

## Passing Down Pollution Calculating Intergenerational Exposure to PCBs

Countries around the world began phasing out production of polychlorinated biphenyls (PCBs) in the 1970s after adverse health and environmental effects of these chemicals came to light. A new study calculating intergenerational differences in human PCB exposure now suggests some of the highest exposures occurred well after the chemicals were phased out [*EHP* 119(5):641–646; Quinn et al.].

PCBs are manufactured organic chemicals that were used in numerous industrial applications starting in the 1920s. Although they have been phased out of production, PCBs are still being released into the environment from preexisting applications and improper disposal of products that contain them. Because of their chemical stability and transportability they bioaccumulate in the food chain, readily enter the human food supply, and pass from mother to infant *in utero* and during breastfeeding, with potentially harmful effects on the endocrine, immune, nervous, and reproductive systems.

In 2010 three authors of the current study and another colleague developed and tested a mechanistic model called CoZMoMAN that accounts for PCBs' emissions history and movement through the environment, the food chain, and the human body to predict concentrations in a population's body fat. For the present study, the authors used CoZMoMAN to calculate how PCB concentrations in fat changed over

the lifetimes of hypothetical Swedish women born each decade between 1920 and 2010. They also analyzed how such factors as the age at which a woman first gives birth, the number of children she bears (parity), and whether she breastfeeds her babies affect both her PCB body burden and that of her children.

The model predicted that a woman's PCB exposure was determined primarily by when she was born. Women born in the 1960s were predicted to have the highest cumulative lifetime exposure—PCBs were already in widespread use when these women were born, and emissions peaked as they were maturing in the 1970s. However, it was women born in the 1980s who experienced the highest prenatal exposure and the highest peak concentration at any given age. Prenatal exposure to PCBs can be associated with serious neurologic and reproductive consequences.

Prenatal exposure was strongly influenced by children's birth order, whereas postnatal exposure was influenced by whether children were fed breast milk or infant formula. A mother's reproductive characteristics—specifically breastfeeding and parity—had a greater impact on her children's PCB burden than on her own, but these characteristics only appeared to matter for infants born after the PCB phaseout. Because both pre- and postnatal PCB exposures have been associated with health complications, the results suggest the health effects of PCBs are likely to persist over several more generations.

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## Window for Dioxin Damage Sperm Quality in Men Born after the Seveso Disaster

Animal studies demonstrate that endocrine-disrupting chemicals such as dioxin and dioxin-like compounds are particularly damaging when exposure occurs during prenatal and early-life development. A new study links dioxin exposure within this time frame in humans to reduced sperm quality in adult males [*EHP* 119(5):713–716, Mocarelli et al.].

In July 1976, a trichlorophenol plant explosion near Seveso, Italy, resulted in dioxin contamination of the surrounding area. Thirty-nine men who were born near Seveso between March 1977 and January 1984 made up the exposed group in the current study, while 58 age-matched controls were born outside the contaminated area. Archived blood serum samples collected from the exposed men's mothers in 1976–1977 were used to estimate the men's prenatal dioxin exposure. All the men completed a health and lifestyle questionnaire and provided blood and semen samples. Blood samples were used for health screening tests, dioxin measurements, and hormone assays, and semen samples were analyzed for sperm motility, concentration, and morphology.

Exposed mothers had an estimated median serum dioxin concentration of 26.0 ppt at conception, whereas the median for the comparison group was estimated at 10.0 ppt. Twenty-one of the 39 exposed men were breastfed, which increased their median estimated total dioxin exposure to 40 ppt at 4–5 months of age, a critical time point for proliferation of Sertoli cells, which determine spermatogenic potential in adulthood.

Breastfed exposed men had significantly decreased sperm concentration, total sperm count, and total number of motile sperm in contrast to the 58 men in the comparison group. Compared with both the 36 breastfed comparison men and the 18 formula-fed exposed men, the 21 breastfed exposed men also had increased

follicle-stimulating hormone and decreased inhibin B, a hormone pattern previously shown to be a marker for impaired spermatogenesis.

This is the first human study to show that dioxin exposure during development may permanently impair sperm production in adulthood. Current serum dioxin concentrations in the U.S. and European general populations of infants are far below levels that would trigger adverse effects. However, the study results may explain widespread low sperm counts in young men exposed during breastfeeding during times when background dioxin concentrations were 10–20 times higher than today. They also raise concerns for areas that are currently undergoing rapid industrial development and potential related contamination with dioxin and other endocrine-disrupting chemicals.

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The 10 July 1976 Seveso disaster resulted in the highest dioxin exposure documented in humans and the largest cohort of dioxin-exposed women.