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## Environmental Pollution Caused by Organic Chemical Compounds

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**ABSTRACT:** The worldwide production and use of organic chemicals (both natural and synthetic) have increased dramatically since World War II. Much of the increase is the result of new compounds developed for consumer use rather than of an increased use of a few known compounds. The environmental impact of using these compounds has only slowly become apparent, and in many cases the findings have caused serious concern. Organic pollutants includes phenols, chlorinated phenols, endocrine disrupting chemicals, azo dyes, polyaromatic hydrocarbons, polychlorinated biphenyls, pesticides, etc., whereas inorganic pollutants includes a variety of toxic heavy metals such as cadmium (Cd), chromium (Cr), arsenic (As), lead (Pb), mercury (Hg), etc. The Stockholm Convention on Persistent Organic Pollutants adopted in 2001 deals with persistent organic pollutants (POPs), i.e. substances that possess toxic properties and resist degradation. They include pesticides such as DDT and lindane, industrial chemicals such as polychlorinated biphenyls (PCBs), and substances such as dioxins, which are the unwanted by-products of manufacturing and combustion processes. As these substances are highly stable and therefore non-degradable to a large extent, they can be transported over long distances and accumulate in the environment.

POPs cause problems because they are stored in the fatty tissue or organs of animals, where they can have toxic effects. For example, they can disrupt the endocrine system, cause cancer or genetic defects, and weaken the immune system.

Various effects of POPs on marine mammals have been investigated. Studies of Baltic ringed seals (*Phoca hispida*) and grey seals (*Halichoerus grypus*) found uterine occlusions, stenoses and tumours, resulting in reduced reproductive ability. Other observed effects included colonic ulcers, as well as reduced bone density, which led to changes in the skeletal system. In seals and porpoises, researchers found indications that POPs depress the immune and endocrine systems. A further topic of discussion in this context is whether these pollutants and the weakening of the immune system affect the spread of epidemics, such as the disease that killed thousands of seals in the North Sea in 1988 and again in 2002 – probably an epidemic of the phocine distemper virus. Humans mainly ingest POPs from food and drinking water, but also from the air (mainly by breathing in dust particles) and through the skin (through direct contact with the chemicals). The highest concentrations of POPs are generally found in marine mammals and humans, both of which are at the top of the food chain.

### I.INTRODUCTION

Organic pollutants include many insecticides and herbicides that have been used in agriculture and pest control. Dichlorodiphenyltrichloroethane (DDT) is a pesticide, highly effective in controlling mosquitos, which was banned in the USA in 1972, but is persistent and its metabolite (DDE) is found in the blood of many populations. Other pollutants were manufactured for use in various industries [e.g. polychlorinated biphenyls (PCBs), phthalates], and others, such as dioxin, are unintended by-products of manufacturing. Phthalates are plasticizers used in bottles, toys and personal care products. PCBs are a large group of similarly structured compounds with variation in toxicity and persistence in the environment and in the body. Some forms are very similar to dioxin. Polybromated diphenyl esters (PBDEs) are fire retardants added to a large variety of consumer items that leach into surrounding materials and now can be detected in many populations.

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Organic pollutants such as PCBs, dioxin and DDT are lipophilic; they are stored in fat cells and can be retained for years. They cross the placenta, and lactation is a significant source of exposure. They are also found in dietary items such as fish, meat and dairy products. PCBs may affect endocrine function, physical growth, maturation and/or cognitive or behavioral development of children and youth. Evidence of the effects of PCBs in humans comes from two types of study: studies of acute poisoning, either food poisoning or an occupational accident, and studies of chronic low-level exposures, usually from ingestion of foods with slight but measurable contamination.[1]

Ingestion of rice oil contaminated with a mixture of PCBs, dioxin and dibenzofurans poisoned thousands of adults and children in Japan in 1968 and in Taiwan in 1978–79, producing diseases called Yusho and Yucheng, respectively. Yusho/Yucheng infants have had higher rates of mortality and lower body weights at birth.<sup>164</sup> Even children born long after their mothers were exposed to the contaminated oil were more often born prematurely and small at birth,<sup>165</sup> probably because during gestation they were exposed to the toxicant mixture that had been stored in their mothers' fat tissue. Reduced postnatal growth also characterizes Yusho/Yucheng children.

Studies of children born to women exposed to smaller amounts of PCBs over a long period have also found growth deficits. A common but not universal finding is that birth weight is reduced in response to PCB exposure. The effect seems not to be due to prematurity but to less growth during gestation. Head circumference may also be reduced. Three studies have related reduced birth weight to fetal exposure to dioxin and dioxin-like compounds. PCB levels and BMI are related early in life and at puberty. DDE, a metabolite of the insecticide DDT, has been related to reduced birth weight and height in several studies, but with increased height and weight for height in others.[2,3]

It is important when interpreting conflicting results to account for differences in exposure among the studies. Certainly one would expect a smaller effect or none at all when the exposure is very low, and it is difficult to ascertain how exposure levels compare across studies because measurement techniques have changed substantially and only the most recent studies measure the contaminant compounds in similar ways. In addition, different effects may stem from differences in the timing of exposure: prenatal versus postnatal. Children exposed to PCBs from maternal consumption of fish from the Great Lakes were significantly lighter at 4 years of age, though not at 11 years of age. The reduction at age 4 was related to the PCB level at birth, reflecting prenatal exposure, but not to their current PCB level. Another longitudinal study found greater height in girls at 5 years of age in a cohort in which there had been differences in size at birth.

Clearly, not all studies of humans agree as to the size and direction of effects that these toxicants have. This creates questions about the influence of differences in levels of exposure, differences in the timing of exposure (prenatal or postnatal) and differences in the mixtures of compounds to which the sample was exposed. Excepting occupational exposures, mundane exposures tend to be a mixture of several compounds. While we tend to group all these persistent organic pollutants as “toxic”, we have learned not to expect them to have similar effects on growth.

We do know that the alterations in growth are best explained as due to interference with hormonal signaling. Further, we know that different compounds affect signaling differently depending on the timing of exposure and their structure as some are agonistic and some antagonistic. Many studies of non-human animals have shown that hormone activity can be altered following exposure to PCBs and related compounds. Thyroid hormone signaling is especially important for normal physical and mental growth and development. Neurological effects are the most consistently reported effects of chronic PCB exposure. A study of Mohawk adolescents, 10–17 years of age, found the combination of reduced thyroxine levels and increased thyroid-stimulating hormone levels (this usually signals low thyroid activity) in relation to levels of persistent PCBs that reflect past exposure, but not in relation to PCBs more reflective of current exposure. This suggests that prenatal or neonatal exposure may be influential.<sup>188</sup> In this same group, there were alterations in performance on some cognitive tests and particular effects on memory.

Studies of the effects of phthalates in children have intensified recently as the material is found in more products and more populations. Effects of phthalates on the thyroid have been reported.<sup>183</sup> Growth and sexual development can also be affected.[4].

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Many types of organic pollutant structurally resemble sex steroids as well as thyroid hormones. The possibility of deranged hormonal signaling of sexual development and reproduction has been a controversial area of research, but there is now sufficient evidence that it occurs in humans. Several studies conducted by different research groups and with different populations have reported that exposure to some pollutants accelerates the development of puberty, although the finding is not universal.

Whether the effect is one induced by prenatal or postnatal exposure, or both, is not clear. Newborns were found to have significantly lower testosterone and estradiol levels in relation to their mothers' levels of dioxin, dibenzofuran (similar to dioxin) and dioxin-like PCBs. Follow-up of boys in the Yucheng cohort who had substantial exposure to dioxin-like compounds showed reduced testosterone levels and increased FSH levels but no difference in Tanner stage. Preliminary results from the same cohort found significantly reduced penile lengths, a possible effect of their exposure prenatally when critical sexual differentiation and development is occurring.

A related compound, PBDE, is a new pollutant of concern. In adult men it has been strongly and inversely associated with a measure of androgen, with LH and FSH, and positively with inhibin B and sex steroid binding globulin. This result and other similar findings strongly suggest that chemicals in our environment can affect levels of hormones directly involved in reproduction and development. In true experimental studies of non-human animals endocrine disruption is clearly evident and this establishes the biological plausibility of the associations seen in observational studies of human populations. Research on children and PBDE is just beginning. If the effects seen in adults are present in children, they certainly could affect growth and sexual maturation.

There is sufficient evidence to be concerned about PCB exposure in children and the fetus. Especially convincing are the controlled laboratory studies of higher primates and rodents that show reductions in growth and alterations in sexual development that are dependent on normally functioning hormonal systems. Although it is not known whether a low-level exposure will produce effects on child development, we do see that high exposures, such as those from food heavily contaminated with PCBs, can produce predictable effects, and the possibility that the fetus is especially sensitive to PCBs, even at low levels, remains a very viable hypothesis.

PCBs are organic pollutants with hydrophobic properties that resist the process of metabolism. PCBs includes chemically-related compounds with insulating and fire-retarding properties . Many forms of PCBs degrade slowly in nature, and thus build up in the food chain .PCBs affect the immune, reproductive, cardiovascular, nervous, and endocrine systems . In addition, these organic pollutants are carcinogenic and overexposure to PCBs has been associated with vascular inflammation and atherosclerosis . PCB toxicity may lead to the inhibition of dopamine transportation . In humans, the most common health problems due to PCB toxicity are skin and eye problems .[5]

Multiple tests involving PCBs have been performed on various animal species. Health effects include reproductive issues, growth inhibition, immunotoxicity, liver toxicity, neurotoxicity, and dermal toxicity among other biochemical effects . For ethical purposes, similar testing has not been performed on humans. The effects of PCBs on humans have been collected through other means. One source of data collection is the history of PCBs in workers through occupational exposure . Another source includes large-scale or widespread accidental exposure in populations A third source includes individuals with low-level environmental exposure in the form of consumption of fish known to contain PCBs . Low-level environmental exposure causes the most concern in the public. Approximately 90% of PCB exposure in humans is because of supplements and the average diet .

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## II.DISCUSSION

Humans may develop PCB toxicity by consuming fish that swim in contaminated waterways. The waters are tainted by the inappropriate disposal of waste materials that contain PCBs. The organic chemicals settle in the sediment rock and are accidentally consumed by bottom dwelling fish. Other fish eat the bottom dwellers and also accumulate PCBs in their systems. Although fish can metabolize some PCBs, the organic pollutants not metabolized or excreted instead accumulate in the fish's fatty tissues. The half-life of PCBs are long in comparison to other chemical compounds. The gradual accumulation of PCB ingestion in humans leads to toxicity. In 2001, the European Union's Scientific Community on Food established a weekly tolerable limit of PCBs of 14 pg WHO-TEQ kg<sup>-1</sup> body weight. One method of reducing PCB exposure is to remove the skin and fat from fish before cooking it. Fortunately, enacted laws have reduced the overall exposure of PCBs in the general population. Research suggests that PCB exposure and concentrations in individuals have shown a significant decrease in the past 25 years. [6]

There are several techniques used to reduce the amount of PCBs in fish oil refining. Generally, PCBs are removed with high temperature deodorization. Another method of removing PCBs from fish oil is the adsorption of the PCBs on apolar adsorbents such as activated carbon. However, this method has not been proven to be very effective. One effective method of removing PCBs from fish oil is steam stripping the chemicals under optimized process conditions. The most effective process of removing PCBs is molecular distillation at an extremely hot and high pressured environment. Unfortunately, this process also removes some of the heart health fatty acids, such as DHA, found in fish products.

Although these techniques are effective in reducing the amount of PCBs in fish, they do not completely remove the organic toxins. Research into ascertaining a safe level of PCBs in food is still being conducted. However, some international organizations, such as the European Union (EU), have established their own maximum levels of PCB intake. A study testing the levels of PCBs in thirty-three fish oil supplements found that twelve of them had levels that exceeded the EU's recommended PCB limit of 14 pg WHO-TEQ kg<sup>-1</sup> body weight. Eleven of these fish oils were extracted from cod, while one of these fish oils came from salmon. The data may show that cod fish have higher levels of PCBs compared to other fish used to produce fish oil. However, research is still being conducted to determine a universally safe level of PCBs. [7,8]

**Organic chemical pollutants** – are those chemicals of organic origin or that could be produced by living organisms or are based on matter formed by living organisms. Common organic chemical pollutants include:

- **Crude oil and petroleum refined products** (e.g., gasoline, diesel fuel, kerosene, mineral spirit, motor oil, lubricating oil);
- **Solvents** (e.g., acetone, MEK, toluene, benzene, xylene) used in industry as well as in many household products;
- **Chlorinated solvents** (e.g., PCE, TCE, 1,1,1-TCA, 1,2-DCA, 1,1,2-TCA) used in industrial degreasing processes, as well as in dry cleaning, and in various household products;
- **PAHs** (polyaromatic hydrocarbons) are found in petroleum products, crude oil, but are also a result of burning activities (e.g., from coal power plants as well as historical manufacturing gas plants);
- **PCBs** (polychlorinated biphenyl ethers) which are now banned but were used in transformers and are already present in large amounts in environment
- **Alcohols** (e.g., ethanol, methanol, isopropanol) are used in a large variety of applications and household products;
- **Trihalomethanes** (e.g., chloroform, dibromochloromethane, chlorobromomethane, bromoform) which are common products of water chlorination
- **Phenols** are usually an indication of waste water and a result of industrial processes;
- **Plastics** are a result of industrial processes as well as our daily activities involving using and disposing of a large variety of plastics (e.g., bags, bottles, containers);
- **Pesticides / Insecticides / Herbicides** are commonly used in agriculture and may contain toxic organic chemicals and metals (such as mercury and arsenic);
- **Detergents** (e.g., nonylphenol ethoxylate) include a variety of chemical compounds with surface activity;

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- **Organo-metallic compounds** (e.g., organo-arsenicals, organo-mercurials) are usually pesticides / insecticides / herbicides.

## III.RESULTS AND CONCLUSION

Volatile organic compounds (VOCs) are emitted as gases from certain solids or liquids. VOCs include a variety of chemicals, some of which may have short- and long-term adverse health effects. Concentrations of many VOCs are consistently higher indoors (up to ten times higher) than outdoors. VOCs are emitted by a wide array of products numbering in the thousands.

Organic chemicals are widely used as ingredients in household products. Paints, varnishes and wax all contain organic solvents, as do many cleaning, disinfecting, cosmetic, degreasing and hobby products. Fuels are made up of organic chemicals. All of these products can release organic compounds while you are using them, and, to some degree, when they are stored.[9]

EPA's Office of Research and Development's "Total Exposure Assessment Methodology (TEAM) Study" (Volumes I through IV, completed in 1985) found levels of about a dozen common organic pollutants to be 2 to 5 times higher inside homes than outside, regardless of whether the homes were located in rural or highly industrial areas. TEAM studies indicated that while people are using products containing organic chemicals, they can expose themselves and others to very high pollutant levels, and elevated concentrations can persist in the air long after the activity is completed.

Health effects may include:

- Eye, nose and throat irritation
- Headaches, loss of coordination and nausea
- Damage to liver, kidney and central nervous system
- Some organics can cause cancer in animals, some are suspected or known to cause cancer in humans.

Key signs or symptoms associated with exposure to VOCs include:

- conjunctival irritation
- nose and throat discomfort
- headache
- allergic skin reaction
- dyspnea
- declines in serum cholinesterase levels
- nausea
- emesis
- epistaxis
- fatigue
- dizziness

The ability of organic chemicals to cause health effects varies greatly from those that are highly toxic, to those with no known health effect.

As with other pollutants, the extent and nature of the health effect will depend on many factors including level of exposure and length of time exposed. Among the immediate symptoms that some people have experienced soon after exposure to some organics include:

- Eye and respiratory tract irritation
- headaches
- dizziness
- visual disorders and memory impairment



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EPA has pursued regulatory control and management of dioxins and furans releases to air, water, and soil. The Clean Air Act requires the application of maximum achievable control technology for hazardous air pollutants, including dioxins and furans. Major sources regulated under this authority include municipal, medical, and hazardous waste incineration; pulp and paper manufacturing; and certain metals production and refining processes. Dioxin releases to water are managed through a combination of risk-based and technology-based tools established under the Clean Water Act. The cleanup of dioxin-contaminated land is an important part of the EPA Superfund and Resource Conservation and Recovery Act Corrective Action programs. Voluntary actions to control dioxins and furans include EPA's Persistent, Bioaccumulative, and Toxics Program and the Dioxin Exposure Initiative, both of which gather information to inform future actions and further reduce risks associated with dioxin exposure.[10]

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