

A Measure of Community Exposure

PFOA in Well Water Correlates with Serum Levels

The first detailed investigation into contamination of private wells with perfluorooctanoic acid (PFOA) and levels of the compound in human blood serum suggests that drinking water was the dominant source of exposure to PFOA in a community industrially exposed to the compound [*EHP* 119(1):92–97; Hoffman et al.]. The study, conducted in 2005 and 2006, included only people who obtained their drinking water from private wells. The results showed that each 1- $\mu\text{g}/\text{L}$ increase of the compound in the participants' water supply was associated with a 141.5- $\mu\text{g}/\text{L}$ increase in people's serum PFOA concentrations.

The participants lived around DuPont's Washington Works facility in Parkersburg, West Virginia, where PFOA (also known as C8) is used in the manufacture of Teflon[®] nonstick polymers. PFOA has been shown to increase risk of cancer, reproductive problems, and liver damage in laboratory animals, although human health effects are less clear. Many of the water monitoring data used in this study were collected as part of an agreement between DuPont and the U.S. Environmental Protection Agency (EPA) to conduct a human health risk assessment for PFOA.

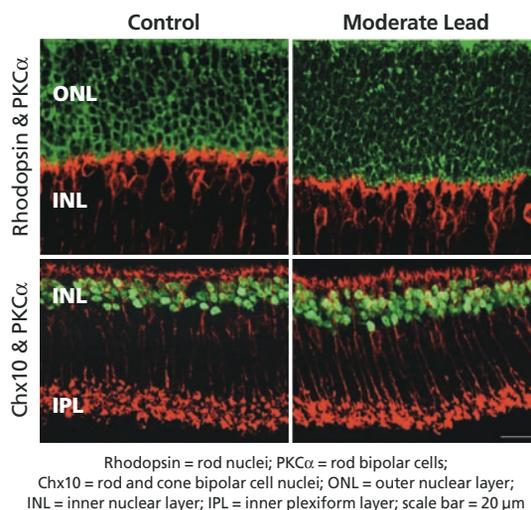
The groundwater in the Parkersburg area had been contaminated by DuPont's releases of PFOA into the nearby Ohio River. A second source of contamination was PFOA that was released into the atmosphere and deposited onto soils, which then leached into the groundwater.

Lead Doesn't Spare the Rod

Low-Level Exposure Supercharges Retinal Cell Production in Mice

Low-level gestational lead exposure has been shown to increase the electrical response of the rod signaling pathway in the retinas of children, monkeys, and rats, which could in turn contribute to retinal disease. Now researchers demonstrate the phenomenon underlying this effect: increased proliferation of retinal progenitor cells, which give rise to functionally differentiated retinal cells that sense and transmit visual information [*EHP* 119(1):71–77; Giddabasappa et al.].

Using a previously described mouse model of low-level gestational lead exposure, the researchers set out to test the hypothesis that such exposure selectively increases rod photoreceptors and bipolar cells in the rod signaling pathway. (The rod signaling pathway detects gradations of light, as opposed to the cone signaling pathways, which detect colors.) Female mice were given water containing varying concentrations of lead: 0 ppm (control), 27 ppm ("low" dose), 55 ppm ("moderate" dose), or 109 ppm ("high" dose). The exposures were administered for 2 weeks before mating, during pregnancy, and through postnatal day 10—a model for the human gestation period. On postnatal day 10, unspiked water replaced the water-lead mixtures for all groups.



Rhodopsin = rod nuclei; PKC α = rod bipolar cells;
Chx10 = rod and cone bipolar cell nuclei; ONL = outer nuclear layer;
INL = inner nuclear layer; IPL = inner plexiform layer; scale bar = 20 μm

The retina comprises several layers; among them, the ONL is composed of rod and cone nuclei, while the INL is composed of bipolar cells that transmit signals from the rods and cones to retinal nerve cells as well as numerous other cell types. Gestational lead exposure selectively increased the number of rods and bipolar cells.

Previous research in this study area linked drinking water supplied by six local water districts and consumption of home-grown vegetables to PFOA levels in participants' serum [*EHP* 118(8):1100–1108; Steenland et al.]. The new study provides a quantitative estimate of the relationship between drinking water and serum PFOA levels based on exposure to a wider range of PFOA levels in drinking water from 62 wells. It also corroborates the earlier finding about consumption of home-grown vegetables.

Many of the wells in the study had PFOA concentrations that exceeded the EPA's 0.4- $\mu\text{g}/\text{L}$ advisory level, although the median concentration in the well water samples was half that level. The concentrations of PFOA in participants' serum ranged from 0.9 to 4,751 $\mu\text{g}/\text{L}$, with a median of 75.7 $\mu\text{g}/\text{L}$, approximately 20 times the average level in the U.S. general population.

The association between PFOA in drinking water and serum was similar for both shorter- and longer-term residents of the area. The researchers found the associations held after excluding participants who reported drinking bottled water and those who worked at the DuPont facility. Compared with other factors (including age, sex, body weight, cigarette smoking, and alcohol consumption), drinking water was consistently the strongest predictor of serum PFOA levels.

The 141.5:1 ratio estimated for drinking water to serum PFOA concentrations is close to the 114:1 ratio predicted by a steady-state pharmacokinetic model employed by the authors. These findings may be useful in developing drinking water guidelines and studying other communities where PFOA is manufactured.

Kellyn S. Betts has written about environmental contaminants, hazards, and technology for solving environmental problems for publications including *EHP* and *Environmental Science & Technology* for more than a dozen years.

The adult mammalian retina consists of six types of neurons and a Müller glial cell. These cell types develop in one of two distinct phases: primarily *in utero* ("early-born") or primarily after birth ("late-born"). In examining controls and exposed mice at postnatal day 60, the researchers found that late-born rod photoreceptors and rod and cone bipolar cells increased by 16–30% in exposed offspring, whereas Müller glial cells (also classified as late-born retinal cells) did not increase. Low and moderate lead doses showed the greatest effects. Gestational lead exposure also increased and prolonged retinal progenitor cell proliferation but did not alter developmental apoptosis (programmed cell death), indicating that the higher numbers of rods and bipolar cells were due to increased production, not decreased apoptosis.

These results demonstrate that gestational lead exposure resulting in blood lead levels of 10 $\mu\text{g}/\text{dL}$ alters retinal development by selectively promoting the development of rod photoreceptor cells and bipolar cells. The authors speculate that the increased number of rods and bipolar cells in the lead-exposed animals could accelerate age-related retinal degeneration. These nonmonotonic dose–response results raise complex issues for neurotoxicology, risk assessment, public health, and children's health.

Angela Spivey writes from North Carolina about science, medicine, and higher education. She has written for *EHP* since 2001 and is a member of the National Association of Science Writers.