of the exposed organisms.

Approximately 90% of human exposure to dioxin comes from food, specifically in the form of beef, fish, and dairy products. Contamination in the food supply comes from dioxin particles that are deposited in water or soil and then proceed up the food chain through fish and livestock, ultimately reaching human tissues through the food we eat. Dioxin bioaccumulates, becoming increasingly concentrated in living tissues as it moves up the food chain.²²⁶

Dioxins are known to be toxic at extremely low doses. Although on average, Americans are exposed to only 1 to 3 picograms per kilogram of body weight per day (one picogram being one billionth of a gram), this level is comparable to doses used in laboratory studies resulting in adverse health effects in animals.²²⁷ Because mother's milk is often highly contaminated, infants receive higher exposures. An average nursing infant receives 60 pg/kg/day of dioxin, not including dioxin-like PCBs. This is 10-20 times more than the average adult, and, in the first year of life, 4-12 percent of his or her entire lifetime exposure.

Daily exposure results in an accumulation of dioxins known as a body burden in oil-soluble media such as lipids, breast milk, and blood.²²⁸ A 1982 EPA study of dioxins in body fat from a representative sample of the U.S. population revealed an average body burden



A polychlorinated dibenzodioxin,
TCDD (2, 3, 7, 8 tetrachlorodibenzo-p-dioxin)
Chemical Structure of TCDD

of 7,000 to 9,000 pg/kg of body weight (7-9 ppt).²²⁹ In most industrialized nations of the world, dioxin body burdens and exposures are in the same range, with levels assumed to be somewhat lower in developing nations, where little testing has been done. The U.S. EPA has found it difficult to define a safe dose of dioxin. The World Health Organization, however, recently lowered by more than half its tolerable daily intake. Fixed previously in 1990 at 10 picograms per kilogram of body weight for TCDD, the standard was reduced to 4 picograms based on a recognition that subtle effects may already occur in the general population in developed countries at levels of two to 6 picograms.²³⁰

Data on trends in dioxin contamination of human tissues are sparse, though one study found that levels might have decreased slightly in the 1980s following consistent increases during the preceding decades.²³¹

Health Effects of Dioxins

Chronic low-dose dioxin exposure can produce long-term health effects that permanently alter animal systems.²³² Dioxins and furans have shown developmental and immuno-toxicity in animals, especially rodents.²³³ They have caused the alteration of estrogen, progesterone, testosterone, and thyroid hormone levels in several species, and have inhibited the action of estrogens in several species. They cause reductions in fertility, litter size, and uterine weights in mice, rats, and primates.²³⁴

In humans, there is evidence that high-level exposure to dioxins and furans can cause variations in serum lipid levels, microsomal enzyme induction, and gastrointestinal alterations.²³⁵ Other studies of high-level occupational exposure have found associations with some types of cancer, and have concluded that *in utero* and lactational exposures to dioxins and furans are capable of affecting the hypothalamic/pituitary/thyroid regulatory system in human infants.²³⁶ According to the U.S.

EPA, effects on humans, including hormonal and metabolic changes, have been documented at dioxin body burdens and exposures only slightly higher than those of the general population.²³⁷

A single cellular mechanism is thought to be responsible for the wide range of effects dioxin can have. It is believed that dioxins affect organisms by binding to pre-existing cellular receptors designed for hormones, entering the nucleus and then manipulating the on or off function of the gene. The genes affected by an imposter like dioxin contain codes for proteins, hormones, enzymes and growth factors, which collectively influence tissue development in the human body. This mechanism is the same in both humans and animals, allowing extrapolation from laboratory experiments involving dioxin effects on animals to a parallel human reaction.²³⁸

Carcinogenic Potential — Through the dysregulation of genes, dioxins can directly affect the growth and differentiation of cancer-causing cells.²³⁹ Animal studies demonstrated that every non-human species chronically exposed to 2,3,7,8-TCDD exhibited clear carcinogenic responses, some at doses as low as 1 part per trillion.²⁴⁰ In 1985, the U.S. EPA declared TCDD the most potent synthetic carcinogen yet tested. More recently, the International Agency for Research on Cancer has classified TCDD as a known human carcinogen, and it is probable that all dioxins are human carcinogens.^{241,242} The EPA estimates that current U.S. background dioxin exposures may result in upper-bound population cancer risk estimates in the range of one in ten thousand to one in a thousand attributable to exposure to dioxin and related compounds.²⁴³

Developmental Effects — Dioxins target many cells in the developmental stages of growth when differentiation and proliferation are occurring.²⁴⁴ This action has especially severe consequences for developing fetuses whose

There is substantial evidence to indicate that populations of wildlife species high on the food chain are suffering health damage due to reproductive and developmental impairment due to background exposures to dioxins and related compounds.

mothers are chronically exposed to low-levels of dioxin such as exist in most food in the human diet. Because dioxins store well in breast milk and the placenta, fetuses and newborns are exposed to a heavy concentration of the toxins during a highly susceptible period.²⁴⁵

Infant exposure can reach dioxin levels that are fifty times higher than those an average adult might experience daily. A 1994 study of 200 babies born to women in Holland with high levels of dioxin in their breast milk found high levels in the infants' umbilical cords, and a variety of dysfunctions in the babies' muscles, reflexes and thyroids.²⁴⁶

Prenatal mortality has been observed in rats and monkeys exposed to 2,3,7,8-TCDD during gestation at levels that produced either minimal toxicity or no effect in mothers.^{247,248}

Rhesus monkeys chronically exposed to 2,3,7,8-TCDD in their diet from pregnancy through lactation suffered a decrease in survival of offspring and significant behavioral modifications in offspring that lived.²⁴⁹ Cognitive deficits as well as impaired learning performance and alterations in peer-group behavior were also observed in offspring.²⁵⁰

Several studies have revealed that 2,3,7,8-TCDD has serious effects on reproductive and immune system development in rats exposed *in utero* and through lactation. Doses of 2,3,7,8-TCDD comparable to daily human intake administered to lactating mice resulted in immunosuppression and thymic atrophy in

their exposed offspring.²⁵¹ Decreased fertility has been observed in both male and female rats exposed *in utero*, and exposure which included lactation showed depressed testosterone level in male offspring as a common outcome.^{252,253}

Reproductive Effects – Dioxin inhibits estrogenic activity in females and may reduce fertility. Female rats exposed to dioxin have experienced altered hormone levels and inhibited estrous cycle and ovulation.²⁵⁴ Effects on male rats include decreased testosterone levels and reduced seminal vesicle weight.²⁵⁵ Animal studies that include chronic doses of 2,3,7,8-TCDD report an increase in severity and incidence of endometriosis in monkeys as well as reduced reproduction rates and increased abortions.²⁵⁶ Most sobering is a study that confirmed reproductive loss and early mortality in confined mink that were fed dioxin-contaminated Great Lakes fish.²⁵⁷

Recent studies have found that men who were occupationally exposed to dioxins had reduced levels of the male sex hormone testosterone.²⁵⁸ Men exposed in the Vietnam war to dioxin-contaminated Agent Orange exhibit reduced testicular size.²⁵⁹

Immune Effects – Dioxins are capable of suppressing both cell-mediated and humoral responses in animals, suggesting that the toxins have a broad range of targets which act to prevent normal functioning of body processes, including innate and acquired immunities.

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V. Challenges and Implications of a Global POPs Agreement

Many countries have already banned or severely restricted the production and use of the twelve POPs in recent decades. Yet they remain a serious problem around the world. Because these chemicals have the ability to travel long distances from their original sources, relying on national-level action alone is ultimately a futile effort. For this reason, the United Nations Environment Program (UNEP) has initiated international negotiations toward a legally-binding global agreement to reduce or eliminate these twelve POPs and set criteria for including new chemicals in the agreement in the future.

The UNEP mandate for treaty negotiations acknowledges the different issues involved with phasing out each of the three types of POPs pollutants:

Pesticides: For the listed POPs pesticides, measures should be taken to rapidly phase out remaining production and subsequent on-going use while alternatives are being made available.

Industrial chemicals: For the listed POPs industrial chemicals, there is need to phase out, over time, PCBs and HCB on a global scale toward the goal of complete elimination. During this transition, remaining use, storage and disposal must be managed in the most protective, feasible, and practical manner.

By-products: For POPs that are generated as unwanted by-products [e.g. dioxins and furans], currently available measures that can achieve a realistic and meaningful level of release reduction and/or source elimination should be pursued expeditiously, and this should be done by actions that are feasible and practical. Additional measures should be explored and implemented.

One of the challenges involved in eliminating POPs is the destruction of obsolete chemicals. Since their bans in many countries, PCBs and pesticides have been stored, awaiting destruc-

tion, in many locations. For example, it is estimated that there are more than 100,000 tons of obsolete pesticides in developing countries, much of it persistent organochlorines, such as DDT and dieldrin. Donor countries, aid agencies, agrochemical companies and recipient governments are all responsible for the steady accumulation of these pesticides, which in Africa alone may cost more than U.S.\$100 million to dispose of.²⁶⁰

Another challenge is the remediation of environmental reservoirs. For example, once in a body of water, POPs may slowly and continually be released from sediments over time as systems slowly purge themselves.²⁶¹ In North America alone, the Great Lakes region is extensively contaminated with PCBs, and the Hudson River is contaminated for 200 miles by PCBs dumped there over the course of decades.²⁶² Because PCBs settle in sediment at the bottom of the water, any clean-up attempt would involve dredging the river bed, an expensive and time consuming process. Overall, 271 of 1,777 hazardous waste sites on the U.S. EPA's National Priority List of Superfund sites are contaminated with PCBs, with clean-up estimated at 18 million dollars each.²⁶³

It is recognized that the elimination of all significant POPs sources, and the remediation of POPs environmental reservoirs will, in many

Three of the 12 POPs, the pesticide DDT, the industrial chemical PCBs, and the by-product dioxin, pose particular challenges because of the ways they are currently used or generated.

cases, be difficult, expensive, and time-consuming. Many of these chemicals will remain in the environment and in the food chain for an extended period of time, even after global elimination measures have been effectively implemented. For this reason, interim management regimes will often be required and appropriate, while longer term phase-out regimes are initiated and begin to take effect.

In North America, for example, the International Joint Commission has advocated the initiation of sunseting POPs in the Great Lakes. Sunseting is defined as the comprehensive process to restrict, phase out, and eventually ban the manufacture, generation, use, transport, storage, discharge and disposal of a persistent toxic substance.²⁶⁴ This approach represents a model for achieving eventual total elimination of POPs around the globe. It recognizes that a ban on chemicals which are currently used, manufactured, or stockpiled in many places around the world cannot be achieved overnight, but advances proactive measures to begin the banning process.

Three of the 12 POPs, the pesticide DDT, the industrial chemical PCBs, and the by-product dioxin, pose particular challenges because of the ways they are currently used or generated.

Pesticides: The Case of DDT

For decades, DDT was the weapon of choice against disease-carrying mosquitoes, for its effectiveness at killing insects with few acute effects on people. Although many countries have since banned DDT, it is still used in some countries, mostly for indoor house spraying as part of government-sponsored public health campaigns. Some countries continue to use DDT for vector control. Countries such as Bolivia, Colombia, Ethiopia, Guinea, India, Kenya, Malaysia, Sudan, Thailand, Venezuela, and Vietnam restrict DDT use for public health use only, with no registered permission for use on agriculture.²⁶⁵ Because of the continued use of DDT, the contaminant

and its metabolites persist in ecosystems far from the source of application.

Malaria currently infects between 300 million and 500 million people each year. Between 1.5 million and 2.7 million die annually, mostly children under five years of age.²⁶⁶ More than 40% of the world's population, in 100 countries, is currently at risk for the disease.²⁶⁷

As of 1994, DDT continued to be manufactured in China, India, Indonesia, Italy, Mexico, the Netherlands, and possibly Russia, Japan and South Korea.²⁶⁸ Many reports of agricultural practices in India claim that illegal DDT use is rampant. DDT compounds have been positively identified in wastewater surrounding open lands at levels that suggest that upstream manufacturers are responsible for the contamination.²⁶⁹

A similar problem exists in Tanzania, where in 1996, pesticide companies were openly selling DDT dust for agricultural application. The DDT product was found in farm retail stores throughout the region in Tanzania that supplies large cities with produce.²⁷⁰ These DDT products were in particular demand because they were less expensive than alternative pesticides. In order to develop and implement a plan to end use of DDT and other organochlorine pesticides, feasible alternatives must be available for insect control.

Mexico is involved in an aggressive research initiative to develop viable alternatives to DDT, and has agreed to begin a phase-out plan for DDT that will attempt to eliminate all use and production by 2007. Alternatives to house spraying with DDT include the use of synthetic pyrethroid insecticides, which though endocrine disrupting, tend not to bioaccumulate. The use of pyrethroids in an integrated pest management (IPM) appears promising. Examples of this approach include the use of biological controls, such as stocking streams with mosquito larvae-eating fish; the use of barriers, such as mosquito nets (often impregnated with pyrethroids) and win-

Historically, PCBs have been disposed of by landfilling or incineration.

dow screens; and aggressive case management. Such methods are safe and sustainable, and have proven to be effective, but they require community participation and a shift in thinking by local and national governments as well as by intergovernmental and aid organizations.

Industrial Chemicals: The Case of PCBs

PCBs pose a particular problem. Due to past production and use, and current improper disposal, significant contamination of soil and water is present throughout many ecosystems.²⁷¹ Although most new production has stopped, these chemicals are still present in large quantities in older equipment still in use, in stockpiles awaiting destruction, and in environmental reservoirs. According to a 1995 report, 4% of all PCBs ever produced have been incinerated, 35% have been released to the environment, and some 60% are either still being used, or are in dumps and landfills; all of these are potential problems.²⁷² These figures are subject to considerable uncertainty, but they illustrate the potential magnitude of the problem of PCB elimination.

There are other problems related to PCB elimination as well. One is the recent discovery that Russia is still producing the chemicals for use in its antiquated electrical system. Previously, it had been thought that no country still produced PCBs. During negotiations leading to the UN Economic Commission for Europe's Aarhus Protocol on POPs, Russia was given a special exemption that allows production until 2005 and calls for destruction of the last of its PCBs by 2020. In the U.S., although the production and use of PCBs as an end product has been banned since 1977, PCBs continue to be produced as by-products and used as intermediates in some chemical processes, representing additional potential for continued environmental contamination.

Historically, PCBs have been disposed of by landfilling or incineration. Landfilling, however,

leaves the PCBs potentially available for future environmental contamination, and incineration leads to the generation of other POPs like dioxins and furans. Neither are acceptable disposal options. Moreover, there are several alternative treatment technologies for disposing of existing stocks of PCBs which are more effective and do not produce toxic by-products. These include, for example, gas phase hydrogenation, which is performed at very high temperatures and leaves only an inorganic ash.²⁷³ No dioxins or furans are produced, and PCBs are reportedly destroyed within 99.9999%.²⁷⁴ This technique can handle most types of PCB waste and has been endorsed by the U.S. Department of Energy. The cost of this method, however, roughly \$400/ton for soils and \$2000/ton for liquids, is very high, as is the case with most of the treatment technologies. The challenge of high cost remains one of the most significant barriers to widespread implementation of these alternative technologies.

By-Products: The Case of Dioxins and Furans

Dioxins are produced when chlorine-containing compounds and products are manufactured or burned. Dioxins cannot be produced without the presence of chlorine and some other organic material. Municipal and medical waste incinerators which commonly burn chlorinated plastics and other materials are the largest sources of dioxins released into the environment in the U.S.²⁷⁵ Pulp and paper mills that use chlorine and chlorine-dioxide bleaches are another major source of dioxins,²⁷⁶ and other sources include hazardous waste incinerators, cement kilns, and facilities that produce chlorine compounds and plastics like polyvinyl chloride (PVC).²⁷⁷ Dioxin sources have been little-regulated in the U.S., and less so in many developing countries.



If dioxin elimination is to be achieved in the U.S. and other countries, it will require a major shift toward alternatives to chlorine in many industrial processes. In some cases, suitable alternatives have not yet been identified, but in others, they are readily available. Chlorine-free plastics, including polyolefins such as polypropylene or

polyethylene, for example, may be substituted for PVC in many products; some U.S. manufacturers of plastic medical products are already doing this. In the pulp and paper industry, alternative bleaching methods that rely on oxygen are now commonly used in some European countries and by a few U.S. manufacturers.

Notes

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VI. The Role of the Public Health Community

National and International Policy

With a global POPs treaty process already underway, now is the time for public health professionals to become involved in this issue. Physicians and public health professionals around the world are seen as opinion leaders and trusted voices in policy debates. As diplomats negotiate an international POPs treaty that will affect the health of all people, they will be looking to the medical community for guidance and expertise. Because of the serious threats to public health posed by the continued manufacture, use, and release of POPs, the health community has a responsibility to be advocates for the public interest.

In particular, the support of health professionals will be crucial to developing the appropriate steps to phase out DDT in vector control without compromising the WHO target of rolling back malaria. Further, health professionals are needed to develop health-based criteria for identifying additional POPs for future international action.

The world's public health associations and their individual members can play an important role in facilitating debate on realistic response strategies, policies and mechanisms for eliminating emissions, discharges, and losses of POPs, and replacing them with safe alternatives. The World Federation of Public Health Associations (WFPHA), for example, is "an international, non-governmental organization bringing health workers throughout the world together for professional exchange, collaboration, and action" which has taken a leading role in addressing these issues.²⁷⁸ The General Assembly of the WFPHA has adopted resolutions on POPs which:

- Note ongoing intergovernmental activity toward a legally-binding global agreement to

eliminate or reduce releases of POPs into the environment;

- Appreciate the need for public health association involvement in the process because much of the rationale for control and elimination of POPs is based on human health effects;
- Recognize that a significant global health educational process will be necessary based on whatever international instrument is adopted;
- Commit to involvement in this global effort; and
- Resolve to seek WFPHA representation at intergovernmental meetings by an appropriate person who will contribute to activities to consolidate information on POPs; analyze transport pathways; examine sources, benefits and risks; evaluate substitutes; and who will observe and report back to WFPHA on activities to assess realistic response strategies, policies and mechanisms for reducing and/or eliminating emissions, discharges and losses of POPs and has initiated the WFPHA POPs Project to implement these objectives.

Because of the serious threats to public health posed by the continued manufacture, use, and release of POPs, the health community has a responsibility to be advocates for the public interest.

Notes

²⁷⁸ World Federation of Public Health Associations, Washington, D.C., http://www.apha.org/wfpha/about_wfpha.htm

VI. Pollution Reduction in Health Care Institutions

Hospitals in the U.S. alone generate over two million pounds of hospital waste a year, more than twice that produced in 1955.²⁷⁹ Changing medical technology clearly has played a role in the rise of the medical waste mountain, due, in part, to the use of more plastic and more disposable products. Experts also point to other factors such as unnecessary red bag disposal of waste, inefficiencies in hospital waste management, excessive use of disposables, and the scarcity of storage space in hospitals. The role of health professionals in affecting the impacts on the environment from these factors is significant. Opportunities for intervention to reduce the waste stream, while still maintaining the highest standards of patient care, are increasing.

One such opportunity is the definition and implementation of practices stemming from the concept of universal precautions. The principle of universal precautions instructs that all body fluids be regarded as potentially infectious in order to properly protect health care personnel. While these precautions are entirely appropriate in the clinical setting, this broad definition also leads to the generation of massive amounts of what is classified as infectious waste. On closer examination, however, the broad designation of what constitutes infectious waste appears to be unjustified.

In reality, only about 10 to 15 percent of hospital waste can be properly described as *infectious waste*. The rest is *solid waste*, made up of paper and paper board, plastics, food waste, metal, glass, wood and other materials. According to the Society for Hospital Epidemiology of America, "Household waste contains more microorganisms with pathogenic potential for humans on average than medical waste."²⁸⁰ Furthermore, according to the U.S. Centers for Disease Control, 2% or less of a typical hospital's waste stream - pathological waste (body parts) - must be incinerated to protect public health and safety.²⁸¹ But hospitals routinely burn 75 - 100% of their waste.²⁸² The unnecessary burning of polyvinyl chloride (PVC) plastic,

paper, batteries, discarded equipment, and other noninfectious materials leads to emissions of dioxins and mercury as well as furans, arsenic, lead, cadmium, and the generation of toxic ash. The U.S. EPA has identified medical waste incinerators as a leading source of both dioxin and mercury pollution of our environment and our food supply.²⁸³

Thus, despite many unique characteristics of health care facilities and enormous variability among those facilities, what constitutes both *infectious waste* as well as most *medical waste* can be reduced; the first by more precise definition and appropriate disposal of actual infectious waste, and the second by using the same waste minimization and reduction techniques used in homes and offices. There are several steps that can be taken to implement these techniques in the health care setting.

Waste minimization and reduction are the most important parts of waste management. Waste reduction begins with the initial process of procurement of hospital supplies. Minimizing packaging and buying products that are durable rather than disposable, when feasible, all lead to reduced waste disposal. By working with vendors, hospital purchasing agents can increase the number of reusable items, reduce the number of disposals, and decrease the amount of

Waste minimization and reduction are the most important parts of waste management.

waste generated hospital-wide. Waste segregation, essential for successful recycling and widely practiced with household waste, is another critical step in reducing the volume and toxicity of the medical waste stream. Paper and cardboard products, glass, some plastics, and metals can readily be recycled in existing markets. Materials sent to waste disposal systems should be true waste and not materials that could or should be reused or recycled. And finally, also of critical importance at the end of the waste cycle, the

waste treatment method used should not create toxic compounds or release pollution into the environment.

Incentives to improve waste stream management are growing as the economic advantages of waste reduction, reuse, and recycling are becoming more apparent. In the U.S. for example, Beth Israel Medical Center in New York City, an institution with thousands of patients, outpatient clinics, and inpatient beds, saves over \$900,000 per year through product purchasing and disposal modifications, including reducing, reusing, and recycling.²⁸⁴

The ability to accomplish this in any health care institution, however, relies on the additional cooperation of professional and institutional staff, who often are not waste-conscious and not accustomed to thinking of the public health impacts of their materials-use practices. Education, persuasion, and, sometimes, if necessary, specific regulatory requirements, are all part of changing the institutional culture into a more waste-conscious environment.

In the professional arena, the World Federation of Public Health Associations has called upon its member national associations to explore methods to eliminate pollution in health care practices without compromising safety or care. It suggests that they:

1. Promote comprehensive pollution prevention practices.
2. Support the development and use of environmentally safe materials, technology and products.
3. Educate and inform health care institutions, providers, workers, consumers, and all affected constituencies about the environmental and public health impacts of the health care industry and solutions to its problems.
4. Advocate the elimination of the nonessential incineration of medical waste and promote safe materials use and treatment practices;
5. Explore methods to phase out the use of PVC (polyvinyl chloride) plastics and persistent toxic chemicals when replaceable by less toxic alternatives.
6. Replace mercury usage in health care products with safer alternatives;
7. Develop just siting and transport guidelines that conform to principles of environmental justice based on the principle that “no communities should be poisoned by medical waste treatment and disposal;
8. Develop effective collaboration and communication structure with other groups concerned about this issue.



Notes

279 Health Care Without Harm. “Greening” Hospitals. Environmental Working Group/The Tides Center. Washington, D.C. 1998.

280 *Ibid.*

281 *Ibid.*

282 *Ibid.*

283 *Ibid.*

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IX. Practical Steps for Health Care Workers to Reduce the Production of POPs:

Health Care Without Harm – A Model For Environmentally Responsible Health Care

The First Nine Steps

Health Care Without Harm (HCWH), a coalition of over 250 organizations in 34 countries, has devised nine practical steps that every health care facility can take to dramatically reduce the harmful impacts of its activities on the environment. In doing so, every facility can benefit financially, improve staff morale, increase worker safety, avoid liability costs, improve regulatory compliance and strengthen its relationship with the community.

STEP ONE: Establish a “Green Team”

Convene a task force of administrators, housekeepers, engineers and others who are currently responsible for waste handling. Authorize this team to:

- Identify the percentage and content of the facility’s waste stream that is currently being incinerated and what is currently being recycled.
- Assign in-house staff or contract with an outside consultant to conduct a waste audit to identify wasteful practices and to design a waste management strategy that incorporates waste reduction, reuse, and recycling measures.

Step Two: Put someone in charge

Assign or hire staff for the full time responsibility of developing and implementing a program that integrates materials purchasing with waste segregation and recycling to reduce the waste stream volume and toxicity through environmentally sensitive work practices.

STEP THREE: Train staff about the environmental consequences of medical waste incineration

Waste handlers, nurses, purchasing staff, boards of directors, medical ethicists, physicians, medical assistants, administrative staff and food service personnel all need to be aware of the problems and costs of unnecessary red-bagging and the availability of less expensive and more protective waste disposal alternatives.

STEP FOUR: Don’t incinerate what you can recycle

Implement or expand your recycling program:

- Cardboard, glass, office paper, drink cans, newspapers and magazines, and plastic have nationwide recycling markets.
- Implement a purchasing program that favors products made of recycled products including recycled paper that has not been bleached with chlorine.
- Communicate with suppliers about the need for totally recyclable or reusable packaging materials.

STEP FIVE: Don't incinerate what you can reuse

Create a plan to assess, on an ongoing basis, the availability of reusable products, and substitute, when feasible, for disposable items.

STEP SIX: Don't incinerate what you can safely dispose of by other methods

The small percentage of hospital waste that is infectious can be sterilized by autoclaving, microwaving, or other alternatives to incineration.

STEP SEVEN: Begin a program to eliminate the use of mercury-containing products within the institution and set a goal to becoming a mercury-free facility by the year 2003

Mercury is present in batteries, thermometers, Miller-Abbot tubes, Cantor tubes, sphygmomanometers, electrical equipment, fluorescent lamps, laboratory reagents and disinfectants. Alternatives already exist for most of these, but where they do not, such as energy efficient, but mercury-containing

fluorescent light bulbs, engage in recycling to avoid releasing mercury into the environment.

STEP EIGHT: Create a plan to reduce the use of chlorinated plastics, such as polyvinyl chloride (PVC), with the five year goal of its near-

complete phase-out from your institution

PVC may be present in ventilator and oxygen therapy tubing, endotracheal tubes, ambu-bags, facemasks and oral airways, IV bags and tubing, dialysis equipment, patient ID bracelets, gloves, protective covers, record binders and mattress covers.

STEP NINE: Assign materials management staff to research and communicate with suppliers concerning the substitution of materials (sterilizing solution, floor cleaners, cooling unit biocides) to:

- reduce toxic chemical inputs
- provide safety to health care employees and
- reduce environmental pollution emissions and impacts.

The WFPHA POPs Project will help hospitals achieve these nine steps by identifying educational materials, referral of experts, guest speaker suggestions, and identifying health care facilities willing to share their experience in becoming environmentally responsible.

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