

## Mass Lead Poisoning in Dakar Battery Recycling Exact a Heavy Toll

In a neighborhood of Dakar, Senegal, 18 children died from an aggressive central nervous system disease between November 2007 and March 2008. Experts from the World Health Organization and local health authorities were called in to investigate the deaths, but cultural prohibitions preempted autopsies of the children. So the researchers examined 32 of the children's siblings and 23 of the siblings' mothers along with 18 unrelated local children and 8 unrelated adults. They concluded that the cause of death likely was encephalopathy resulting from severe lead poisoning [EHP 117:1535–1540; Haefliger et al.]. The source of the lead, in turn, was determined to be contamination resulting from the reclamation of used lead-acid batteries, a lucrative business in developing countries that often is performed in the open with few pollution controls.

Since 1995, local people had broken apart batteries from vehicles and appliances and sorted the components in an open sandy area of the neighborhood. They sifted through the sand for scraps of valuable lead to sell, even carrying sacks of contaminated sand into their homes. People were probably exposed by inhaling and ingesting lead dust, with children particularly exposed through hand-to-mouth activity and eating the contaminated soil.

The developing nervous system of children is particularly vulnerable to the toxic effects of lead. Blood lead concentrations as low as

The father of a fatally lead-poisoned Dakar child holds lead extracted from a spent car battery, September 2008



10 µg/dL are known to impair neurologic development, resulting in permanent intellectual impairment. However, recent evidence suggests there may be no safe threshold of exposure.

Among the 50 children tested, blood lead levels ranged from 39.8 to 613.9 µg/dL. Seventeen of the 50 children showed neuropsychiatric symptoms including convulsions, irritability, and aggression, and 21 showed gastrointestinal symptoms such as anorexia and vomiting. Adult blood lead levels ranged from 32.5 to 98.9 µg/dL, and their most commonly reported symptom was gastrointestinal upset.

Recycling activity reportedly ended by March 2008 following a public awareness campaign, and the neighborhood soil was partially remediated. Nevertheless, lead concentrations measured in the sandy work area after this time still reached 209,000 mg/kg, and levels inside homes reached 14,000 mg/kg. The U. S. Department of Housing and Urban Development sets 400 mg/kg as for the standard for lead in bare soil in children's play areas (there is no comparable standard in Senegal).

The lead poisoning in this study was severe enough to catch the attention of health experts, but the global incidence of lead poisoning from battery recycling is unknown. The authors believe many cases go unaddressed in developing countries because local authorities lack resources to recognize, diagnose, and manage lead toxicity. However, they write, lead poisoning can be prevented through measures such as public education and the implementation and enforcement of lead recycling guidelines.

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## Variable Vulnerability Genotype Determines Timing of PON1 Capability to Detoxify Pesticides

Infants are extremely vulnerable to certain pesticide exposures because they are still developing the ability to produce the enzyme paraoxonase-1 (PON1), which detoxifies certain organophosphate pesticides such as chlorpyrifos and diazinon. A study published in 2003 indicated that children may reach near-adult levels of PON1 activity by age 2 years. But a new larger-scale study of participants in the Center for Health Assessment in Mothers and Children of Salinas cohort has found that many children are still ramping up PON1 levels until at least age 7 and that PON1 activity can vary dramatically among children of the same age [EHP 117:1632–1638; Huen et al.].

Organophosphates have been largely banned from home use but are still widely used in agriculture. These pesticides target the nervous systems of insects and also affect the human nervous system. Prior work by researchers involved in the current study revealed associations between prenatal exposure to organophosphate pesticides and increased reports of developmental delays and disorders in children.

Genetic variation in the *PON1* gene affects the type and quantity of the enzyme produced. A single-nucleotide polymorphism at position 192 of the gene's coding region changes the enzyme's configuration, affecting its overall and pesticide-specific efficiency; the R allele (form) of the PON1<sub>192</sub> polymorphism is more efficient than the Q allele at detoxifying organophosphate metabolites. A single-nucleotide polymorphism

at position –108 of the gene's promoter region affects the amount of PON1 produced, such that persons with the C allele have higher levels of PON1 activity than persons with the T allele.

In the current study, researchers measured PON1 activity from birth to age 7 years in 458 Mexican-American children living in a heavily agricultural area. Blood samples were taken at up to five time points (birth, 1 year, 2 years, 5 years, and 7 years) and analyzed using three assays that measured PON1 quantity and efficiency. Blood samples were also genotyped for *PON1* variations. Researchers obtained a total of 1,143 samples, with 108 children providing samples at four or more time points.

Enzyme activity increased with age in all the children. However, compared with other children, PON1 activity was higher at birth and increased more quickly with age in children with the RR *PON1*<sub>192</sub> genotype or the CC *PON1*<sub>–108</sub> genotype (found in 24% and 28% of the children, respectively), such that in some cases average levels of activity against some pesticide metabolites were lower in children at age 7 than they were in other children at age 2. Children with both the RR and CC genotypes had the highest PON1 activity levels, and children with both the QQ and TT genotypes had the lowest levels.

These results suggest that some children may be vulnerable to the effects of organophosphate pesticides for longer than previously thought. The authors therefore recommend that policy makers consider this new information to ensure standards for pesticide exposures adequately protect young children.

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## Disinfection By-Products and Congenital Anomalies Evidence Still Inconclusive

Many observational studies have investigated a possible association between maternal exposure to mutagenic disinfection by-products (DBPs) in the water supply and congenital anomalies in offspring, but literature reviews to date have shown the evidence to be inconclusive. Now researchers have reviewed newer epidemiologic studies that include more categories of anomalies, but again have found the evidence inconsistent [EHP 117:1486–1493; Nieuwenhuijsen et al.]. They suggest several guidelines that would help future studies clarify this issue.

The authors reviewed all published epidemiologic studies that examined the association between congenital anomalies and exposure to DBPs, which form when organic matter in treated water reacts with chlorine disinfectant. The studies used a variety of indices of exposure, including the use of chlorination, DBP measurements in the public water supply, and information from participants about activities such as drinking, showering, and bathing.

When three or more studies evaluated the same exposure index and the same congenital anomalies, the authors performed a meta-analysis to derive a summary risk estimate comparing high- and low-exposure groups. When five or more studies investigated the relationship between total trihalomethane (TTHM) concentration and a specific anomaly, the authors conducted a meta-analysis to arrive at exposure–response relative risk estimates per 10 µg/L TTHM.

## PBDEs in Diet Meat Fat a Leading Source

Although researchers have long known polybrominated diphenyl ethers (PBDEs) appear in foods such as beef, chicken, fish, and milk, the results of a new study are the first to link blood levels of prevalent PBDEs with food intake over a wide population [EHP 117:1520–1525; Fraser et al.]. Researchers from the Boston University School of Public Health examined data from the 2003–2004 National Health and Nutrition Examination Survey (NHANES) and found that people who reported eating the most poultry and beef had higher average serum PBDE levels than people who ate lower amounts of these meats.

PBDEs are used as flame retardants for electronics, fabrics, packing materials, and other products. Some of the formulations that trigger the most concern—for example, penta-BDE, which contains BDE-28, -47, -99, -100, and -153—are no longer produced in Europe and the United States, but persistent PBDEs remain in such items as older electronics and upholstered furniture, and often turn up in household dust. Past surveys have found high levels of these compounds in human tissues. Fat-soluble and persistent, PBDEs cause numerous adverse health effects in experimental animals and are suspected endocrine disruptors in humans.

The study included NHANES participants for whom there were serum measurements of up to 10 PBDE congeners as well as information on what the participants had

TTHM concentrations in local water sources have often been used in epidemiologic studies as a proxy for DBP exposure.

These meta-analyses provide little evidence for an association. For all congenital anomalies combined, a statistically significant excess risk was found for high versus low exposure to chlorinated water or to TTHM, but the analysis was based on a small number of studies. The authors also found a statistically significant excess risk for ventricular septal defects, but that analysis included only three studies, and there was little evidence of an exposure–response relationship. In addition, the meta-analyses were weakened by the fact that studies used very different exposure criteria. For instance, among studies that measured TTHM, one defined low exposure as lower than 60 µg per L water, whereas another defined high exposure as higher than 20 µg/L.

The authors point to the need for studies that take into account the complex mixtures of DBPs to which people are exposed, which can vary over time, by geographical area, and by route of exposure. This includes conducting studies in places with water supplies that have large amounts of specific DBPs, such as Barcelona, Spain, or Perth, Australia, which have high levels of brominated DBPs. Furthermore, showering, bathing, and swimming activities should be examined in more detail because these activities may yield different levels of exposure compared with drinking water exposure. The review also calls for studies that focus on specific congenital anomalies for which there is likely to be complete case diagnosis and reporting, in addition to case–control studies that follow up on possible associations with ventricular septal defects, which are particularly prone to under-reporting in registry-based studies.

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eaten the day before the interview and their usual diet over the past year. The researchers compared the diet survey information with the results of the blood sample analyses, which had revealed detectable levels of five PBDE congeners in more than 60% of the study population.

The researchers found that, overall, vegetarians had serum PBDE concentrations approximately 25% lower than those of omnivores. The people most likely to have the highest total PBDE concentrations were young and male. Levels of all five congeners were significantly associated with high poultry fat intake. All five congeners also were associated with high intake of red meat fat, with statistically significant associations for BDE-100, BDE-153, and total serum PBDEs. However, the team found no significant associations between serum

PBDE concentrations and high dairy or fish consumption—even though these foods also contain measurable levels of PBDEs. The authors hypothesize that although both farmed and wild fish can contain high levels of PBDEs, Americans may not consume enough fish to make this a significant source of exposure.

Next steps should include measuring food intake against BDE-209, which is not yet included in NHANES data, and direct comparisons with exposure via dust. The authors write that researchers will also have to watch for shifts in humans' exposure—old products will remain as reservoirs for further exposure even as manufacturers move toward alternative flame retardants, which themselves may have unexpected toxic effects.

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