

October 15, 1984

In Reply Refer To: 561/15

David E. Weil, Ph.D.
U.S. Environmental Protection Agency
Environmental Criteria & Assessment
Office (MD-52)
Research Triangle Park, North Carolina 27711

Dear Dr. Weil:

Thank you for the opportunity to address the plausibility that blood lead correlates with blood pressure and that hypertension correlates with stroke and heart attacks as suggested in the abstract by Pirkle et al (1) and the memorandum prepared by Joel Schwartz on "Blood lead and blood pressure" for Docket EN-84-05.

While I am unable to evaluate the statistical models used for the analyses, I believe the conclusion that "the relationship of systolic and diastolic blood pressure to blood lead levels was statistically significant", is entirely consistent with accumulated evidence on the role of lead in cardiovascular disease. Our own studies indicate that unrecognized lead absorption is an important contributor to hypertensive renal disease in subjects usually considered to have "essential" hypertension (2). While the patients we studied had considerably greater exposure to lead than was encountered in the NHANES II population, their excessive body lead stores were superimposed upon a background of lead absorption which rendered them vulnerable to the severe medical complications. Although our study focused on hypertensives with renal failure, it is clear that there must be another population of lead induced hypertensives without renal failure who will develop renal failure later in the course of their disease. Moreover, present knowledge indicates that hypertension represents the upper end of continuum of blood pressure levels in the general population. There is every reason to believe that this continuum extends down to blood pressure levels presently considered to be in the "normal" or "safe" range. ~~It is apparently the impact of environmental lead in this group that has been identified in the NHANES II analysis presented by Pirkle et al.~~

Once blood pressure rises to abnormal levels, all the complications of hypertensive disease can be anticipated including stroke and heart disease. It is noteworthy that the cost analysis presented by Joel Schwartz omits any mention of the contribution of lead to the End-Stage Renal Disease (ESRD) program. Chronic dialysis and transplant programs presently cost the United States government over two billion dollars a year. While the contribution of lead nephropathy to this financial burden has not been precisely defined, lead may be responsible for as much as half of those

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patients presently believed to require ESRD care because of hypertensive nephrosclerosis.

References:

1. Pirkle JL, Schwartz JH, Landis JR, & Harlon WR. The relationship between bloodlead levels and blood pressure and its cardiovascular risk implications. Am. J. Epidemiology. In press.

2. Batuman V, Landy E, Maesaka JK, & Wedeen RP. Contribution of lead to hypertension with renal impairment. N. Engl. J. Med. 309:17-21, 1983.

Sincerely,


RICHARD P. WEDEEN, M.D.
Professor of Medicine
UMDNJ/New Jersey Medical School
Associate Chief of Staff for
Research & Development

When serum creatinine levels become available in the NHANES II data, it would be of great interest to test for correlations with both BP and B_{Pb} . It may be possible to support the hypothesis that lead mediates blood pressure via effects on the renal vasculature. The correlation with creatinine if present supports the view of mechanism, if absent does not effect the present conclusions.

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