Methylmercury and IQ

Dose–Response Estimate of Prenatal Effect

Methylmercury, the most biologically active mercury compound, is well known to cause serious health effects, particularly to the developing fetal nervous system. Effects can include attention deficits as well as IQ, motor, memory, and language impairment. A new analysis now combines data from three earlier studies to produce an integrated estimate of the dose–response relationship between maternal mercury exposure during pregnancy and lowered childhood IQ [***EHP* 115:609–615; Axelrad et al.**].

Smart move. A new analysis takes a first step toward quantifying the benefits of reducing mercury exposure, which may include avoiding IQ deficits.

The authors analyzed combined IQ data from three longitudinal studies conducted in the Faroe Islands, the Seychelles Islands, and New Zealand. These studies measured a variety of neurodevelopmental end points, including IQ, attention, and motor skills. The range of prenatal exposures in the three populations is comparable to those of some U.S. populations. For example, a 2003 study found the lowest maternal blood mercury level in the Faroe Islands to be 0.53 µg/L, and the CDC reported in 2004 that more than half of U.S. women had blood mercury concentrations higher than this. Geometric mean blood concentrations in the United States from 1999 to 2002 were 0.92 µg/L for women of childbearing age; for children the mean was 0.33 µg/L.

The New Zealand and Seychelles studies reported results in terms of ppm of hair mercury, whereas the Faroe Islands study reported effects in terms of ppb of cord blood mercury. So the team converted the Faroe Islands results to their equivalents in units of hair mercury. They found a childhood IQ decrease of 0.18 points for each ppm rise in maternal hair mercury. The team assumed a linear, nonthreshold dose–response curve. However, they noted that if very low exposures produce a steeper curve, as has been found recently with childhood lead exposure, their calculation may underestimate the effects of prenatal mercury exposure. Similarly, certain cognitive abilities such as word retrieval and retention of verbally presented information are not captured by IQ scores, so relying only on IQ as a measure of cognitive function will also underestimate mercury's effects.

Eating fish is the most common route of human exposure to methylmercury. In 2004 the FDA and the EPA issued a joint statement advising women of childbearing age and children to limit their weekly consumption of commercially caught fish to 12 oz (6 oz for locally caught fish) in order to avoid harmful exposure. The EPA has set a reference dose of 0.1 µg/kg/day for methylmercury as an estimate of the daily exposure unlikely to cause harm over a lifetime.

Methylmercury's effect on IQ is separate from its effect on attention and motor skills. But because IQ is a well-established end point used in cost–benefit and economic analyses of the effects of environmental contaminants, establishing the dose–response relationship for IQ is a first step in quantifying the benefits of reducing mercury exposure. **–Valerie J. Brown**

Peril of the Shallows?

Elevated Arsenic in Kelp Supplements

Kelp, widely consumed in Asian countries, is a growing part of the U.S. supplement market. It generally is marketed as a concentrated source of iodine and other essential minerals. Because kelp is a nutritional supplement and not a drug, the FDA does not require manufacturers to demonstrate safety or efficacy. Now researchers at the University of California, Davis, report the case of a woman who received toxic doses of arsenic from kelp supplements [***EHP* 115:606–608; Amster et al.**].

Herbal loophole. As a dietary supplement, kelp is exempt from drug safety testing.

Arsenic occurs naturally in some soils, and can contaminate bodies of water. The metalloid concentrates in fish that eat arsenic-rich algae and can also be found in plants that absorb it from the soil or water in which they are grown. Human exposure typically comes from diet, contaminated drinking water, or occupational exposures, as in smelters; people ingest an average of 40 µg per day.

The researchers investigated kelp supplements after a 54-year-old woman taking the pills was referred to the university's occupational medicine clinic. The patient had started taking kelp to treat minor memory loss and fatigue. She initially took the dose recommended on the bottle, then doubled it when her symptoms failed to improve. She took kelp for one year, during which her fatigue worsened to the point that she had to switch from full- to part-time work. She also experienced rash, diarrhea, vomiting, severe headaches, and hair loss.

A urine test revealed an arsenic concentration of 83.6 µg/g creatinine. Creatinine, a muscle metabolite, is excreted at a relatively constant rate and is used for reporting urinary biomarkers, as an adjustment for the high variability of urine dilution. A normal arsenic concentration is less than 50 µg/g creatinine.

The researchers sent the patient's kelp supplement, along with several other brands purchased from local health food stores, to be tested at a state lab after ruling out occupational, dietary, and drinking-water exposure. Only one of the nine samples tested contained no detectable arsenic. Concentrations among the other eight ranged from 1.59 ppm to 65.5 ppm by dry weight. Samples taken from three batches of the patient's brand had concentrations of 1.59, 2.28, and 34.8 ppm. The FDA tolerance level for arsenic is 2 ppm.

Three weeks after she abandoned kelp, the woman resumed full-time work. Her urine arsenic concentration dropped more than a third in two months and was undetectable after another two months. Eventually all her syptoms resolved.

Past studies have shown that many herbal remedies are contaminated with potential toxicants including mercury and lead. To prevent more inadvertent poisonings, the authors recommend that manufacturers be required to prove safety before marketing their products. **–Cynthia Washam**

Top to bottom: Imagezoo/Images.com/Corbis; Chris Reuther/EHP

Door of Perception

NIEHS Portal Shows Way to Better Disaster Response

Hurricane Katrina—which killed 1,300 people, disrupted the lives of 650,000, and produced an estimated $125 billion in recovery and reconstruction costs—brought the need for better disaster response into sharp focus. In this issue, researchers introduce the NIEHS Portal, a state-of-the-art web-based system to improve decision making during disaster response [*EHP* 115:564–571; **Pezzoli et al.**].

The portal was designed to fulfill three objectives: to monitor disaster-related human and environmental health impacts; to assess and reduce pollutant exposures caused by disasters; and to develop science-based recovery strategies. The portal does this by combining geographically referenced data on roads, power plants, contaminant release sources, flood measures, and local demography in a cyber-infrastructure called "Telescience," which was developed at the University of California, San Diego. This cyberinfrastructure lets users share computer power and storage over the Internet.

A user-friendly interface provides access to project-relevant databases and data integration tools. High-speed network connections allow researchers to use supercomputing facilities and massive data storage sites as if they were on their desktop. A geographic information system (GIS) manages the data, and an accessible interface allows users to contribute new information and participate in online discussions and collaborative workspaces. Contributors are responsible for the accuracy of the data they provide. Access to data associated with any given research project is governed by the members of that research group.

SOS. Nineteen months after Katrina, Gulf Coast residents are still in the storm's shadow.

In its current deployment, the portal assembles GIS data for Texas, Louisiana, and Mississippi, and includes high-resolution data layers for the regions that were affected by Hurricanes Katrina and Rita in 2005. Scientists have begun exploring ways to use the system. One project supports ongoing efforts to study and mitigate the health effects of flood-induced indoor mold, particularly asthma among exposed children. Scientists link high mold concentrations with population information to identify locations of potential high exposures. The portal is also supporting studies of toxic sediments, particularly hot spots generated by the release of contaminants such as sewage and industrial chemicals during the storms.

The authors stress how the portal can also address the research needs of exposure biology and gene–environment interactions. The system's massive computing power can integrate population-level studies of genetics with real-time exposure monitoring and environmental sampling, advancing the NIEHS's goal of studying molecular processes in environmental health. **–Charles W. Schmidt**

A Modified Effect on Asthma

Ozone and Secondhand Smoke Outweigh Genetic Influence

Individual variations in genes, known as single-nucleotide polymorphisms (SNPs), help to explain why some children are more susceptible to asthma and allergies. But does exposure to ozone or secondhand smoke alter this genetic susceptibility? Public health experts from Mexico and the United States report that, in 596 families with asthmatic children living in Mexico City, where ozone levels rank as the highest in North America, parental smoking can indeed modify the risk conferred by a particular SNP [*EHP* 115:616–622; **Wu et al.**].

Smoke screen? For certain people, secondhand smoke exposure may override genetic influence on asthma risk.

Top to bottom: Laurie Barr/Shutterstock; Elena Kouptsova -Vasic/Shutterstock

Complex interactions among genes and environmental triggers are known to contribute to asthma and allergic reactions in children. Exposure to ozone, for instance, turns on the *TNF* gene for the production of tumor necrosis factor-α, a cytokine that causes airway inflammation. So does exposure to cigarette smoke.

The children, who ranged in age from 4 to 17 years, largely had mild asthma. Half lived with a smoking parent. The researchers measured variations in *TNF* and the gene for lymphotoxin-α (*LTA*) in the children and their parents. *TNF* and *LTA* lie next to each other on chromosome 6 and share receptors. Two SNPs for *LTA* and four for *TNF* capture most of the variation in these two genes.

The team found that *LTA* was not associated with asthma risk, but one SNP for *TNF* (coded 308A) raised the risk by 50% among all children. This SNP and one other (238A) more than doubled the risk of asthma among children living with nonsmoking parents. None of the SNPs for *LTA* or *TNF* were linked to asthma among children living with smoking parents. In addition, allergic reactions generated with skin prick tests were not related to any of the SNPs tested.

The researchers suspect that secondhand smoke and ozone may synergistically increase production of tumor necrosis factor-α, an effect that overrides the minor influence of genetic variation. Therefore, the effects of certain SNPs on risk of asthma may stand out more clearly in children who are not exposed to secondhand smoke. **–Carol Potera**