Dioxin, PVC, and Health Care Institutions

What is dioxin?

Dioxin is the name given to a group of persistent, very toxic chemicals. The group includes chlorinated dibenzo-furans, and dibenzo-dioxins, the most toxic of which is 2,3,7,8 -tetra-chlorodibenzo-p-dioxin (TCDD). The group also includes related compounds which are structurally similar and are dioxin-like in their activity. The toxicity of these compounds is measured against TCDD using "toxic equivalents," which assign a fractional potency to each dioxin. Dioxins, defined here as dioxins and furans, have equivalence factors assigned to them. The US Environmental Protection Agency (EPA) has not assigned equivalence factors for brominated dioxins, brominated furans, brominated biphenyls and polychlorinated biphenyls (PCBs), although it is believed each group includes some dioxin-like compounds.

Dioxins and related compounds are highly persistent in the environment and in living organisms. They are bioaccumulative and fat-soluble. Their concentrations increase as they biomagnify up the food chain.

What are the hazards of dioxin?

Dioxins are extremely toxic and potent environmental contaminants. They modulate and disrupt growth factors, hormones, enzymes, and developmental processes. In animals, dioxin causes cancer in multiple organ systems, sometimes at exposure levels as low as nanograms per kilogram of body weight. Prenatal exposure to dioxin in rodents substantially increases the risk of breast cancer later in life.[1] Human epidemiological studies conclude that dioxin causes cancer in humans as well.[2] A draft report by the EPA estimates that as many as one in 1,000 of the most highly exposed people in the general population are at risk of developing cancer because of dioxin.

Dioxin also has widespread effects on reproduction and development, as shown in animal and human studies. Tiny doses in the range of nanograms (one thousandth of one millionth of a gram) to micrograms (one millionth of a gram) per kilogram of body weight of dioxin can cause harm. Exposure to these levels on a single day during pregnancy cause permanent disruption of male sexual development in rodents, including delayed testicular descent, lower sperm counts, and feminized sexual behavior.[3] In primates, small dietary exposures to dioxin are associated with an increased risk and severity of endometriosis.[4] A study in humans also shows higher levels of dioxin in women with endometriosis than in a control population.[5]

Dioxin is particularly toxic to the developing immune system. Animal tests show that nanograms per kilogram doses given 1-4 times during pregnancy cause permanent alterations in the immune system of offspring.[6] Human studies also show an increased susceptibility to infection and changes in immune system parameters as a result of in utero exposure to ambient environmental levels of dioxin and dioxin-like compounds.[7, 8] Low levels of exposure during pregnancy also alter thyroid hormone levels in mothers and offspring, perhaps explaining neurological effects, including learning disabilities, that are seen in carefully conducted primate studies.[9]

How are we exposed?

The US EPA estimates that over 90% of our exposure is through food, with major sources including beef, dairy products, fish, pork, and breast milk. Dermal, oral and inhalation routes of exposures can also be important for people living near dioxin sources.

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What is the level of exposure in the general population?

The general population, through ordinary dietary exposures, carries a current body burden of dioxin that is near or above the levels that cause adverse effects in animal tests. Through food alone, Americans are getting 22 times the maximum daily dioxin exposure considered by the US EPA to be without adverse effects.

Breast milk contamination is such that the nursing infant, during vulnerable periods of development, is exposed to dietary levels of dioxin 35 to 65 times the amount considered safe. Nonetheless, breast feeding remains far superior to formula feeding for a variety of reasons, and reducing breast feeding is not an appropriate public health response.

What are the sources of dioxins?

Dioxins are unintentionally formed during a variety of industrial processes that include chlorinated substances. Dioxin-like compounds can be generated and released to the environment from various combustion processes when chlorine donor compounds are present. Chlorine donor compounds can include polyvinyl chloride (PVC) plastic and other chlorinated compounds. Dioxin compounds can also be formed during the manufacture of chlorinated materials like PVC, chlorinated solvents and pesticides. Dioxins can also be formed during the bleaching of paper with chlorine, and in other industrial and combustion processes that include the presence of chlorine.

The primary source of dioxins from the health care sector has historically been waste incineration. Chlorine-containing products burned in incinerators, including medical devices and products, provide the chlorine necessary for dioxin formation.

New federal rules have resulted in the closing of most medical waste incinerators. In addition, large incinerators that will continue to operate must meet stricter emission limits. Those rules will eventually reduce the health care sector's contribution to dioxin levels in the environment. Although the contribution of dioxin from incinerators is declining, dioxin sources related to health care remain. The production of materials to create chlorinated health care products, like PVC IV bags and gloves, can result in dioxin formation. In addition, chlorinated health care waste that is burned in backyard burn barrels, or catches fire once taken to the landfill, has the potential to create and disperse dioxin. Once dioxin is emitted into the air from incinerators and other sources, rain, snow and dust can carry it to the surface of the earth, where it can enter the food chain.

What is the evidence that the manufacture of PVC feedstocks is linked to dioxin formation?

The draft dioxin reassessment recently released by the EPA reviews the contribution of PVC manufacturing to dioxin emissions.[10] According to calculations of the Vinyl Institute (an industry trade association), reviewed and given a medium confidence rating by the EPA,[11] the production of PVC and its feedstocks result in air releases of 11.2-31.0 grams toxic equivalency (TEQ)[12] dioxins and furans per year. These levels may understate the contribution of dioxin from the manufacture of PVC throughout its life cycle, both because there may be dioxin releases to land and water during the production phase, and because dioxin may be formed during disposal of the end product.

Under what conditions can the combustion of PVC result in dioxin formation?

The draft EPA dioxin reassessment also reviews the contribution of waste incineration to dioxin emissions. The report summarizes a large body of literature that finds carbon and catalysts must be present in an incinerator in order for dioxins to form.[13] PVC is usually the largest chlorine source in municipal and medical waste incinerators. The relationship between chlorine inputs into an incinerator and dioxin formation, however, depends upon combustion conditions.

For uncontrolled combustion, such as open burning of household waste, landfill fires, or building fires, a direct association between chlorine content of the combusted material and dioxin formation has been established. For example, a study of the open burning of household waste showed that waste containing larger amounts of PVC (4.5% vs. 0.2%) produced substantially larger amounts of dioxins in air emissions (269 vs. 44.3 microgram/kg waste burned) and ash (7,356 vs. 489 microgram/kg waste burned).[14]

In modern, commercial waste incinerators, the rate at which dioxins are formed and released depends upon chlorine inputs, incinerator design, operating conditions, the presence of catalysts, and pollution control equipment. While the EPA concludes, based on studies of modern waste incinerators, that the largest determinants of dioxin formation are operating conditions (including overall combustion efficiency, post-combustion flue gas temperatures, and residence times — and the presence of iron or copper catalysts) rather than chlorine content alone, there is little doubt that chlorine content of the waste feed is critical.

Several laboratory and incinerator pilot studies have found a direct relationship between chlorine loading and dioxin emissions.[15] In addition, the EPA's conclusion appears to rest largely on an analysis of incinerator emissions data by Rigo, et al. (1995), which has serious methodological flaws.[16] It is also important to note that the EPA conclusion refers only to stack gas emissions, which are a relatively small fraction of total dioxins released from incinerators, and does not consider releases in fly ash, bottom ash, and water discharges.

For any given waste incinerator, according to the EPA, conditions may exist in which changes in chlorine content of waste feed will correlate highly with dioxin and furan emissions. These conditions may prevail during start-up or shut-down, changes in waste feed rate, or operational upsets. Although modern commercial waste incinerators are designed and intended to be operated to minimize release of dioxins and other hazardous air pollutants, they are, nevertheless, an important source of dioxin releases.

What is Health Care Without Harm's position on dioxin, PVC, and medical waste incineration?

Available data reveal a complex relationship among chlorine feed, design and operating conditions, and dioxin emissions. It is certain that chlorine sources are necessary for dioxin emissions, and PVC products are a large chlorine source. It is also certain that combustion, even in well controlled incinerators, will release dioxins in stack gases, fly ash, bottom ash, and water discharges. Moreover, even modern, well-designed incinerators do not consistently operate at optimal combustion conditions. Further, not all burning of chlorinated products occurs in controlled conditions, and uncontrolled burning can result in large dioxin releases.

For these reasons, along with concern about other hazardous pollutants emitted from waste incinerators — including mercury, particulates, sulfur and nitrous oxides, and hydrochloric acid — Health Care Without Harm has taken the pollution prevention position that PVC use should be minimized and ultimately eliminated, alternatives should be used when available without compromising patient safety or care, and all unnecessary waste incineration should be avoided.

Notes

1. Brown NM, Manzolillo PA, Zhang JX, et al. Prenatal TCDD and predisposition to mammary cancer in the rat. Carcinogenesis 19(9):1623-1629, 1998.
2. Steenland K, Piacitelli L, Deddens J, et al. Cancer, heart disease, and diabetes in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin. J Natl Cancer Inst 91(9):779-786, 1999.
3. Mably TA, Moore RW, Peterson RE. In utero and lactational exposure of male rats to 2,3,7,8-tetrachlorodibenzo-p-dioxin: 1. Effects on androgenic status. Toxicol Appl Pharmacol 114:97-107, 1992; and Schantz SL, Bowman RE. Learning in monkeys exposed perinatally to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). Neurotoxicol Teratol 11(1):13-19, 1989.
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6. Birnbaum LS. Workshop on perinatal exposure to dioxin-like compounds. V. Immunologic effects. Environ Health Perspect 103(suppl 2):157-160, 1995.
7. Weisglas-Kuperus N, Koopman-Esseboom C, et al. Immunologic effects of background prenatal and postnatal exposure to dioxins and polychlorinated biphenyls in Dutch infants. Pediatr Res 38:404-410, 1995.
8. Weisglas-Kuperus N, Patandin S, Berbers G, et al. Immunologic effects of background exposure to polychlorinated biphenyls and dioxins in Dutch preschool children. Environ Health Perspect 108(12):1203-1207, 2000.
9. Koopman-Esseboom C, Morse DC, Weisglas-Kuperus N, et al. Effects of dioxins and polychlorinated biphenyls on thyroid status of pregnant women and their infants. Pediatr Res 36(4):468-473, 1994.
10. See US EPA, Report #: EPA/600/P-00/001Ab, March 2000
11. The EPA developed a three-part confidence rating scheme: "high" means the estimate is derived from a comprehensive survey; "medium" is based on estimates of average activity and number of facilities or a limited survey; and "low" is based on data judged possibly non-representative
12. Since the toxicity of the various congeners of dioxins and furans varies, the toxicity of a given mixture of congeners is usually expressed as TEQs, where the most toxic form is assigned a value of one and the relative contribution of others is calculated accordingly.
13. Dioxins/furans form most readily in commercial incinerators as the combustion gases reach cooler temperatures, primarily in the range 200-450°C.
14. Lemieux PM. Evaluation of emissions from the open burning of household waste in barrels. US EPA. EPA/600/SR-97/134, 1998.
15. For example, see: Bruce, et al, The role of gas phase Cl2 in the formation of PCDD/PCDF during waste combustion, Waste Management, 11: 97-102, 1991; Kanters, et al, Chlorine input and chlorophenol emission in the lab-scale combustion of municipal solid waste, Environmental Science and Technology, 30: 2121-2126, 1996; and Wagner and Green, Correlation of chlorinated organic compound emissions from incineration with chlorinated organic input, Chemosphere, 26: 2039-2054, 1993.

16. In 1995, the Vinyl Institute commissioned a report, prepared for the American Society of Mechanical Engineers, that purported to examine the relationship between PVC in incinerator waste feed and dioxin emissions (Rigo HG, Chandler JA, Lanier WS, *The relationship between chlorine in waste streams and dioxin emissions from combustors*, The American Society of Mechanical Engineers, 1995). After examining data from dozens of burns in a number of municipal and medical waste incinerators, the report concludes that there is no statistically significant relationship between fuel chlorine content and dioxin emissions. The analysis, however, is flawed in a number of significant ways. First, there was no attempt to control for differences in incinerator design or operating conditions so that the question of interest could be addressed independent of other variables. Second, the authors used data collected for regulatory compliance purposes and not intended to examine the relationship between chlorine input and dioxin output. Without actually knowing the PVC content of the waste feed, they were forced to use hydrochloric acid emissions as a surrogate for chlorine loading. Hydrochloric acid emissions can be used to approximate chlorine loading but do not provide precise estimates. Moreover, in the tested incinerators, dioxin concentrations were sampled at various points in the exhaust stream – from boiler outlet to further downstream – predictably a source of variability, since dioxin can be formed at various points in the exhaust, depending on temperature and fly ash composition. This sampling strategy provides a poor estimate of total dioxin emissions to the air and ash. In summary, this analysis relies on data that are poorly suited to answer the question of interest. A more complete referenced discussion of the connection between PVC incineration and dioxin formation may be found in: Thornton J., *Pandora's Poison: Chlorine, Health, and a New Environmental Strategy* (Chapter 7), MIT Press: Cambridge MA, 2000.

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