Let's face it, we're devoting enormous amounts of time and energy to minimize our exposures to toxins (think BPA, pesticides, and all the rest of the seemingly ubiquitous chemicals). But now an emerging body of research points to the disturbing possibility that such self-protective strategies might sometimes come decades, or even a century, too late.

If your great-grandmother experienced a brief toxic exposure, these studies suggest, you and your children could be at risk for reproductive illnesses and possibly other conditions. The presumed mechanism of this unfortunate inheritance is not a mutation in the DNA itself but rather changes in the biochemical on-off switches that determine whether or not specific genes get activated—a field of study known as epigenetics.

Most recently, researchers from Washington State University, led by biology...
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"...of the iceberg." o

The emerging science raises the stakes, with these recent effects scratching the surface about the developmental impacts of environmental contamination. "We're still just scratching the surface about the developmental effects of in utero exposures," Morello-Frosch said. "But this emerging science raises the stakes, with these recent studies suggesting that looking at the immediate offspring may be only looking at the tip of the iceberg."

Rachel Morello-Frosch, an epidemiologist and environmental health professor at the University of California-Berkeley, said these new findings of transgenerational effects push the boundaries of current understanding of the long-term consequences of environmental contamination. "We're still just looking at the tip of the iceberg."
It's no surprise, of course, that environmental factors, whether encountered in utero or early in life, can influence gene expression. Parental nutritional status, smoking, behavior and other factors as well as toxic exposures have all been shown to exert an impact on which genes get activated among offspring. In a 2003 experiment, a diet rich in B vitamins, which can promote DNA methylation, caused pregnant rats to give birth to normal pups; when rats did not receive the B vitamins, their pups had yellow skin and were more likely to suffer from obesity and diabetes. Other experiments have shown that rats whose mothers lick them in the first week after birth exhibit calmer reactions to stressful situations; the licking, apparently, causes epigenetic changes that lead to an increase in cellular receptors for critical steroid hormones known as glucocorticoids.

Of course, similar experiments are impossible to conduct on humans. However, the inadvertent human trial with the drug diethylstilbestrol, or DES, given to women from the 1940s through the 1960s in the belief that it would prevent some complications of pregnancy, provides a sobering example of multigenerational effects. DES caused a range of disorders in those exposed in utero—specifically, unusual vaginal cancers among women, and testicular abnormalities among men. Studies of the grandchildren of DES mothers are just starting to emerge; the National Cancer Institute notes that early research suggests that members of this generation might also suffer disproportionately from infertility, reproductive birth defects, and some cancers.