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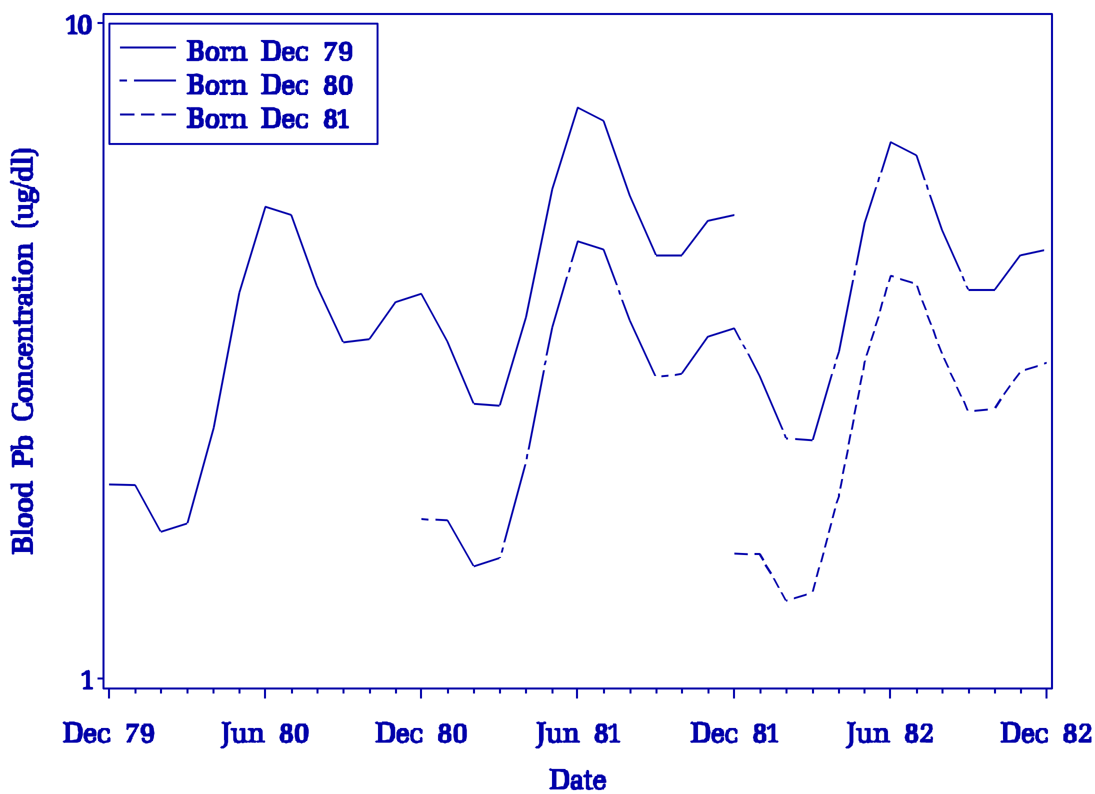
SEASONAL RHYTHMS OF BLOOD-LEAD LEVELS: BOSTON, 1979-1983

FINAL REPORT



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Technical Programs Branch
Chemical Management Division
Office of Pollution Prevention and Toxics
Office of Prevention, Pesticides, and Toxic Substances
U.S. Environmental Protection Agency
Washington, D.C. 20460

DISCLAIMER

CONTRIBUTING ORGANIZATIONS

This study was funded and managed by the U.S. Environmental Protection Agency. The study was conducted by Battelle Memorial Institute and Midwest Research Institute under contract to the Environmental Protection Agency. Each organization's responsibilities are listed below.

Battelle Memorial Institute (Battelle)

Battelle was responsible for the development of the analysis approach, for conducting the statistical analysis of the data, and for writing the final report.

Midwest Research Institute (MRI)

Midwest Research Institute was responsible for the completion of the final report.

U.S. Environmental Protection Agency (EPA)

The Environmental Protection Agency was responsible for managing the study, for reviewing the final report, and for arranging the peer review of the final report. The EPA Work Assignment Manager was John Schwemberger. The EPA Project Officers were Jill Hacker and Phil Robinson.

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Executive Summary

Several researchers have observed increased incidence of lead poisoning during summer months. Reasons for seasonal rhythms in blood-lead levels, if such a phenomenon is real, are not immediately apparent. Altered human physiology and higher levels of lead exposure during the summer months have both been postulated as reasons for the temporal variations.

This study was undertaken to examine temporal variation in blood- and environmental-lead levels in data observed for a sample of 249 children in Boston between 1979 and 1983 at the Brigham and Women's Hospital. The two primary objectives of this study were to:

- Determine the extent to which blood-lead levels recorded in the study conducted at the Brigham and Women's Hospital exhibit seasonal variation.
- Determine if any existing seasonal trends in blood-lead levels are correlated with seasonal trends in environmental levels.

For each child in the study, blood-lead and environmental-lead measurements were collected longitudinally over a period of two years. Levels of lead in air, dust, water, and soil were included in the environmental data. Nominally, between two and five measurements were taken for each response (blood or environmental lead) in six month increments.

For the investigation of seasonal trends in the blood and environmental measures, each response was analyzed separately. For statistical reasons, responses were log transformed before analysis. In addition to seasonal variations, the child's date of birth, and age were considered for possible effects. Because significant correlations were observed between the repeated measures taken on individual children, these correlations were estimated and incorporated into the model estimates.

In determining whether seasonal components of variation existed for each response, the first step was to model monthly averages and determine whether they exhibited systematic monthly variation. Although this approach reflected a significant source of variation, the interpretation is cumbersome. Therefore, because many of the media sampled exhibited higher levels in the summer and lower levels in the winter, a sinusoidal (Fourier) model was investigated for the seasonal component with parameters to represent the magnitude as well as the phase, or month of the peak level. This approach was sufficient for modeling lead levels in the environmental media. However, for blood, where the maximum and the minimum did not occur six months apart, a slightly more complicated Fourier model was required.

Blood-lead levels were found to have highly significant seasonal variations ($p<0.0001$), with the maximum modeled to occur in late June, and the minimum in March. The estimated maximum-to-minimum ratio was 2.5. Without adjusting for other effects, observed geometric mean blood-lead levels by month of year ranged from 2.1 μg/dl in February to 7.5 μg/dl in July. Age of child was also found to be a significant factor; the square root of age was found to be more linearly related to blood-lead levels than was age itself. Consistent with other studies, blood-lead levels in children were found to increase with age.

Air-, floor dust-, furniture dust-, and window sill dust-lead levels all exhibited highly significant seasonal variation. The estimated maximum-to-minimum ratios were 2.3 for air lead, 1.5 for floor dust lead, 1.4 for furniture dust lead, and 1.6 for window sill dust lead. Modeled lead levels for air, floor dust, and furniture dust all had peaks in July. Oddly, peak window sill dust-lead levels were modeled to occur in November. Each of these responses were also significantly related to the date of measurement, with a decrease observed over time. This is not unexpected due to the concurrent reduction in the use of leaded gasoline.

The extent to which levels of lead in blood were correlated with levels of lead in the environment was also evaluated. As stated above, the seasonal component of variation in blood-lead levels was highly statistically significant. However, after adjusting for the linear effects of environmental measures, the (residual) blood-lead levels did not exhibit even marginally significant seasonal variation (at the 10 percent level).

These results do not necessarily imply a causal relationship between seasonal variation in environmental-lead levels and seasonal rhythms in blood-lead levels. The fact that there were arguably parallel rhythms in blood- and, say, floor-dust lead levels, doesn't imply that the blood-lead levels are *influenced* by the floor-dust lead levels. In particular, if the floor dust-lead levels were to be multiplied by two, while retaining the same blood lead levels, and models were refit, the same statistical significance levels would be reported by this analysis approach. Thus, it would be important to develop a physiological model relating levels of lead in the environment to those in blood, before proclaiming a causal relationship.

Nonetheless in this data, which was collected in the early 1980's from a specific set of children in Boston, there was abundant evidence supporting the existence and parallelism of the seasonal variations among blood-, air-, floor dust-, and furniture dust-lead levels. The three environmental-lead measures peak in July which is very near the blood-lead peak month of June. In addition, the maximum-to-minimum ratios in the environmental-lead measures, ranging from 1.4 to 2.3, are of the same order of magnitude as the blood-lead ratio of 2.5. Thus, based on the results of this study, it is quite plausible that seasonal variations in environmental-lead levels contribute to the blood-lead rhythms.