Dioxin: A Fact Sheet

Dioxin is the name given to a group of persistent, very toxic chemicals. The most toxic form of dioxin is 2,3,7,8-tetrachlorodibenzo-p-dioxin or TCDD. TCDD is more commonly recognized as the toxic contaminant found in Agent Orange and at Love Canal, New York and Times Beach, Missouri. Dioxin is not deliberately manufactured. Rather, it is the unintended by-product of industrial processes that use or burn chlorine.

Garbage incinerators and medical waste incinerators are two of the largest sources of dioxin identified by the U.S. Environmental Protection Agency. Dioxin released from these and other sources has been found to travel long distances in the atmosphere. Some of the highest levels of dioxin in people have been found in the Arctic, even though there are no sources within hundreds of miles.

The US EPA completed its first health assessment of dioxin in 1985. The agency’s estimate in this report of the cancer risk to humans from dioxin exposure was by far the highest defined for any chemical by any government agency anywhere in the world at the time. Industries that would be affected by regulations to limit or eliminate dioxin emissions and discharges into the environment challenged EPA’s findings. As a result, EPA began an internal review of the cancer risk of dioxin in 1986 and then, in 1991, EPA Administrator William Riley announced that the agency would conduct a full reassessment of the health risks posed by dioxins.

In 1994 the EPA released a draft report titled “Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds,” which is commonly known as the Dioxin Reassessment. This draft report was reviewed by the agency’s Science Advisory Board (SAB), which approved most of the document but requested revision of several chapters. These revisions were made by the agency, which released its final draft report in June 2000. These revisions were reviewed by several external peer review groups and by another committee of the SAB, which approved the reassessment document completing the final step in the agency’s peer review process. On June 1, 2001, the EPA’s Science Advisory Board sent a letter to Administrator Christine Whitman asking that the agency “proceed expeditiously” to complete and release the Dioxin Reassessment to the American people.

Instead, the agency sent the revised final draft to an Interagency Working Group (IWG) for one more review. The IWG was organized by the White House and consisted of representatives from multiple federal agencies and was heavily influence by the Office of Budget and Management. It took eighteen months for the EPA to send the reassessment to the IWG who failed to come to consensus on the dioxin reassessment. A Congressional rider to the 2003 EPA appropriations bill then required the National Academy of Sciences to review the EPA’s reassessment. A year later the NAS committee was formed and in July 2006, the NAS released its report and on the dioxin
reassessment. Since that time, the EPA has been preparing a response that is scheduled to be released in the spring of 2010. This response will then be reviewed by the agency’s SAB which is scheduled to be completed by the end of 2010. At that time the EPA will complete and release the reassessment report. EPA Administrator Lisa Jackson has committed publicly to completing the reassessment.

According to the EPA, over 96 percent of human exposure occurs through the diet, primarily foods derived from animals. Dioxin in air settles onto soil, water, and plant surfaces. It does not readily break down in the environment and over time accumulates in the grazing animals that eat these plants. People then ingest the dioxin contained in meat, dairy products and eggs. Some exposure also results from eating dioxin-contaminated fish. Dioxin exposure of the general population is a problem of cumulative emissions from many sources.

Dioxin causes a wide array of adverse health effects in both animals and humans. Dioxin is a potent cancer-causing agent and is considered to be a “human carcinogen” by the World Health Organization’s International Agency for Research on Cancer and the U.S. Department of Health and Human Services’ National Toxicology Program. According to the US EPA draft report on dioxin's health effects, the levels of dioxin-like compounds found in the general population may cause a lifetime cancer risk as high as one in 1,000. This is 1,000 times higher than the generally "acceptable" risk level of one in a million. The human epidemiological evidence provides consistent findings of increased risk for all cancers combined and lung cancer in occupational studies as well as evidence of tissue specific increases in cancer. Increased mortality from soft-tissue sarcomas and all cancers among workers exposed to dioxin has also been reported.

Dioxin also causes a wide range of non-cancer effects including reproductive, developmental, immunological, and endocrine effects in both animals and humans. Animal studies show that dioxin exposure is associated with endometriosis, decreased fertility, inability to carry pregnancies to term, lowered testosterone levels, decreased sperm counts, birth defects, and learning disabilities. In children, dioxin exposure has been associated with IQ deficits, delays in psychomotor and neurodevelopment, and altered behavior including hyperactivity. Studies in workers have found lowered testosterone levels decreased testis size, and birth defects in offspring of Vietnam veterans exposed to Agent Orange.

Effects on the immune system of the developing organism appear to be among the most sensitive endpoints studied. Animal studies show that dioxin decreased immune response and increased susceptibility to infectious disease. In human studies, dioxin was associated with immune system depression and alterations in immune status leading to increased infections. Dioxin can also disrupt the normal function of hormones—chemical messengers that the body uses for growth and regulation. Dioxin interferes with thyroid levels in infants and adults, alters glucose tolerance, and has been linked to diabetes.

References


