Hypertension and Low-Level Lead Exposure: A Scientific Issue or a Matter of Faith?

To the Editor:

Vupputuri et al.1 analyzed the NHANES III data. They reported that among blacks, a 3.3 μg/dL increment in the blood lead concentration was associated with an increase in systolic pressure averaging 0.82 mm Hg in men and 1.55 mm Hg in women. In contrast, blood pressure was not associated with the blood lead level among white men and women.

We analyzed the same NHANES III database.2 In line with Vupputuri’s article, we also found a significant and positive relationship between systolic blood pressure and the blood lead concentration in blacks. On the other hand, our analyses revealed significant and negative associations between diastolic pressure and blood lead in whites. We concluded that, across the NHANES III race and sex strata, the relationship between blood pressure and lead exposure was not consistent and should probably be attributed to residual confounding rather than to causation. Vupputuri et al emphasized the positive results and disregarded alternative interpretation of their findings.

A recent meta-analysis of 31 studies revealed only a weak association between systolic blood pressure and blood lead, which is probably not causal in nature.3 Overall, a twofold increase in the blood lead concentration was associated with a 1.0 mm Hg rise in systolic pressure (95% CI: +0.5 to +1.4; P<0.001) and with a 0.6 mm Hg increase in diastolic pressure (95% CI: +0.4 to +0.8; P<0.001).

Although Vupputuri’s findings were confined to blacks, the authors speculated that reducing the environmental exposure to lead might lead to a population-wide decrease in diastolic pressure of 2 mm Hg, which would result in a 17% reduction in the prevalence of hypertension and a 15% decrease in the risk of stroke. Our meta-analysis suggests that, if the relationship between blood pressure and low-level lead exposure would be causal and reversible, for which there is no evidence, the estimates of benefit in Vupputuri’s extrapolations are highly speculative.

The possible role of low-level environmental exposure to lead in the pathogenesis of hypertension is a fascinating hypothesis with many uncertainties. The matter is not served by selective presentation or interpretation of data.

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