more general categories of behavior frequency (e.g., 100% vs 0% use). Indeed, the amount of behavioral-measurement precision that is needed for determining if a person has a nonzero risk for HIV or other STDs is probably not great.

On another issue, in the context of cross-sectional vs longitudinal designs, Dent and Cohen reach a conclusion about self-reported behavior and "objective" assessments that is somewhat confusing. Cohen and Dent's design of "self-reported cross-sectional" and "objective longitudinal" is confounded. To examine the issues they raise one would need a design that examined self-reported condom use and STD reports in both cross-sectional and longitudinal designs. □

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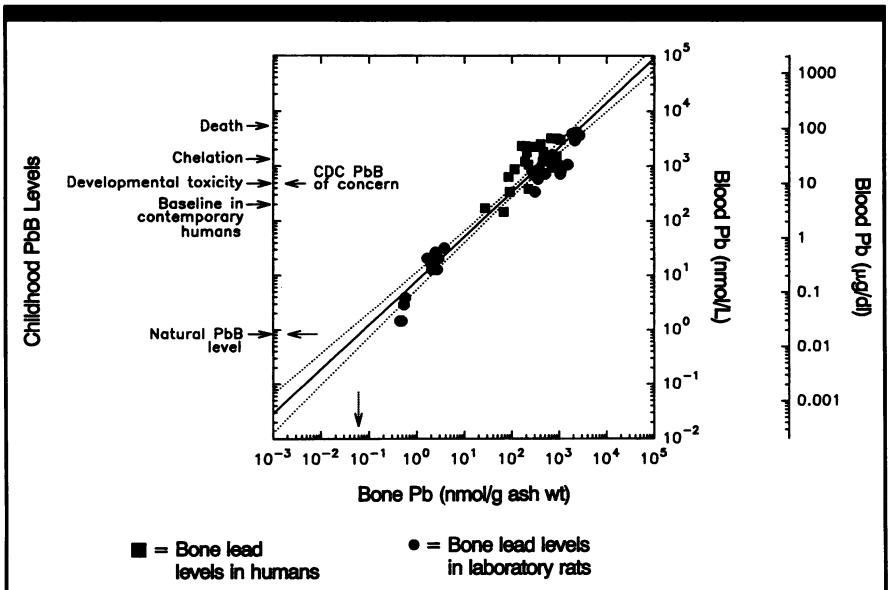
The Public Health Implications of Humans' Natural Levels of Lead

We have recently estimated the natural levels of lead in human blood to be 0.8 nmol/L (0.016 μg/dL)[1] (Figure 1). This has immense public health implications because it indicates that blood lead (PbB) concentrations of contemporary humans are 200 to more than 1000 times above natural levels. Of particular significance is the magnitude of this new estimate relative to the Centers for Disease Control's proposed PbB level of concern for early toxic effects in children (480 nmol/L or 10 μg/dL).[2] As shown in Figure 1, the PbB levels that are now considered acceptable in children (< 480 nmol/L or < 10 μg/dL) are nearly 600 times greater than estimated natural levels, and they are only about 10 times lower than levels (approximately 4800 nmol/L or 100 μg/dL) associated with encephalopathy and death.

The clinical significance of the relationship between natural and contemporary PbB levels is substantiated by recent studies that have indicated there may be no threshold for lead toxicity in humans.[3–7] While those and other studies have contributed to defining subclinical lead toxicity, that definition has existed on a relative, rapidly changing scale. This is evidenced by the sixfold reduction in childhood PbB action levels over the past several decades (Figure 2).

We believe that the recent CDC proposal of 480 nmol/L (10 μg/dL) as the PbB level of concern for early toxic effects in children will result in a marked improvement in the effective identification and treatment of subclinical childhood lead poisoning. However, it may still fall short of providing a definitive basis for protecting the nation's children from this disease.



Note. The natural lead concentration in blood of humans (0.8 nmol/L or 0.016 μg/dL, dashed arrow on the ordinate) was derived from the reported concentrations of lead in preindustrial human bone (dashed arrow on the abscissa) by graphical extrapolation of the relationship between paired bone- and blood-lead concentrations of humans (squares) and laboratory rats (circles). See Flegal and Smith[1] for details. The perspective on childhood PbB levels was adopted with modification from CDC.[2]

FIGURE 1—Perspective on childhood lead levels.

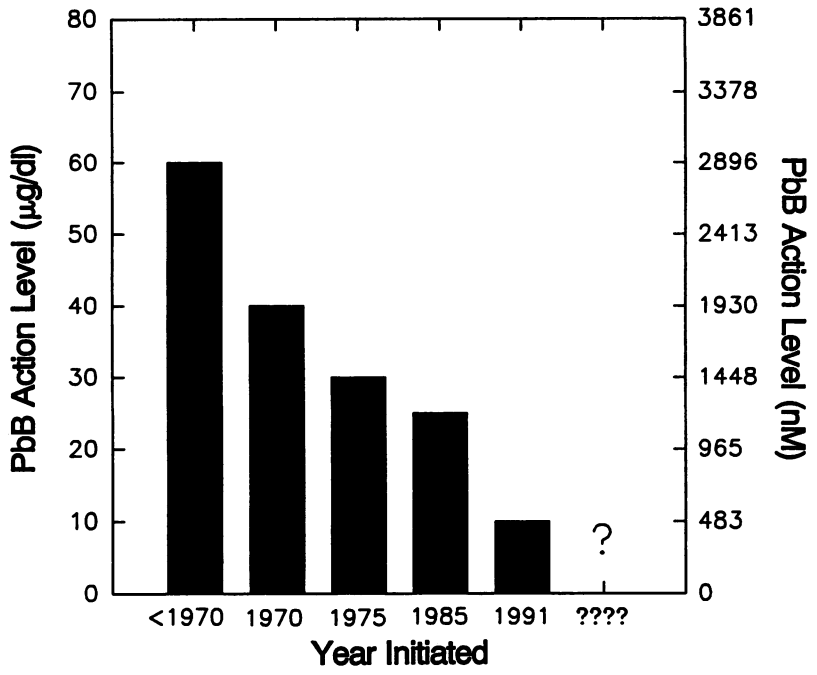


FIGURE 2—Blood lead (PbB) action levels proposed by the Centers for Disease Control and the Public Health Service[2].

This is based on our estimate of the natural PbB concentration in humans and on recent studies that have documented neurobehavioral and cognitive deficits in infants with PbB levels as low as 340 nmol/L (7 μg/dL).[3,5]

Our estimate also indicates that current studies may be insensitive in detecting the pervasiveness of low-level lead toxicity, because their control groups are exposed to elevated environmental levels of lead that result in "baseline" PbB concentrations that are 200 to 600 times above natural levels. These order-of-magnitude increases in environmental and body lead levels have occurred throughout the terrestrial biosphere due to industrialization, and they have confounded work to determine subclinical toxicity thresholds for lead. This is acknowledged in the CDC statement,[2] which points out that limitations in the ability to detect low-level lead poisoning may thwart establishing lower PbB action levels. Therefore, public health agencies should now focus on the development of strategies to achieve body lead burdens that more closely approach natural lead concentrations. □

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Community Health and Odor Pollution Regulation

I read with great interest the report by Haahtela et al. on community health complaints after an episodic release of hydrogen sulfide (H_2S) and mesityl oxide from a pulp mill.[1] The authors documented symptoms at H_2S exposure levels that, although exceeding the World Health Organization's (WHO) half-hour guideline of 7 μg/m^3 (0.005 ppm) for "odor nuisance," were below the WHO's 24-hour guideline of 150 μg/m^3 (0.11 ppm) for "health hazards."[2] As I will argue, the patterns of H_2S-related symptom reporting should lead one to question the distinction between "odor nuisance" and "health hazard."

Haahtela et al. point out that the toxicology literature does not lead one to expect irritative effects from H_2S below approximately 15 mg/m^3 (10 ppm).[3] My colleagues and I previously reported on a cluster of odor and symptom complaints that occurred in a community downwind of a hazardous-waste site, following the release of reduced sulfur gases (including H_2S) from settling ponds.[4] Like Haahtela et al.'s, our investigation documented subjectively reported headaches, upper respiratory tract irritation, eye irritation, and nausea among community complainers. However, in contrast to the Haahtela et al. study, although the exposure levels associated with acute and reversible symptoms in our study were linked with odors, they were below levels of detection by sensitive air monitoring techniques. (Levels of detection were 0.0001 ppm [0.14 μg/m^3] for H_2S, and 0.00002 ppm for various mercaptans.) The similarity of symptoms reported after exposures to reduced sulfur compounds at "high" (0.025 to 0.1 ppm or 35 to 135 μg/m^3) and "low" (below 0.0001 ppm) levels gives rise to speculation regarding the role of odor perception per se as a mediator of symptoms. This possibility has been explored elsewhere in a meta-analysis of symptoms near hazardous waste sites[5] and in a review of community health studies near industrial, hazardous waste, and agricultural odor sources.[6]

Complicating the picture described by Haahtela et al. was the issue of coexposure to mesityl oxide, which was alluded to but not dealt with extensively in their article. The "odor safety factor" (or ratio between irritant and odorant thresholds) is lower for mesityl oxide (15 ppm