Determination of Numbers of Lead-Exposed Women of Childbearing Age and Pregnant Women: An Integrated Summary of a Report to the U.S. Congress on Childhood Lead Poisoning

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In a Congressionally mandated study carried out under the aegis of the U.S. Agency for Toxic Substances and Disease Registry (ATSDR) and summarized in this article, the authors have provided estimates of the numbers of American women of childbearing age and the numbers of American pregnant women whose lead exposure is sufficiently elevated to pose an intrauterine toxicity risk. Exposures associated with such risk were defined as blood lead (PbB) levels > 10, > 15, > 20, and > 25 μg/dL. Using PbB prevalence projection techniques based on the Second National Health and Nutrition Examination Survey (NHANES II), we first generated projected 1984 prevalences of these PbB levels in white and black women of childbearing age, ages 15 to 19 and 20 to 44. White women in the two age bands had rates of PbBs > 10 μg/dL of 9.2 and 9.7%, respectively. For black women, the corresponding rates were 8.2 and 19.7%, respectively. Combining these rates with standard metropolitan statistical areas (SMSAs) based 1980 Census and other population enumerations show, for example, that 4.4 million U.S. women of childbearing age are estimated to have had PbBs > 10 μg/dL.

Pregnant black and white women in U.S. SMSAs are approximately 9% of the U.S. black and white childbearing age total, i.e., 3.6 million out of a 41.3 million SMSA total. Of these, 403,200 pregnant women were estimated to have PbB levels > 10 μg/dL. Cumulative totals of exposed fetuses with persisting long-term exposure will be greater, in as much as a given fetus is never counted more than once in this type of survey. Over 10 years, the number would be over 4 million fetuses at elevated risk of health effects.

Introduction

In pregnant women, lead readily crosses the placental barrier early in gestation (1–4). In utero exposure therefore occurs at periods of embryological development when important organ and system elaboration can be affected adversely by lead uptake. Severe, clinically manifested in utero effects had been known for many years (4) in pregnant women who were occupationally exposed. Currently, it is the intrauterine effects of low-level lead exposure that command the attention of the public health communities (4–8).

Two important points can be noted concerning in utero lead exposure: in utero impact can be irreversible based on current data, and the adverse impacts of maternal blood lead have been found at low levels, as low as 10 μg/dL and somewhat lower, based on current studies. While pregnant women are recognized as a high risk population segment because of intruterine exposure of the fetus (4), they were not specifically identified for consideration in recent Federal legislation mandating a report to Congress on U.S. childhood lead poisoning. This report, prepared under the sponsorship of the Agency for Toxic Substances and Disease Registry (ATSDR), did take account of the fact that intrauterine lead exposure is a component in developmental lead toxicity, and the ensuing assessment is the basis of this article.

In this article, the authors of this section of the Congressional report analyze the quantitative aspects of the impact of low-level lead exposure on the developing fetus (9). We used various blood lead (PbB) toxicity criterion levels and 1980 Census data to arrive at relevantly stratified U.S. female population exposure numbers. Risk assessment factors in the current analyses imply that every pregnancy potentially represents a fetus at risk if the mother has a blood lead...
level of 10 µg/dL or higher. Since the composition of pregnant women is not a predictable segment of the population, women of childbearing age are also examined in this analysis.

**Strategies and Methods**

We have first established numbers of women of childbearing age and pregnant women by two race and two age groups. We decided to examine women residing in standard metropolitan statistical areas (SMSAs) and of childbearing age for 1984, a relatively recent date that matches our examination of young children, and for which relevant PbB prevalences could be estimated. In addition, estimates of pregnant women for that year were obtained to illustrate the extent of risk to fetuses at any given time.

The following methodological steps established the required information for the populations and estimates of prevalences of blood lead levels among them: a) U.S. Census Bureau population projections for 1984 were used, as well as 1980 census data on residential distribution, to estimate the number of white and black women aged 15 to 19 and 20 to 44 who live in SMSAs. b) From the Division of Vital Statistics of the National Center for Health Statistics data, the numbers of live births occurring in SMSAs in 1984 were counted and fetal deaths for 1984 apportioned to SMSAs. Data for legal abortions for 1984 were not yet available, and other data from CDC (10,11) were used. From these three sources, the total number of pregnancies regardless of outcome were estimated for the four race/age strata of women in SMSAs. c) Estimates of prevalences of PbB levels of interest in pregnant women for 1984 were provided by the U.S. EPA Office of Policy Analysis, Washington, DC, by the same methodology as cited to project PbB level prevalences in young children for effects of gasoline lead phase-down action (12). These projections used baseline prevalences of PbB levels found in the 1976–1980 Second U.S. National Health and Nutrition Examination Survey (NHANES II) (13).

Specifically, the same technique of logistic regression analysis was carried out as before and as described in detail in Appendix G of the Congressional report (14) and in an earlier EPA impact analysis report directed to regulatory initiatives for phase-down of lead in gasoline (12). The PbB criterion values of concern were >10, >15, >20, and >25 µg/dL. Projected estimates of prevalences for use in 1984 were necessitated by the observed declines in gasoline lead occurring in PbB during the time of the NHANES II survey period to more recent years.

With respect to the methodological approaches employed in this chapter, several points require discussion. The selection of the two age categories for women of childbearing age is based on the fact that women below 20 years of age tend to have a high risk of pregnancies with poor outcomes in general and without specific reference to the blood lead status of the mother (15). Further, the NHANES II data for women of childbearing age indicate not only that blood lead levels showed variations for white and black women, but that women of either race showed variation by age.

We therefore examined four categories in this population: white, aged 15 to 19; white, aged 20 to 44; black, aged 15 to 19; and black, aged 20 to 44.

The attempt to estimate all pregnancies regardless of outcome is based on the recognition that the various outcomes are not intrinsically statistically relevant to the risk of fetal exposure to maternal blood lead levels. Legal abortions were included because data are available for the extent of this outcome; fetal wastage, that is, spontaneous abortions before 20 weeks of gestation, were not considered because no data exist.

The estimate of women of childbearing age includes some proportion of women who will never experience pregnancy. We know of no method to estimate this proportion. However, we believe that consideration of the number of pregnancies in a given year provides some measure of assessing the size of the surrogate population at risk.

The Census Bureau projects future population using three methods of calculation. The middle series projections for 1984 were used (16), which provide estimates by age, sex, and race, and have a 12% error for the 1984 projections (16). These are projections for the entire country, and to establish the proportion of women living in SMSAs, the 1980 residential distribution was applied to the projected 1984 figures for the four categories of women (17).

The data available for 1984 live births consisted of computer output (Division of Vital Statistics, National Center for Health Statistics) from which we had established the number of live births for each SMSA as defined in 1980. The births were identified by race, but not by age of the mother. We used the latest available data, 1981, for distribution of live births for each race by the mother’s age (18) to allocate the 1984 live births of all SMSAs. The fetal deaths for 1984 were provided by the Division of Vital Statistics and were allocated the maternal age categories within race groups by applying published information for 1981 (19).

Information for 1984 legal abortions is not yet available, and we used 1983 data. Examining the data available for the years 1971 through 1983, a peak in rates appears in 1980 and since then rates have declined very gradually. Since the number of women of childbearing age has increased steadily, the actual number of legal abortions continued to increase through 1982. In 1983, not only the rates but the actual number of abortions showed a decrease. Since the decrease of the rates is quite gradual and the actual number for 1983 below those for 1980, 1981, and 1982, employing the 1983 rates for 1984 seemed conservative.

CDC data (11) concerning legal abortions provides ratios of abortions to live births (per 1000 live births) and a rate of abortions (per 1000 women aged 15 to 44) for each of the states and the District of Columbia. A national ratio and rate are also presented. Information on the distribution of abortions by age and race are also provided, but these data are not available on a state basis.

We used two methods of estimating 1984 abortions for the SMSAs and used the result that provided the smaller numbers. An earlier, discarded method consisted of applying the ratio for each state to the number of live births that had occurred in SMSAs in that state in 1984. The sum of
Table 1. Estimated percentages of women of childbearing age exceeding selected PbB values by race and age for populations in all SMSAs, 1984.

| Race | Age, years | PbB, μg/dL | >10 | >15 | >20 | >25 |
|------|------------|------------|-----|-----|-----|-----|
| White| 15-19      | 9.2        | 0.5 | 0.1 | 0.03| 0.1 |
|      | 20-44      | 9.7        | 1.8 | 0.4 | 0.1 |
| Black| 15-19      | 8.2        | 1.3 | 0.2 | 0.05| 0.2 |
|      | 20-44      | 19.7       | 3.7 | 0.7 |

*Estimates of prevalences computed by EPA's Office of Policy Analysis, Washington, DC, using methods in Appendix G (14).

Table 2. Estimated number of women of childbearing age and estimated number of pregnant women and projected numbers above four selected PbB criterion values by race and age in all SMSAs, 1984.

| Race/age, years | Number | PbB, μg/dL |
|-----------------|--------|------------|
| Women in SMSAs* |        |            |
| White 15-19     | 5,478,000 | 504,000   | 27,400 | 5,500 | 1,600 |
| 20-44           | 29,740,000 | 2,884,800 | 553,300 | 119,000 | 29,700 |
| Black 15-19     | 1,088,000 | 90,000    | 14,300 | 2,200 | 500 |
| 20-44           | 4,984,000 | 981,800   | 184,400 | 34,900 | 10,000 |
| Total           | 41,300,000 | 4,460,600 | 761,400 | 161,600 | 41,800 |
| Pregnant women in SMSAs* |        |            |
| White 15-19     | 433,000 | 39,800    | 2,200 | 400 | 100 |
| 20-44           | 2,380,000 | 230,900  | 42,800 | 9,500 | 2,400 |
| Black 15-19     | 187,000 | 15,300   | 2,400 | 400 | 100 |
| 20-44           | 595,000 | 117,200  | 22,000 | 4,200 | 1,200 |
| Total           | 3,595,000 | 403,200  | 69,400 | 14,500 | 5,200 |

*Method of calculating explained in text.

Results and Discussion

The projected prevalence rates for the four strata of women of childbearing age and pregnant women are shown in Table 1 for the four PbB criterion levels. It should be borne in mind that the criterion value of 10 μg/dL lies in a very narrow portion of the PbB range. Certain prevalence values in the table, particularly that for the older group of black women, appear to be unusually high when compared to the other prevalences. However, because of the narrowness of the PbB range at 10 μg/dL, rather small changes in the mean PbB values will account for rather large differences in prevalences.

The pattern of increasing group values with increasing age, discussed in Chapter 10 of EPA's lead criteria document (4), can be seen in the means for each of the two racial groups. U.S. EPA (4) reports an increase of approximately 1 μg for each decade of age increase. The differences in geometric means for young and older women in each of the racial groups amounts to about 2 μg/dL.

The prevalence estimates were then applied to the estimated population strata and the findings are presented in Table 2. Census projections for 1984 and demographic distributions for 1980 were used to estimate the 41,300,000 white and black women of childbearing age who lived in SMSAs in 1984. The estimated numbers of these women in the four race/age categories above the selected PbB levels were >10 μg/dL, 4,460,600; >15 μg/dL, 761,400; >20 μg/dL, 161,600; and >25 μg/dL, 41,800.

The estimating procedure outlined above yielded a total of 3,595,000 pregnant women for 1984. Of these, 403,200 are estimated to have a PbB level above 10 μg/dL, 69,400 above 15 μg/dL, 14,500 above 20 μg/dL, and 3,800 above 25 μg/dL.

Estimated prevalences of PbB at these selected levels for 1984 are lower than those obtained from the survey data collected during 1976 to 1980 (13) which is attributable to the reduction in ambient air lead pollution (4). However it should be noted that women have a smaller uptake of airborne lead than children on a body weight basis. Unlike children, women obtain the major portion of the total body burden of lead from food and water; a smaller fraction is derived from paint dust and soil lead relative to that of preschool children. Lead in the food of adults, providing exposure for teenage and adult women, may not be reduced by the same amounts as for infants and toddlers (4), although reductions are certainly occurring across all age groups.

These projections for women should be viewed in light of the methodological variables that will contribute to both overestimation and underestimation. The logistic regression analysis accounts for the declines in women's PbB due to the phase-down of lead in gasoline but does not account for the reductions of lead in food over this time span. This would result in an overestimation.

The original NHANES II survey did not include enough women of "other race" to establish statistically reliable prevalences of PbB levels. Consequently this entire population segment is excluded from the estimates presented, which are restricted to white and black women. Women of other race constitute sizable segments of the female populations in SMSAs in the West and Southwest of the country. Finally, the women not residing in SMSAs, about 20%, were omitted entirely from the calculations presented and result in significant underestimations.

Women of childbearing age represent about 45% of the total female population. The prevalence rates for PbB levels significant to the impairment of healthy fetal development equate to about 4,460,600 women in the urban population. At any given time almost 9% are pregnant, and in a given year about 400,000 pregnancies are at risk for adverse health effects from maternal lead (>10 μg/dL PbB).

Since pregnant women within this population segment are continuously and not readily identifiable, the same
quantitative problem recurs until abatement reduces the lead in the environment of these women. In other words, no fixed, identifiable group of individuals has a one-time exposure risk. Over a 10-year period, for example, the cumulative number of individual fetuses at risk will be 10 times that of a single-year tally in the absence of steps to abate the levels of lead exposure producing the current numbers in Table 2. This amounts to over 4 million fetuses. In 50 years, this cumulative tally is about 20 million fetuses.

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REFERENCES

1. Barltrop D. Transfer of lead to the human foetus. In: Mineral Metabolism in Pediatrics (D. Barltrop and W. L. Burland Eds.), Davis Co., Philadelphia, PA, 1969, pp. 135–151.
2. Horiiuchi, K., Hiroguchi, S., and Suelkane, M. Studies on the industrial lead poisoning. I: Absorption, transportation, deposition and excretion of lead. The lead contents in organ-tissues of the normal Japanese. Osaka City Med. J. 19: 1–5 (1969).
3. Alexander, F. W., and Delves, H. T. Blood lead levels during pregnancy. Int. Arch. Occup. Environ. Health 48: 35–39 (1981).
4. U.S. Environmental Protection Agency. Air Quality Criteria for Lead. EPA report no. EPA-600/8-83/028aF-D. 4v. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC, 1986.
5. Needleman, H. L., Rabinowitz, M., Leviton, A., Linn, S., and Schoenbaum, E. The relationship between prenatal exposure to lead and congenital anomalies. J. Am. Med. Assoc. 251: 2956–2969 (1984).
6. Dietrich, K. N., Krafft, K. M., Shukla, R., Bornschein, R. L., Hammond, P. B., Berger, O., Succop, P. A., and Bier, M. Effects of low-level fetal lead exposure on neurobehavioral development in early infancy. Pediatrics 89: 721–730 (1987).
7. Bellinger, D., Leviton, A., Wateraux, C., Needleman, H., and Rabinowitz, M. Longitudinal analyses of prenatal and postnatal lead exposure and early cognitive development. N. Engl. J. Med. 316: 1037–1043 (1987).
8. McMichael, A. J., Baghurst, P. A., Wigg, N. R., Vimpani, G. V., Robertson, E. F., and Roberts, R. J. Port Pirie cohort study: environmental exposure to lead and children’s abilities at the age of four years. N. Engl. J. Med. 319: 468–475 (1988).
9. ATSDR. Examination of numbers of lead-exposed women of childbearing age and pregnant women. In: The Nature and Extent of Childhood Lead Poisoning in the United States: A Report to Congress, Chapter VII. Submitted to Congress July 1988 by the Agency for Toxic Substances and Disease Registry, Public Health Service, Atlanta, GA.
10. U.S. Centers for Disease Control. Abortion Surveillance, 1979–1980. CDC, Atlanta, GA, May, 1983.
11. U.S. Centers for Disease Control. Abortion Statistics. U.S. 1982-1983 Center for Health Promotion and Education, Atlanta, GA, 1986.
12. U.S. Environmental Protection Agency. Costs and Benefits of Reducing Lead in Gasoline: Final Regulatory Impact Analysis. EPA report no. EPA-230/05-85-006, Office of Policy Planning and Evaluation, Washington, DC, 1985.
13. Annest, J. L., and Mahaffey, K. R. Blood Lead Levels for Persons Ages 6 Months–74 Years, United States, 1976–80. DHHS publication no. (PHS) 84-1683. (National Health and Nutrition Examination Survey Series 11, no. 233), U.S. Department of Health and Human Services, Washington, DC, 1984.
14. ATSDR. The Nature and Extent of Childhood Lead Poisoning in the United States: A Report to Congress. Submitted July 1988 by the Agency for Toxic Substances and Disease Registry, Public Health Service, Atlanta, GA.
15. National Research Council. Risking the Future. Adolescent Sexuality, Pregnancy, and Childbearing. Vol. I (C. B. Hayes, Ed.), National Academy Press, Washington, DC, 1987.
16. U.S. Bureau of the Census. Current Population Reports, Series P-25, No. 952, Projections of the Population of the United States by Age, Sex and Race: 1983 to 2080. U.S. Department of Commerce, Washington, DC, 1985.
17. U.S. Bureau of the Census. Census of Population 1980. Characteristics of the Population, General Social and Economic Statistics: United States Summary. U.S. Department of Commerce, Washington, DC, 1985.
18. U.S. Department of Health and Human Services, Public Health Service, National Center for Health Statistics. Vital Statistics of the United States, 1981. Vol. I, Natality. NCHS, Hyattsville, MD, 1985.
19. U.S. Department of Health and Human Services, Public Health Service, National Center for Health Statistics. Vital Statistics of the United States, 1981. Vol. II, Mortality Part B. NCHS, Hyattsville, MD, 1986.