

“Developmental Effects of Endocrine-Disrupting Chemicals in Wildlife and Humans” (1993), by Theo Colborn, Frederick S. vom Saal, and Ana M. Soto

“Developmental Effects of Endocrine-Disrupting Chemicals in Wildlife and Humans,” was published in 1993 in *Environmental Health Perspectives*. In the article, the authors present an account of two decades' worth of scientific research that describes the effects of certain pollutants on the health of wildlife, domestic animals, and humans, particularly when exposure takes place during embryonic growth. The term endocrine disruptor was coined in the article to describe the chemical pollutants that target the development and function of the endocrine system. Since its publication, “Developmental Effects” has increased research interest in endocrine disruption and has raised awareness among the general public, the scientific community, and government organizations about the effects that some chemicals may have on development and reproduction.

The article was a collaboration between environmental health analyst Theo Colborn from the W. Alton Jones Foundation in Charlottesville, Virginia, and the World Wildlife Fund, in Washington, DC; developmental biologist Frederick S. vom Saal from the University of Missouri, in Columbia, Missouri; and cell biologist Ana M. Soto from Tufts University, in Boston, Massachusetts.

Endocrine disruptors are chemical compounds that can interfere with the development of the endocrine system of developing embryos or fetuses, as well as the development of organs that respond to endocrine signals. In some cases, the chemical compound mimics a hormone, such as estrogen, and binds directly to and activates some hormone receptor sites that are on special target cells in organs. In other instances, the endocrine disruptor binds to the hormone receptor sites, but deactivates them. The endocrine disruptor may also modify the metabolism of natural hormones, modify the number of receptor sites in a cell, or modify the production of natural hormones. The chemical compounds driving such changes are in some drugs, pesticides, plastics, industrial by-products, and in naturally produced botanical chemicals.

When Colborn, vom Saal, and Soto began collaborating, researchers had yet to extensively document the effects of endocrine-disrupting chemicals. Colborn and team devote the first portion of the article to describing the risks of endocrine disruptors and emphasizing the results of exposure during embryonic, fetal, or neonatal life. The authors then discuss the effects of endocrine disruptors on non-human animals, documented from observations and experiments on wildlife exposed to such chemicals. In the third section of the article, the authors examine the use and effects of diethylstilbestrol (DES), a synthetic estrogen prescribed to women in the US and other countries, including the UK and France, between 1948 and 1971. DES was used as a treatment to reduce complications during pregnancy. Colborn and colleagues present this period as an example of the effects on fetuses of exposure to estrogenic chemicals during human development. The fourth section reviews the prevalence and effects of various endocrine disrupting chemicals, focusing on dichlorodiphenyltrichloroethane (DDT), a pesticide; polychlorinated biphenyls (PCBs), a group of chemicals used in industrial applications; and dioxins, a group of chemicals created as industrial waste. In the conclusion of the article, the authors summarize the overarching effects of endocrine disruptors on wildlife, domestic animals, and humans.

In the introduction of “Developmental Effects of Endocrine-Disrupting Chemicals,” Colborn, vom Saal, and Soto discuss the long-term health consequences of exposure of developing organisms to endocrine-disrupting chemicals. Endocrine-disrupting chemicals are prevalent in rain water, well water, lakes and oceans, and they contaminate food products from some fresh water, marine, and

terrestrial sites. The authors present the widespread occurrence of these chemicals as a pressing issue. Exposure to these chemicals is difficult to avoid, the chemicals can affect humans during development and in adulthood, and the effects can be difficult to track, as in many cases the manifestation of associated defects may be delayed until sexual maturity. Evidence supports that exposure to endocrine disruptors during early embryonic, fetal, or neonatal life causes the most severe developmental effects. A Developing organism can be exposed to disruptors during early stages of life if the pregnant female encountered the disruptors prior to pregnancy. Because many endocrine-disrupting chemicals dissolve easily in fat, a disposition called lipophilic, they can be stored in body fat that may then be used to fuel embryonic or fetal development, or to produce milk during lactation, thereby exposing offspring during development even if the pregnant female is not exposed during offspring development. In addition to prenatal and neonatal exposure, animal studies demonstrate that even in low concentrations, adult organisms chronically exposed to disruptors can have permanent changes in their brain, vaginal or prostate tissues.

Exposure of wildlife to endocrine-disrupting chemicals has a variety of effects, depending on the species and on the type of chemical exposure. Sites where man-made chemicals, such as pesticides or chemical waste, are present, correlate with the strongest deviations from typical body functions. Birds and fish that have been exposed to endocrine disruptors exhibit abnormal thyroid functions in addition to decreased fertility, an effect that also impacts exposed shellfish and mammals. In fish, turtles, and birds, the viability of offspring decreases following exposure. Other effects of exposure include altered immune function as well as potential for demasculinization and feminization, terms used to describe the suppression of functions that are sex-specific, such as those performed by the reproductive systems.

Colborn and colleagues also examine the lasting effects of endocrine-disrupting chemicals on bald eagles. In the Great Lakes of Canada and the US, many salmon have high concentrations of endocrine-disrupting chemicals in their tissues, resulting in developmental abnormalities associated with the exposure, such as enlarged thyroids, poor egg survival, and eggs with low amounts of thyroid hormone. Many bald eagles fed on the contaminated fish of the Great Lakes and had offspring that died due to weakened eggshells. As the survival rate of chicks failed to maintain a steady population, bald eagles from other islands migrated into the Great Lakes population. However, after two years of feeding from the lakes, the new eagles began to suffer the same consequences from the contaminated food supply.

Scientists found that the eagles carried elevated levels of multiple endocrine disruptors, some of which had been regulated in the US, such as DDT and PCBs. Though regulated in the US, many of these chemicals are still manufactured and widely used abroad. DDT and PCB chemicals vaporize easily and can travel long distances in the atmosphere. Researchers estimated that 90 percent of the DDT and PCB found in Lake Superior reaches the area through the movement chemicals through the air.

After describing the specific effects of endocrine disruptors on wildlife, Colborn, vom Saal, and Soto address the diethylstilbestrol (DES) syndrome in humans as well as in rodents. The offspring of approximately one million women who took DES between 1960 and 1970 serve as a model for early life exposure to estrogenic chemicals. Studies indicate that reproductive organ dysfunction, abnormal pregnancies, reduction in fertility, immune system disorders, and depression are common outcomes for daughters whose mothers took DES while pregnant. Furthermore, women exposed to estrogenic chemicals in utero have greater than normal incidence of reproductive organ cancers. Researchers also observed similar effects on organs and reproductive and immune systems in mice exposed to DES at a young age. Colborn, vom Saal, and Soto point toward agricultural and industrial chemicals, whether produced in the US or abroad, as capable of disrupting estrogen receptors in a similar manner to DES.

After demonstrating the effects of endocrine disruptors on wildlife and humans, the authors reiterate their main points and provide additional statistics and evidence to further characterize endocrine-disrupting chemicals. Colborn, vom Saal and Soto reinforce that thousands of endocrine-disrupting chemicals have been released into the environment and that PCBs and DDTs are prevalent in human breast milk and fat tissue. Because endocrine disruptors take a long time to break down, readily dissolve in fat, and can be transported through the atmosphere, it is difficult to inhibit

widespread distribution of endocrine disruptors. The authors note case studies of women who fed on Lake Michigan fish for six years preceding their pregnancies. The women's newborn offspring, when compared to normal offspring, were slightly preterm with lower birth weights, smaller skull circumferences, and deficits in cognitive, motor, and behavioral skills, and some had lasting neurological effects. At least five percent of the babies born in the US are estimated to have been exposed to quantities of PCBs sufficient to cause neurological impairments. It is probable that breast milk accounts for a large percentage of this exposure, as breast milk has a much higher concentration of endocrine-disrupting chemicals than does maternal blood.

Colborn, vom Saal and Soto conclude by summarizing the harmful effects that endocrine-disrupting chemicals in the environment can cause in wildlife and human populations. The authors note that at the time of publication, there were no specific US federal mandates to slow the spread of endocrine-disrupting chemicals. They said that they hoped the article would raise awareness of the risks of endocrine-disrupting chemicals and alert their readers of the need for more extensive research as a first step toward remedy.

"Developmental Effects of Endocrine-Disrupting Chemicals in Wildlife and Humans" has been cited greater than 2800 times as of 2014 and helped prompt the US government to create relevant policy. In 1996, the Endocrine Disruptor Screening Program was founded as a subsidiary of the US Environmental Protection Agency. The same year, Congress passed the Food Quality Protection Act and amended the Safe Drinking Water Act, mandating the screening and testing of food and water in order to determine whether they possess endocrine-disrupting chemicals. In 2003, Colborn founded the Endocrine Disruption Exchange in Paonia, Colorado, a non-profit organization devoted to environmental health factors caused by endocrine disruptors. She also went on to establish and direct the Wildlife and Contaminants Program at World Wildlife Fund in the US.

Sources

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