letter noting the absence of *public* involvement in this process makes 2 relevant but, in my opinion, inadequate points: (1) the public had the opportunity to react to drafts of the code on the American Public Health Association Web site and (2) feedback was solicited "from a broad range of stakeholders, including the public."

The response does not mention even one non–health professional person or organization that provided feedback or was specifically solicited for feedback. It also does not explain why there was no non–health professional ("public") person on the drafting committee. Sometime soon, I hope, this committee will enrich the code with the experiences and views of the *public* regarding the ethical aspects of public health practice.

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WHAT LEVEL OF LEAD IN BLOOD IS TOXIC FOR A CHILD?

Bernard states that current knowledge does not warrant lowering the Centers for Disease Control and Prevention's (CDC's) definition of pediatric lead intoxication below the current level of 10 μg/dL.[1] Bernard cites, in support, economic considerations, inadequate health risk data, and limited options for intervention.

As investigators of lead toxicity and pediatricians who have treated poisoned children, our position is that only health-based criteria are acceptable for setting a health standard. Cost–benefit analyses and policy issues are peripheral and subordinate to the central question: What level of lead in blood is toxic for a child?

Over the past century, as knowledge of lead toxicity has evolved, levels of lead in blood once considered safe have been found not to be. Governmental authorities have responded by lowering the definition of pediatric lead poisoning.

Only 100 years ago, when childhood lead poisoning was first described, physicians doubted the very existence of the disease. After the reality of pediatric lead poisoning was accepted, the received doctrine was that there were only 2 outcomes: death or complete recovery. The first follow-up study of children who had "recovered" from lead poisoning showed that almost all had severe learning difficulties or behavior disorders. Only children who displayed signs of encephalopathy were then thought to show residual brain damage. In the 1970s studies showed that blood lead levels too low to evoke symptoms produced IQ deficits, attentional dysfunction, and slowed growth.

Consequently, the definition of lead toxicity was lowered by the CDC, from 60 μg/dL in the 1960s to 10 μg/dL in 1991. Two factors brought about this reduction: improved investigational strategies and reduced background lead levels due to the removal of lead from gasoline. The mean blood lead level in this country in 1975 was 15.5 μg/dL. It is now 2 μg/dL, permitting contrasts with subjects with lead levels of 1 μg/dL.

Three studies now show that lead can cause IQ deficits in children at levels below 10 μg/dL.[2–5] Further, the slope of the IQ/lead regression in these studies is steeper at levels below 5 μg/dL than at higher levels. The meaning of this surprising finding (found in all 3 studies) is clear: a large part of the damage occurs at the lowest doses. Only partisans of the lead industry quarrel with these data.

To protect America's children, we must again lower the officially defined standard to conform to only the best science. Policy matters must adjust to the facts.

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HEALTH EFFECTS OF BLOOD LEAD LEVELS LOWER THAN 10 MG/DL IN CHILDREN

The thoughtful commentary provided by Bernard[1] is a welcome addition to deliberations about whether the Centers for Disease Control and Prevention (CDC) should respond to recent reports of adverse effects of blood lead levels (BLLs) lower than 10 μg/dL in children by lowering the BLL at which individual intervention is recommended.[2,3] The CDC's Advisory Committee on Childhood Lead Poisoning Prevention is reviewing the scientific evidence of the health effects of BLLs lower than 10 μg/dL in children. A finding of adverse effects across a large number of studies will raise important questions about what changes, if any, the CDC should make in its recommendations for medical and environmental management of individual cases. Several suggested changes, including Bernard's suggestion that very young children with BLLs above the national average for young children be tested more frequently, deserve further consideration.

Bernard also advocates widespread education about the dangers of lead, the use of blood lead surveillance and other data (such as housing data) to identify populations at risk, and improved screening of children enrolled in Medicaid. We concur with these recommendations and have asked state and local programs funded by the CDC to work aggressively in these areas. We also agree

that control or elimination of lead hazards is essential in "repeat offender" housing where children with elevated BLLs have repeatedly been identified.

Also relevant to these considerations is the lack of effective interventions to lower elevated BLLs.[4,5] Taken together with the recent reports of health effects of BLLs lower than 10 µg/dL, these studies suggest that elimination of childhood lead exposure requires the implementation of creative strategies for primary prevention. However, shifting our focus to primary prevention does not require changing the intervention level or preclude using this level as one tool for identifying populations of children at highest risk. In fact, it is extremely important that we continue to focus our efforts on those populations. Moreover, we believe that primary prevention efforts, including effective partnerships with housing and other agencies to direct scarce abatement and prevention resources to high-risk neighborhoods, should be our highest priority. Emphasizing primary prevention is the only way we can achieve the nation's 2010 health objective of eliminating childhood lead poisoning as a public health problem.[6] ■

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