



Information Sheet 1

Dioxin: *Summary of the Dioxin Reassessment Science*

Scientists from the Environmental Protection Agency (EPA), other federal agencies and the general scientific community have conducted a reassessment of dioxin exposure and human health effects since 1991. This information sheet summarizes the draft reassessment, which is entitled *Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds*. A more in-depth discussion can be found in the companion piece, *Dioxin: Scientific Highlights from the NAS Review Draft of EPA's Dioxin Reassessment*.

The term “dioxin” refers to a group of chemical compounds that share certain similar chemical structures and mode-of-action biological characteristics. A total of 30 of these dioxin-like compounds exist and are members of three closely related families: the chlorinated dibenzo-*p*-dioxins (CDDs), chlorinated dibenzofurans (CDFs) and certain polychlorinated biphenyls (PCBs). The term dioxin is also used for the most well-studied and one of the most toxic dioxins, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). CDDs and CDFs are not created intentionally, but can be produced inadvertently in nature and by a number of human activities. Combustion, chlorine bleaching of pulp and paper, certain types of chemical manufacturing and processing, and other industrial processes all can create small quantities of dioxins. PCBs are no longer manufactured in the United States but formerly were widely used as coolants and lubricants in electrical equipment.

Combining Risks from Dioxins - the Toxic Equivalents Approach:

Dioxins are believed to cause toxic effects in similar ways; that is, they share a “common mechanism of toxicity.” As a result, EPA and others use an approach that adds together the toxicity of individual dioxins in order to evaluate complex environmental mixtures to which people are exposed. Because dioxins differ in their toxic potential, the toxicity of each component in the mixture must be accounted for in estimating the overall toxicity. To do so, international teams of scientists have developed Toxic Equivalency Factors that compare the toxicity of different dioxins. Given these factors, the toxicity of a mixture can be expressed in terms of its Toxic Equivalents (TEQ), which is the amount of TCDD it would take to equal the combined toxic effect of all the dioxins found in that mixture. The use of the TEQ approach represents a key assumption upon which many of the conclusions in the reassessment are based.

Dioxin Toxicity:

The reassessment finds that, based on all available information, dioxins are potent animal toxicants with potential to produce a broad spectrum of adverse effects in humans. Dioxins can alter the fundamental growth and development of cells in ways that have the potential to lead to many kinds of impacts. These include, for example, adverse effects upon reproduction and development; suppression of the immune system; chloracne (a severe acne-like condition that sometimes persists for many years); and cancer. EPA characterizes the complex mixtures of dioxin to which people are exposed as a “likely human carcinogen.” This is based on the fact that individual components of this mixture could be characterized as “human carcinogens” or “likely human carcinogens” under EPA’s draft cancer risk assessment guidelines (1996, 1999). In particular, TCDD, the most toxic of the dioxins, can be identified as a “human carcinogen” under the Agency’s draft guidelines, based on the weight of the animal and human evidence, and the other dioxins as “likely human carcinogens.”

Dioxin Exposure:

The reassessment proposes that most dioxin enters ecological food webs by being deposited from the atmosphere, either directly following air emissions or indirectly by processes that return dioxins already in the environment to the atmosphere. Once they reach the environment, dioxins are highly persistent and can accumulate in the tissues of animals. EPA estimates that most dioxin exposure occurs through the diet, with over 95% of dioxin intake for a typical person coming through dietary intake of animal fats. Small amounts of exposure occur from breathing air containing trace amounts of dioxin on particles and in vapor form, from inadvertent ingestion of soil containing dioxin, and from absorption through the skin contacting air, soil, or water containing minute levels. These processes result in widespread, low-level exposure of the general population to dioxins.

Dioxin levels in the environment have declined significantly since the 1970s following EPA regulatory controls and industry actions. EPA's best estimates of emissions from sources that can be reasonably quantified, indicate that dioxin emissions in the United States decreased by about 75% between 1987 and 1995, primarily due to reductions in air emissions from municipal and medical waste incinerators, and substantial further declines continue to be documented. Regulations promulgated in 1995 for municipal waste combustors and 1997 for medical waste incinerators should result in a greater than 95% reduction in dioxin emissions from these two categories. Uncontrolled combustion such as burning of household waste is expected to become the largest quantified source of dioxin emissions to the environment. Dietary intake of dioxin also appears to be declining.

Dioxin Effects in Human Populations

EPA estimates that the amount of dioxin found in the tissues of the general human population (which is known as the "body burden") closely approaches (within a factor of 10) the levels at which adverse effects might be expected to occur, based on studies of animals and highly exposed human populations. Despite the potential risks, currently there is no clear indication of increased disease in the general population attributable to dioxin-like compounds. This may be due to limitations of current data and scientific tools rather than indicating that dioxin exposure is not causing adverse effects. For cancer, EPA estimates that the risks for the general population based on dioxin exposure may exceed 1 in 1,000 increased chance of experiencing cancer related to dioxin exposure. Actual risks are unlikely to exceed this value and may be substantially less. This range for cancer risk indicates an about 10-fold higher chance than estimated in EPA's earlier (1994) draft of this reassessment.

Children and Other Groups of Concern

Fetuses, infants, and children may be more sensitive to dioxin exposure because of their rapid growth and development. Data on risks to children are limited, however, and it is not known if the children in the general population are experiencing adverse effects from dioxin. Although breast milk appears to be a significant source of dioxin exposure for nursing infants, the overwhelming body of evidence supports the health benefits of breastfeeding despite the potential presence of dioxin. Other populations have experienced elevated exposures to dioxin as a result of food contamination incidents around the world, through the workplace or from industrial accidents, or from consumption of unusually high amounts of fish, meat, or dairy products containing elevated levels of dioxins. In some cases, such as U.S. Air Force personnel exposed to the herbicide Agent Orange contaminated with dioxin during the Vietnam War, dioxin exposure has been associated with adverse health effects.

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