

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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BERNARD RESPONDS

I appreciate the comments by Needleman and Landrigan and by Brown and Meehan, and I thank them for their valuable contributions to the field of childhood lead poisoning prevention.

Needleman and Landrigan describe recent research identifying adverse effects of lead exposure resulting in childhood blood lead levels (BLLs) below 10 µg/dL. This research is also noted in my article, in which I stressed the importance of continuing epidemiological and toxicological studies in this area. I also set forth there the history of the Centers for Disease Control and Prevention’s (CDC’s) intervention level over the 20th century.

The question I addressed was not what level of lead in blood is toxic to a child, but whether lowering the CDC’s blood lead screening and intervention level to 5 µg/dL would result in a reduction of risk to children now and in the future. Needleman and Landrigan do not identify any interventions they would recommend implementing for children with BLLs between 5 µg/dL and the current intervention level of 10 µg/dL. My analysis suggests that the public health goals of primary and secondary prevention would best be served by universal lead exposure risk screening by means of a validated questionnaire, universal education of parents and guardians about lead exposure risks, and invigorated implementation and enforcement of targeted screening efforts and requirements. Renewed emphasis on these measures would help protect the health of all lead-exposed children, regardless of their BLLs.

I am encouraged that Brown and Meehan concur in these recommendations. I also appreciate the CDC’s willingness to consider my recommendation that the time frame for repeat screening of infants with BLLs greater than or equal to 5 µg/dL be shortened; the current screening guidelines provide for only annual screening, which may be inadequate to prevent such children from becoming toddlers with BLLs of 10 µg/dL or higher.

As Brown and Meehan note, the lack of effective interventions to lower elevated BLLs and the recent studies showing adverse effects at BLLs lower than 10 µg/dL suggest that an emphasis on primary prevention by the CDC and other federal and state entities is warranted. In this spirit, I recommended that children be protected from lead exposure through regulatory health-based standards set with the goal of preventing childhood BLLs even below those for which adverse health impacts currently can be quantified. ■

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