Massive Childhood Lead Poisoning

The Price of Nigerian Gold

Childhood lead poisoning on a scale unheard of for decades has been detected in rural northwestern Nigeria [***EHP* 120(4):601–607; Dooyema et al.**]. The culprit: lead in gold ore processed using artisanal techniques. Chelation therapy for hundreds of children, soil replacement, and an education campaign to discourage processing ore inside homes may now have radically reduced child mortality in the hardest-hit villages, but the long-term effect of lead poisoning on the surviving children remains to be seen.

The outbreak surfaced in the spring of 2010 when health professionals noticed abnormally high rates of child illness and death among young children in 4 villages of Zamfara State. Blood tests on 8 children returned blood lead levels (BLLs) of 168–370 mg/dL, at least 16 times the level of concern set by the U.S. Centers for Disease Control and Prevention (CDC). The Nigerian authorities quickly assembled an international team to identify the source of the exposure and to respond, focusing on the 2 worst-affected villages.

Blood samples were collected from 59% of children under age 5. Of these, 97% had BLLs of at least 45 mg/dL, the threshold at which the CDC recommends chelation therapy. The BLLs of 85% surpassed the portable sampling devices' maximum detection limit of 65 mg/dL.

A survey of the villagers revealed that 25% of all children under age 5 had died in the previous year, most of them in the half-year before the study. This translates to a mortality rate of 255/1,000 live births, compared with a national average of 157/1,000. The problem was the lead-contaminated gold ore being processed in many of the family compounds. Two-thirds of these families had started the activity within the last year.

A child awaits treatment for lead poisoning in 2010. Nearly all children tested in the worst-affected villages had blood lead levels high enough to warrant chelation therapy.

Soil samples were collected from nearly all the family compounds where processing occurred, with 85% showing heavy lead contamination. The worst reached 250 times the U.S. Environmental Protection Agency safety limit of 400 ppm for play areas. Similarly, water lead concentrations far exceeded U.S. recommendations.

Not every child's blood could be tested, and a lack of medical data for the deceased meant their deaths could not be definitively linked to lead poisoning. Further, the locally recruited survey staff had limited training in administering questionnaires and collecting environmental samples, which may have affected the results. Nonetheless, the evidence clearly suggests these villages were hit by lead poisoning due to artisanal processing of contaminated gold ore.

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Brain Tax

Estimating the Population-Level Impact of Environmental Chemicals on IQ Scores

Many studies report the impact of various environmental chemicals on children's neurological development as "clinically insignificant." But this designation, which describes the individual child, does not always reflect the broader implications of exposure for the overall population—i.e., that a mild but frequent impact could add up to a substantial population-level burden. A new analysis establishes priorities for reducing adverse neurodevelopmental impacts for U.S. children aged 5 years and younger [***EHP* 120(4):501–507; Bellinger**].

The author assessed the population impact of various risk factors, including exposure to environmental chemicals, on Full-Scale IQ (FSIQ) scores. FSIQ values are a useful measure for such an assessment because past studies have shown that these scores correspond to work productivity and associated lifetime earnings. FSIQ declines therefore can be used to estimate economic consequences for various risk factors.

The data necessary for such an analysis, including information on both prevalence of exposure and effects, were available for only 3 environmental chemicals: lead, methylmercury, and organophosphate pesticides. The loss in FSIQ points was also calculated for a variety of medical conditions, neurodevelopmental disorders, traumatic brain injury, failure to thrive as a result of neglect and abuse, and iron deficiency. Loss of IQ points attributable to each risk factor was estimated based on meta-analyses or pooled analyses of existing data, then generalized to the 25.5 million children aged 0–5 years estimated to live in the United States, based on the prevalence of exposure to each risk factor.

Preterm birth resulted in the largest population health burden, with estimated losses of more than 34 million IQ points populationwide, followed by lead exposure at 22.9 million points lost. Organophosphate pesticides and attention deficit/hyperactivity disorder both resulted in estimated losses of more than 16 million points, iron deficiency in 9.4 million points, pediatric bipolar disorder in 8 million points, autism spectrum disorders in 7 million points, brain injury in 5.8 million points, and failure to thrive in 5.3 million points. The remaining risk factors resulted in estimated cumulative IQ losses of fewer than 1 million points each. Interactions between factors certainly exist, although the current study was not able to assess these.

A surprising finding was that a large fraction of the total estimated IQ loss associated with lead was contributed by children in the lower reaches of the blood lead distribution—that's because so many children have low levels of exposure. But the author estimates that reductions in U.S. lead exposure nevertheless "saved" more than 100 million IQ points between the 1976–1980 and 2005-2006 iterations of the National Health and Nutrition Examination Survey. This suggests that similar reductions in nations where lead exposures remain high could result in dramatic improvements in neurodevelopment on a population level.

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