# Economic Gains Resulting from the Reduction in Children's Exposure to Lead in the United States

*Scott D. Grosse,[1] Thomas D. Matte,[1] Joel Schwartz,[2] and Richard J. Jackson[1]*

[1]National Center for Environmental Health, Centers for Disease Control and Prevention, Atlanta, Georgia, USA; [2]School of Public Health, Harvard University, Cambridge, Massachusetts, USA

**In this study we quantify economic benefits from projected improvements in worker productivity resulting from the reduction in children's exposure to lead in the United States since 1976. We calculated the decline in blood lead levels (BLLs) from 1976 to 1999 on the basis of nationally representative National Health and Nutrition Examination Survey (NHANES) data collected during 1976 through 1980, 1991 through 1994, and 1999. The decline in mean BLL in 1- to 5-year-old U.S. children from 1976–1980 to 1991–1994 was 12.3 μg/dL, and the estimated decline from 1976 to 1999 was 15.1 μg/dL. We assumed the change in cognitive ability resulting from declines in BLLs, on the basis of published meta-analyses, to be between 0.185 and 0.323 IQ points for each 1 μg/dL blood lead concentration. These calculations imply that, because of falling BLLs, U.S. preschool-aged children in the late 1990s had IQs that were, on average, 2.2–4.7 points higher than they would have been if they had the blood lead distribution observed among U.S. preschool-aged children in the late 1970s. We estimated that each IQ point raises worker productivity 1.76–2.38%. With discounted lifetime earnings of $723,300 for each 2-year-old in 2000 dollars, the estimated economic benefit for each year's cohort of 3.8 million 2-year-old children ranges from $110 billion to $319 billion.** *Key words:* **child, cognition disorders/chemically induced/prevention and control, environmental exposure/economics, environmental monitoring, intelligence, lead/adverse effects, blood lead.** *Environ Health Perspect* **110:563–569 (2002). [Online 15 April 2002]** ***http://ehpnet1.niehs.nih.gov/docs/2002/110p563-569grosse/abstract.html***

The removal of lead from automotive fuel, new residential paint, food containers, children's toys, and municipal drinking water systems constitutes one of the major public health success stories of the last quarter century in the United States. These actions have been accompanied by at least an 80% reduction in human exposure to lead, as measured in blood lead levels (BLLs) in the U.S. population (*1,2*). Although both adults and children continue to be exposed to lead, chiefly through lead-contaminated dust in and around older housing, tremendous progress in reducing exposure has already been achieved. The health and economic benefits resulting from regulatory and market-based actions that have reduced the amount of lead added to the environment illustrate the potential economic contribution of environmental health policies, including actions aimed at eliminating ongoing lead poisoning.

Lead exposure can cause many adverse health effects. Severe lead poisoning, at levels exceeding 70 μg lead/dL of blood, can lead to encephalopathy and death. Other health effects associated with less severe exposures include hypertension, anemia, and impaired nerve conduction (*3*). At levels of lead exposure commonly observed in the United States today, the most common risk of harm among children is subtle impairment of neurodevelopment, with small but measurable effects on cognitive and behavioral outcomes. The best-established measure of neurologic deficit associated with early lead exposure is reduced cognitive performance as measured on standardized tests of general intellectual ability, or IQ. Information about the specific "behavioral signature" of neuropsychologic effects of lead is a topic of current research (*4*). Researchers are investigating how much we can reverse the negative effects of lead exposure on cognitive ability in children with already elevated BLLs by removing them from exposure to lead (*5*).

Differences in IQ associated with low-to-moderate levels of lead exposure are generally small and may be difficult to detect. When aggregated across millions of individuals, even small differences in IQ can make a major impact. Cognitive ability affects school performance, educational attainment, and success in the labor market, and hence is positively associated with earnings. Improvements in cognitive ability benefit society by raising economic productivity, including profits and tax revenues, and by reducing crime and other behaviors with negative impacts on others. Because society has borne the costs of preventing lead exposure, through higher prices or lower profits and through public expenditures, examining the benefits from society's perspective makes sense.

## Model and Sources of Data

Below, we first describe the causal model we used to estimate the economic benefits of reducing lead exposure in the population of U.S. children through the impact of lead on IQ levels and of IQ on earnings. Then, for each component of the model underlying our benefit calculations, we briefly discuss the available evidence. Finally, we present the assumptions for our model and the results of our analysis. Both the conceptual model and specific methods relating to the impact of lead on cognitive ability and the economic valuation of cognitive ability are largely based on previously published work of one of the authors (*6*).

We represented the impact of lead in the environment on economic productivity by a simplified causal model (Figure 1). First, lead in the environment produces human exposure to lead, as quantified through BLLs. Second, BLLs influence impairment of cognitive ability among children, as measured in scores on intelligence tests conducted at school age. Third, cognitive ability proportionally influences individual earning potential, which is described as a percentage difference from average earnings in the population. Fourth, the expected earnings for a person are the product of the person's ability and the aggregate level of productivity determined by macroeconomic conditions. We described average productivity as the present value of average earnings for a cohort, taking into account expected changes in future productivity.

We calculated the economic benefit realized by reduced lead exposure in the United States since the late 1970s through a series of steps, each associated with a component of the model in Figure 1. First, we estimated the amount by which BLLs have fallen over time through secondary analysis of data from the National Health and Nutrition Examination Surveys (NHANES). Second, we applied estimates from published studies of the strength, shape, and magnitude of the association between BLLs and cognitive ability test scores. In particular, we examined two published meta-analyses to arrive at estimates of

Address correspondence to R.J. Jackson, Centers for Disease Control and Prevention, 4770 Buford Highway NE, Mail Stop F-29, Building 101, Room 3125, Atlanta, GA 30341 USA. Telephone: (770) 488-4575. Fax: (770) 488-7024. E-mail: rjjackson@cdc.gov

We recognize the following persons for their helpful comments on the manuscript: R. Barlow, C. Brown, S. Buchanan, T. Hodgson, A. Honeycutt, J. Pirkle, T. Sinks, and D. Sussman.

Received 11 June 2001; accepted 30 October 2001.

the ratio of change in BLL to change in IQ. Third, on the basis of a brief review of literature on the association between cognitive ability and earning potential, we estimated the percentage change in earnings associated with absolute differences in IQ levels. Fourth, we calculated the present value (2000 dollars) of the percentage change in earnings.

The product of estimates derived from these four steps leads to the estimated dollar value of increased productivity for a representative newborn relative to an individual from an earlier generation exposed to a higher level of lead in the environment:

$$\Delta E = \Delta BLL \times \left(\frac{\Delta IQ}{\Delta BLL}\right) \times \left(\frac{\frac{\Delta E}{E}}{\Delta IQ}\right) \times E$$

where ΔE = change in earnings (in dollars), ΔBLL = change in BLL (in micrograms per deciliter), ΔIQ/ΔBLL= change in IQ points per unit BLL, ΔE/E/ΔIQ = percent change in earnings per IQ point, and E = discounted lifetime earnings (in dollars).

***Trends in blood lead levels, 1976–1999.*** Exposure of people to environmental chemicals can be assessed through biomonitoring, the measurement of chemical concentrations in human biologic samples. In particular, the concentration of lead in blood reflects a mixture of current exposure through ingestion of lead and the release of lead previously stored in bone. The Centers for Disease Control and Prevention (CDC) uses biomonitoring to measure a range of chemicals in blood and urine samples collected from a national sample of the U.S. population each year through NHANES (*7*).

Blood lead measures are available for NHANES II (conducted from 1976 to 1980), NHANES III (conducted from 1988 to 1994), and the initial year of data (1999) for the ongoing NHANES (*1,2,7,8*). NHANES III was conducted in two distinct phases: phase A during 1988–1991 and phase B during 1991–1994. Nationally representative estimates are available for NHANES II, NHANES IIIA and IIIB, and 1999 NHANES data (*1,2,7,8*). Because the distribution of BLLs is skewed to the left, the CDC reports geometric mean BLLs as the most appropriate measure of the central tendency of the distribution of BLLs. In the NHANES IIIB data, the arithmetic mean BLL among children 1–5 years of age is 3.6 μg/dL, compared with a geometric mean of 2.7 μg/dL, and a median of 2.6 μg/dL.

Data from NHANES (Table 1) show a dramatic decline in average (geometric mean) BLLs in 1- to 5-year-old U.S. children, from 15.0 μg/dL during 1976–1980 to 3.6 μg/dL during 1988–1991 and 2.7 μg/dL during 1991–1994 (*1,2*). The decline in geometric mean BLL for children from the late 1970s to the early 1990s parallels declines in BLLs among older people.

The decline in BLLs from the mid-1970s to the present is somewhat greater than the estimate of an 82% decline from 1976–1980 to 1991–1994 (from 15.0 to 2.7 μg/dL). First, BLLs were declining during the late 1970s while NHANES II was being conducted. For all ages in NHANES II, the average BLL declined from 14.6 μg/dL in February 1976 to 9.2 μg/dL in February 1980, a decline of 37% (*8*). The ratio of the 1976 value (14.8 μg/dL) relative to 1976–1980 (12.8 μg/dL) is 1.14. The same ratio applied to the period mean BLL for 1- to 5-year-old children implies an estimated mean BLL for children in 1976 of 17.1 μg/dL. This is a conservative estimate because the percentage change in BLLs for 1976–1980 was greater among young children than among persons of all ages (*8*).

BLLs have continued declining since 1994. The most recent estimate from NHANES for 1999 shows a geometric mean BLL of 2.0 μg/dL among 1- to 5-year-old children (*9*). From 17.1 μg/dL in 1976 to 2.0 μg/dL in 1999, geometric mean BLL among 1- to 5-year-old children declined by 15.1 μg/dL during these 24 years. The estimates for both 1976 and 1999 are not precise because samples are smaller for single survey years than they are for multiple years.

***Lead exposure and cognitive ability.*** The best documented adverse health effect of moderate-to-low levels of lead exposure among children is impaired neurodevelopment, specifically, performance on standardized intelligence tests. Results of most observational studies of associations between lead exposure in children and performance on intelligence tests show significant inverse associations between postnatal lead exposure and general cognitive ability. Although the inverse association is not significant in every case, the findings across the studies show "striking consistency" (*3*). Results from any individual study should be placed in the context of the range of estimates from peer-reviewed studies.

The inference of a causal effect from observational studies may be questionable. Earlier observational studies of lead and child development had a number of limitations, including a lack of control for confounding by markers of social deprivation that may be associated with both greater exposure to lead and poorer developmental outcomes. These limitations have been addressed in subsequent studies through improved study designs, including prospective enrollment from the prenatal period and rigorous measurement of potential confounders. We can place more confidence in the results of prospective cohort studies that measure lead exposure over multiple time points and assess the characteristics of the home environment. In addition, experimental studies performed in animals support the cause-and-effect association observed in humans (*3*).

The most comprehensive evidence of the strength of the association between BLLs and IQ levels comes from two published meta-analyses of epidemiologic studies (*10,11*). Meta-analyses combine results from multiple studies to allow for increased statistical power in testing hypotheses because small sample sizes in individual studies may contribute to lack of statistical significance. Two meta-analyses have analyzed regression coefficients of blood lead on IQ (*10,11*). One of these included three prospective studies and four cross-sectional studies (*10*). The other, which employed less restrictive inclusion criteria, included five prospective or longitudinal studies and 14 cross-sectional studies (*11*).

A meta-analysis entails pooling published estimates of strength of association, typically regression coefficients, along with standard errors. One complication for meta-analysis of BLLs and IQ is that original research studies have used different statistical models; some studies report linear regression coefficients, whereas others report nonlinear regression coefficients based on models in which the IQ score is regressed on the natural logarithm of blood lead. To combine estimates from the two types of studies, one must evaluate the nonlinear regression coefficients over a specific range of BLLs dictated by the distribution of observations in the original study populations. Both meta-analyses evaluate the

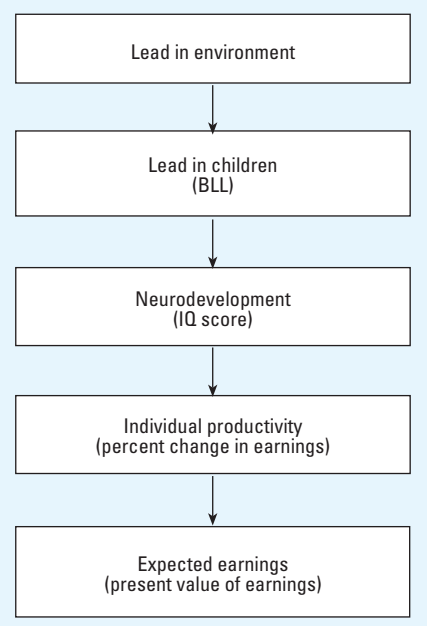


**Figure 1.** Causal model of lead exposure and economic productivity.

pooled regression coefficients as the change in IQ points as a BLL rises from 10 µg/dL to 20 µg/dL.

Schwartz (*10*) reported that the average slope is –2.57 IQ points. The effect sizes in the three longitudinal studies, all of which controlled for confounding, were –5.8, –1.3, and –3.3 IQ points. The pooled effect size from the three longitudinal studies is –2.96 IQ points. Pocock et al. (*11*) reported a slope of –1.85 IQ points for five cohort studies and –2.53 IQ points for 14 cross-sectional studies. The two additional cohort studies included in the analysis of Pocock et al. had adjusted effect sizes of –1.1 and 0.39. These two studies did not meet Schwartz's inclusion criteria of measures of intelligence in school-aged children and BLLs measured through venous blood samples (*10*). In addition, Pocock et al. (*11*) noted that their method gave the smallest weight to the cohort study with the largest numbers of observations.

Studies published since the two 1994 meta-analyses (*10,11*) are consistent with previously published studies. In particular, in a prospective cohort study conducted in Kosovo, Yugoslavia, Wasserman et al. (*12*) found that after controlling for a wide range of confounders, including home environment and maternal intelligence, cumulative postnatal lead exposure was significantly associated with IQ measured at 7 years of age. A change in BLL from 10 µg/dL to 30 µg/dL was associated with a difference in IQ of 4.3 points, after adjusting for confounders.

Previous analyses of the societal benefit of lead poisoning prevention on aggregate IQ scores have applied the slope from the Schwartz's meta-analysis for the BLL range from 10 µg/dL to 20 µg/dL (*6*) to the entire distribution of BLLs. This approach makes two critical assumptions. First, it assumes that there is no BLL threshold below which lead does not have an adverse impact on cognitive ability. This assumption is consistent with the absence of evidence of a threshold (*3,13*). It also assumes that the association is linear across all BLLs. This assumption is less likely to be valid.

The association between BLLs and IQ appears to be nonlinear. Evidence points to a slope that is greater in absolute magnitude between 5 µg/dL and 15 µg/dL than at levels > 15 µg/dL. For example, Schwartz (*10*) noted that the mean effect size is –3.23 for studies with a mean BLL of ≤ 15 µg/dL compared to –2.32 for studies with a mean BLL > 15 µg/dL. Analyses of data from the Port Pirie study in Australia (*14*) using log-linear regression imply an increasing slope at lower absolute BLLs; in particular, by comparing cumulative BLLs to IQ measured at 7 years of age and adjusting for confounders, Tong et al. found that each log unit of BLL is associated with a 4.6 IQ point differential. When IQ was evaluated with a change in BLL from 10 µg/dL to 20 µg/dL, the differential equaled –3.2 points. This is close to the mean estimate of effect size from Schwartz's meta-analysis for longitudinal studies (*10*). When BLL is evaluated in the range of 20–30 µg/dL, the IQ differential equals –1.9 points. The effect of raising BLL from 5 µg/dL to 10 µg/dL appears to be –3.2 IQ points, which equals the effect size of a change in BLL from 10 µg/dL to 20 µg/dL. However, extrapolating results to values below those observed in that study (i.e., < 5 µg/dL) could be misleading.

Evidence from one study (*15*) suggests that the strong association between BLL and IQ in the range of BLLs from 5 µg/dL to 15 µg/dL may be somewhat attenuated at lower BLLs. Schwartz (*15*) published a nonparametric curve fit on the basis of data from the Boston cohort study (*16*) that related BLL measured at 2 years of age to IQ measured at 10 years of age. Visual interpolation of this graph suggests that at BLLs < 7 µg/dL a linear approximation of the relation is –1.7 IQ points per 10 µg/dL. This magnitude is less than one-third of the approximate effect size of –5.8 IQ points per 10 µg/dL for the BLL range between 7 µg/dL and 15 µg/dL.

In a recently published study, Lanphear et al. (*13*) found no attenuation of the relation between BLL and ability at low BLLs, although the measure of ability was academic achievement, not IQ. Analysis of NHANES III survey data on children 6–16 years of age provided evidence that the magnitude of the relation between BLLs and academic achievement is greater for BLLs < 10 µg/dL than at higher levels (*13*). Lanphear et al. (*13*) estimated the relation between BLLs and academic achievement for all children with BLLs in the sample and for subsets with BLLs < 10, 7.5, 5, and 2.5 µg/dL. For both arithmetic and reading test scores, the impact of a 1 µg/dL reduction in BLLs on performance was larger, the lower the maximum BLL for the group, although statistical significance was reduced with smaller sample sizes at levels < 2.5 µg/dL. One limitation of Lanphear et al.'s study (*13*) is that the data did not permit the researchers to control for important confounders, including parental intelligence and the home environment. Preliminary results from a new prospective cohort study have confirmed that the slope between BLL and IQ is greater for BLLs < 10 µg/dL (*17*).

***Cognitive ability and earnings.*** The impact of cognitive ability on earning potential is not easy to define. Econometric studies of earnings determinants have often used scores on the Armed Forces Qualifying Test (AFQT) or specific subtests to control for differences in ability (*18*). AFQT scores reflect achieved academic ability as well as innate cognitive ability and are not the same as IQ test scores. In this section, we present estimates from three empirical studies that model the edfects of cognitive ability, proxied by AFQT scores, on earnings in young adults.

Cognitive ability influences productivity through a direct effect on earning potential (hourly wages) and annual earnings and an indirect influence through years of schooling and employment (Figure 2). Analyses of just the direct effect of ability on earnings understate the economic value of cognitive ability. Conversely, estimates that include indirect effects may overstate the effect of cognitive ability by attributing shared variation to the effect of ability alone.

Earnings are the product of hourly earnings and annual hours of work, and annual hours of work is the product of probability of participation and hours of work conditional on participation. Schwartz (*6*) implicitly assumed that the annual number of hours of work is a constant. He presented the following algebraic formula for calculation of change in earnings:

$$\Delta E = P \times \Delta W + W \times \Delta P + \Delta W \times \Delta P,$$

where $\Delta E$ = change in earnings, $P$ = work participation, $W$ = hourly wages, $\Delta W$ = change in wages, and $\Delta P$ = change in participation. For small changes in wages and participation, the last term in the equation is negligible. Setting this term equal to zero,

**Table 1.** Geometric mean BLLs from NHANES, 1976–1999 (µg lead/dL blood).

| Year(s) | Age of children 1–5 years | Age of children ≥ 1 year |
|---|---|---|
| 1976 (February) | 17.1 | 14.6 |
| 1976–1980 (average) | 15.0 | 12.8 |
| 1988–1991 (average) | 3.6 | 2.8 |
| 1991–1994 (average) | 2.7 | 2.3 |
| 1999 | 2.0 | 1.6 |

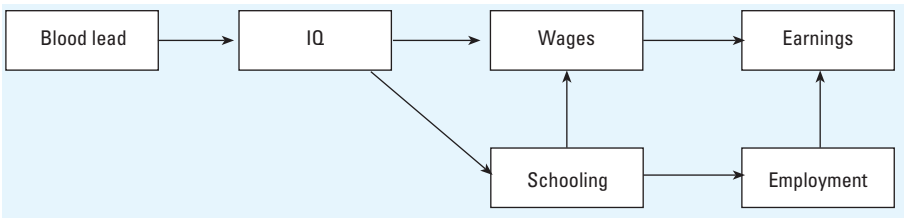


**Figure 2.** Causal model of cognitive ability and economic productivity.

using the identity that $E = P \times W$, and substituting $E/W$ for $P$ and $E/P$ for $W$ in the formula above, the expression is algebraically equivalent to

$$\Delta E = E \times \left( \frac{\Delta W}{W} + \frac{\Delta P}{P} \right)$$

This expression indicates that the change in earnings in absolute dollars is the product of the level of earnings and the sum of the percentage changes in wages and participation rates.

Schwartz (*6*) calculated that the total effect of a 1-point difference in cognitive ability is a 1.76% difference in earnings. Of this amount, 0.5% is the direct effect of ability on earnings. Schwartz (*6*) took this estimate from an econometric study by Griliches (*19*) that was representative of other econometric studies from the 1970s. Schwartz (*6*) assumed that a given difference in IQ scores observed in school-aged children can be expected to lead to a comparable difference in achieved cognitive ability in young adults.

The indirect effect of ability on earnings, which accounts for the remaining 1.26% difference, is modeled through two pathways. One is the effect of ability on years of schooling multiplied by the effect of years of schooling on hourly earnings. Needleman et al. (*20*) reported that a 4.5-point difference in IQ between groups with high tooth lead and with low tooth lead was associated with a 0.59 difference in grade level attained. The ratio of the two numbers implies a difference of 0.131 years of schooling for 1 IQ point. If each additional year of schooling results in a 6% increase in hourly wages, 1 IQ point would lead to a 0.79% increase in expected earnings through years of education.

Second, Schwartz (*6*) modeled ability as influencing employment participation through influence on high school graduation. On the basis of the analysis of Needleman et al. (*20*) and 1978 survey data reported by Krupnick and Cropper (*21*), Schwartz (*6*) calculated that 1 point in IQ is associated with a 4.5% difference in probability of graduating from high school and that high school graduation is associated with a 10.5% difference in labor force participation. On the assumption of an equivalent percentage change in annual earnings, this leads to a 0.47% difference in expected earnings.

Salkever (*22*) published an alternate estimation of the effect of cognitive ability on earnings. Salkever directly estimated the effect of ability on annual earnings, among those with earnings. The estimated association of ability with annual earnings incorporates both the effect of ability on hourly earnings and its effect on annual hours of work. He also added a direct pathway from ability to work participation independent of education.

According to Salkever (*22*), a 1-point difference in ability is associated with a 1.931% difference in earnings for males and a 3.225% difference for females. The direct effect on earnings is 1.24% for males and 1.40% for females. Salkever (*22*) analyzed income and educational attainment data from the 1990 wave of the National Longitudinal Study of Youth (NLSY) in combination with AFQT scores collected during 1979–1980, when the respondents were 14–23 years of age.

For the indirect effect of ability on schooling attainment, Salkever (*22*) reported that a 1-point difference was associated with 0.1007 years of schooling attained for both males and females in the NLSY data. Also, 1 year of schooling attainment raised hourly earnings by 4.88% for males and 10.08% for females in the 1990 NLSY data. According to these results, a 1-point difference in ability is associated, through an indirect effect on schooling, with a 0.49% difference in earnings for males and a 1.10% difference in earnings for females.

Salkever (*22*) reported that the direct effect of a 1-point difference in ability was a 0.1602 percentage point difference in probability of labor force participation for males and a 0.3679 percentage point difference for females. In addition, he calculated that 1 year of schooling raised labor force participation rates by 0.3536 percentage points for males and 2.8247 percentage points for females. Subtracting the other components from the totals, a 1-point change in cognitive ability is associated with a difference in earnings of 0.20% for males and 0.72% for females through effects on labor force participation.

Finally, in an analysis of the 1990 NLSY earnings data, Neal and Johnson (*23*) reported smaller estimates of the effect of cognitive ability on earnings. They included workers who took the AFQT test when they were 14–18 years of age and excluded those who took the AFQT test at 19–23 years of age to make the test scores more comparable. They also estimated the total effect of ability on hourly earnings by excluding schooling variables. Their estimates indicate that a 1-point difference in AFQT scores is associated with a 1.15% difference in earnings for men and a 1.52% difference for women. Their estimate of the direct effect of ability on hourly earnings, controlling for schooling, is 0.83% for men; they reported no estimate for women.

The analysis of Neal and Johnson (*23*) has no link from ability to labor force participation. According to Salkever (*22*), a 1-point difference in ability leads to a 0.20% difference for males and 0.72% for females. If we add Salkever's figures (*22*) to the estimates from Neal and Johnson (*23*), the total effect of a 1-point difference in ability on earnings is 1.35% for males and 2.24% for females.

The sex-specific estimates of the effect of cognitive ability on earnings from the two studies discussed above (*22,23*) can be weighted using the share of each sex in aggregate earnings. Data from the Current Population Survey (*24*) reveal that women in 2000 accounted for 34.3% of aggregate earnings. Using this number to weight Salkever's estimate (*22*) of a 3.225% difference in earnings for females and 65.7% to weight the 1.931% estimate for males, the weighted difference in earnings associated with a 1-point difference in ability in Salkever's analysis is 2.37%. The weighted average difference in Neal and Johnson's analysis (*23*), together with the addition of the labor force participation component estimates from Salkever (*22*) is 1.66%, similar to Schwartz's estimate of 1.76% (*6*).

***Present value of lifetime earnings.*** The dollar value of cognitive ability is calculated as the percentage difference in earnings attributable to cognitive ability multiplied by the value of expected earnings. We defined earnings to include the value of payroll taxes and fringe benefits such as health insurance, and we calculated expected earnings as the mean of the entire earnings distribution. A percentage change in individual earnings multiplied by the number of people in a cohort will yield the aggregate reduction in earnings expected by the cohort as a whole. The impact on any individual cannot be readily predicted.

The calculation of lifetime earnings projects future earnings by taking the current age profile of earnings and adjusting for survival and future labor productivity growth. The usual practice is to assume constant 1% growth in future real earnings, consistent with recent historical experience. Finally, earnings in future years are converted to present values through discounting (equivalent to the inverse of compound interest) to reflect time preference. The social rate of time preference reflects society's preference between present and future consumption, independent of individual risk and longevity. It is assumed to be approximated by the long-term return on risk-free investments after adjusting for inflation. In recent U.S. history, this rate of return has varied from 2.5% to 5% (*25*). In 1996, an expert panel convened by the U.S. Public Health Service recommended a social discount rate of 3% for economic analyses conducted from the societal perspective (*26*). The expert panel suggested that results of economic evaluations also be reported using a 5% discount rate to ensure comparability with previous studies.

We took the estimates of present value of earnings from a new set of calculations that are published elsewhere (*27*). These estimates are based on earnings and employee compensation data for 2000. We used a weighted average of estimates of present values at birth and at 5 years of age to represent the present value at 2 years of age, and calculated the present value of earnings to be $723,300 using a 3% discount rate and assuming 1% annual growth in labor productivity. With a 5% discount rate, the present value is calculated to be $353,400. Without discounting, the value of expected lifetime earnings is $2,448,400.

## Assumptions and Results

We used the 12.3 µg/dL decline in geometric mean BLLs between NHANES II (1976–1980) and the second phase of NHANES III (1991–1994) for our lower-bound analysis (*1,2*). An alternative estimate, based on a comparison of single-year NHANES estimates for 1976 and 1999, is a decline of 15.1 µg/dL (*7,8*). The 1999 BLL is less precisely calculated because of small numbers for a single survey year. Insufficient numbers of observations on BLLs among children are available from NHANES II to directly calculate estimates for children by year for 1976. We believe our estimation method is conservative and that the true number in 1976 was probably higher. Further, given that BLLs were already declining when NHANES II began, values before 1976 likely were even higher. For these reasons, we used the estimate of a decline of 15.1 µg/dL as the assumption for the base-case analysis. Because no basis exists for calculation of a higher specific estimate, we also used this number for the upper-bound analysis.

The exact shape of the relation between children's BLLs and cognitive ability is not known. No evidence exists of a threshold BLL below which no adverse effects occur. If the association were strictly linear, use of arithmetic mean BLL along with a constant slope relating BLL to IQ would be appropriate. However, because the association between BLL and IQ is smaller at BLLs > 20 µg/dL (*14*), the use of an arithmetic mean BLL would overstate the aggregate change in IQ resulting from reductions in BLLs at higher levels. If the association were log-linear, use of the mean logarithm of BLL along with a regression slope of log BLL to IQ would be appropriate. Extrapolation of the log-linear relation observed at higher levels implies an extremely high proportional slope at very low BLLs (< 5 µg/dL).

The most conservative approach, which we retain, is to apply a linear slope coefficient, evaluated at 10–20 µg/dL, to the change in geometric mean BLL. A linear slope is preferable because of the availability of estimates pooled from multiple studies. Sufficient evidence to calculate a nonlinear estimate of the association of blood lead with IQ at BLLs < 10 µg/dL is not yet available. Evidence that the slope appears to be greater at BLLs < 10 µg/dL suggests that this method may understate the change in aggregate IQ points attributable to reduction in BLLs.

The most commonly used estimate of the association between BLLs and cognitive ability is a slope of 2.57 IQ points for each 10 µg/dL of BLL, or 0.257 per 1 µg/dL (*10*). We take this estimate as the assumption for our base-case analysis. Sources of uncertainty include the selection of studies to be pooled, the form of the statistical model, and the relevant range of BLLs, and the association may be either larger or smaller. For our lower-bound estimate, we use the effect size (0.185) from cohort studies in the meta-analysis by Pocock et al. (*11*). For our upper-bound estimate, we take the estimated effect size (0.323) for the group of studies in the Schwartz meta-analysis with mean BLL below 15 µg/dL (*10*).

**Table 2.** Assumptions and results of three calculations of the economic benefit to the 2000 birth cohort resulting from reduction in lead exposure since the 1970s.

| Parameter | Lower bound | Base case | Upper bound |
|---|---|---|---|
| Assumptions | | | |
| A. Reduction in BLL (µg/dL) | 12.3 | 15.1 | 15.1 |
| B. IQ–BLL slope | 0.185 | 0.257 | 0.323 |
| C. Earnings–IQ slope (%) | 1.76 | 2.00 | 2.37 |
| D. Present value of earnings of 2-year-old (in 2000 dollars) | 723,300 | 723,300 | 723,300 |
| E. Size of 2-year-old cohort | 3,800,000 | 3,800,000 | 3,800,000 |
| Results (formulas) | | | |
| Value of one IQ point (in 2000 dollars) (C × D) | 12,700 | 14,500 | 17,200 |
| Gain per 1 µg/dL (in 2000 dollars) (B × C × D) | 2,350 | 3,720 | 5,550 |
| Benefit per individual (in 2000 dollars) (A × B × C × D) | 29,000 | 56,100 | 83,800 |
| Benefit per cohort (in 2000 dollars) (A × B × C × D × E) | 110 billion | 213 billion | 318 billion |

Two published estimates of the overall effect of cognitive ability on earnings are available (*6,22*). A 1-point difference in IQ was assumed to raise earnings for males and females together by 1.76% by Schwartz (*6*), and by 2.37% by Salkever (*22*). A third estimate, of 1.66%, derived from the study by Neal and Johnson (*23*), is essentially the same as the estimate by Schwartz, which we retained as our lower-bound estimate. For our base-case analysis, we used a 2.00% estimate, based on modification of Salkever's estimate (*22*) to exclude one of the two participation pathways. For our upper-bound estimate, we used Salkever's weighted average estimate of 2.37%.

The economic gain enjoyed by each year's birth cohort as a result of the already realized reduction in lead exposure can be calculated as the product of five parameters. Table 1 specifies one or more values for each parameter. As discussed above, Table 1 shows two values for the reduction in BLL in the United States from the 1970s to the 1990s. For the percentage difference in earnings resulting from one IQ point difference, we used three estimates from two sources (*6,22*). For the slope between BLL and IQ, we took three values from two other sources (*10,11*). We used one estimate of the present value of lifetime earnings for a 2-year-old child, calculated using a 3% discount rate ($723,300). In the "Discussion" below, we also present results with the present value of earnings calculated using 0% and 5% discount rates. Finally, we specify a single value for the size of the annual cohort of children reaching 2 years of age, 3.8 million.

Table 2 presents three summary measures of the calculations: lower bound, base-case, and upper bound. Using the most conservative assumptions, we found that the cohort of children reaching 2 years of age during 2000 will gain an aggregate of at least $110 billion in the present value of future earnings as a result of being protected from the level of lead exposure to which the cohort's counterparts in the mid-1970s were exposed. With the upper-bound assumptions, the cohort will gain $318 billion in the present value of lifetime earnings.

## Discussion

Preventing childhood lead poisoning is one of the great success stories of public health in the United States in the last quarter century. Many different criteria have been used to define unacceptable exposure to lead, with the threshold of concern falling over time as scientific knowledge of low-level health effects has increased and as exposure levels have decreased (*3*). The 1985 CDC criterion for elevated blood lead was a BLL of ≥ 25 µg/dL (*3*). The percentage of children with

lead exposure at this level fell from 9.3% in 1976–1980 to 0.2% in 1991–1994 (*1,2*). Since 1991, the CDC has recommended a cutoff of 10 µg/dL as the elevated BLL above which children should be monitored (*3*). During 1976–1980, 88.2% of 1- to 5-year-old children in the United States had BLLs above this level of concern. During 1991–1994, only 4.4% of children had BLLs above this level of concern. However, even if no children had BLLs > 10 µg/dL, the problem of subtle impairment of neurodevelopment at BLLs < 10 µg/dL would remain.

This paper is the first to place a dollar value on societal gains resulting from the dramatic reduction in the exposure of the U.S. population to lead in the environment since the 1970s. Specifically, we estimated one major component of economic benefit—the gain in earning power that each year's newborns experience as a result of not being exposed to the same level of lead as their counterparts were a generation earlier. The analysis represents an extension of a published framework that has received widespread support in previous economic evaluations of lead poisoning prevention (*6*). Rather than presenting a single point estimate, we present a range of estimates, given the uncertainty associated with many of the parameters underlying the estimates. The estimates range from a lower bound of $110 billion to an upper bound of $318 billion. Even the most conservative of these estimates is extraordinarily large.

The base-case estimate of $213 billion in economic benefit for each cohort is based on conservative assumptions about both the effect of IQ on earnings and the effect of lead on IQ. All of the published estimates of the effect of cognitive ability on earnings have been based on studies of earnings in young adults. Because skill differentials in earnings rise with age (*18*), the true effect of ability on lifetime earnings is likely to be substantially greater than the proportionate effects assumed in this analysis. The causal nature of the association between lead exposure and cognitive ability, assumed in this study, is supported by epidemiologic and experimental evidence and is widely accepted by scientists (*3*). The base-case estimate of the magnitude of the association derived from two meta-analyses is conservative because the association between BLLs and IQ varies with initial BLL. The evidence from studies of the lead–IQ association in which BLLs ranged from 5 µg/dL to 15 µg/dL suggests that an average slope of 3.23 IQ points per 10 µg/dL may be more likely. Furthermore, excluding dollar estimates that reflect other end points of harmful lead exposure means that these estimates may indicate only a fraction of the economic value of societal gains.

The single most influential parameter affecting the results reported in Table 2 is the discount rate, which influences the present value of future earnings. We used a 3% discount rate, which is regarded as the most appropriate rate to use for an economic analysis from the societal perspective (*25*). Use of a 5% discount rate in the calculation would cut these estimates by a little more than one-half: $53.8 billion, $104 billion, and $156 billion for the lower bound, base-case, and upper-bound estimates, respectively. Conversely, if one argues on the basis of intergenerational equity and sustainability that discounting unduly devalues long-term health risks, the estimates can also be recalculated using a discount rate of 0%. In that scenario, the totals range from $373 billion to over $1 trillion.

One limitation or source of understatement of benefits in this analysis is that we did not account for the economic value of avoiding other adverse health effects associated with exposure to lead. These include risk for hypertension, low birth weight, and infant mortality, which were included in the economic assessment by Schwartz (*6*). In our analysis, we also did not include more speculative estimates of the potential effects of lead exposure on criminal and delinquent behavior, as assessed by Nevin (*28*).

This analysis has other limitations. First, the estimates are conditional on the extrapolation of the effect of lead on IQ to lead levels below the range at which analyses were originally conducted. The estimates are based on studies of groups of children with BLLs > 5 µg/dL. The strength of the association at lower BLLs may be higher than the range of estimates used here (*17*). Second, the impact of childhood lead exposure on labor market outcomes has not been validated by a follow-up cohort study among young adults whose blood lead and IQ scores were measured in childhood. Such a study would be invaluable in clarifying a number of sources of uncertainty.

An important question is whether children's IQ scores have increased during this period in accord with our assumptions and calculations. Between 1976 and 1991–1994, geometric mean BLL decreased by 14.4 µg/dL (with an additional 0.7 µg/dL decrease through 1999). On the basis of these data, an effect size of blood lead on IQ from 0.185 to 0.323 implies an increase in average IQ level in U.S. children of 2.7–4.7 points between the late 1970s and early 1990s, other things being constant. If we use the lower estimate of a change in BLL of 12.3 µg/dL from 1976–1980 to 1991–1994, the implied change in average IQ is 2.2 to 3.9 points.

The periodic renorming of intelligence and ability tests to ensure that scores have a mean of 100 and standard deviation of 15 allows for analysis of trends in cognitive ability. When two versions of the same test are administered to the same group of children, average scores can be compared. The difference in scores can reflect changes in ability since the previous renorming or changes in test characteristics. Each renorming of IQ test scores in this century has shown substantial increases in test-taking ability. In particular, between the development of the first Wechsler Intelligence Scale for Children (WISC) test in 1947–1948 and that of the revised WISC (WISC-R) in 1972, average IQ scores rose by 8.5 points, or 0.35 points per year (*29*). The renorming of the WISC-III test in 1989 showed a further increase in average IQ scores by 9 points from 1972 to 1989, an increase of 0.52 points per year.

Another source of information is the 1992 renorming of the Cognitive Abilities Test (CogAT) administered to school children in grades 3–12 (*30*). Nevin (*28*) used this source to calculate age-specific changes in cognitive ability by comparing test scores on the 1984 and 1992 versions of the test for the same children. These data indicate that average scores, standardized like an IQ test on a mean of 100, rose from 1984 to 1992 by 4–5 points on average among school children 9–10 years of age. This age group tested in 1984 was younger than 6 years of age during the NHANES II period, when gasoline lead levels started their dramatic decline. Scores among 12- to 13-year-old children increased only slightly, and scores among 15- to 16-year-old children actually declined from 1984 to 1992 (*30*). Nevin (*28*) argued that older children tested in 1992 may not have benefited from reduction in lead exposure to the same extent because they were older when the decline in BLLs occurred.

The change in test scores in recent decades should be interpreted with caution. An attempt to apportion observed changes in test scores would require a fuller analysis of other, more important influences on cognitive ability. On the other hand, although these data do not prove an effect of the reduction in lead levels, they are consistent with what would have been expected if nothing but lead exposure had changed.

In conclusion, the benefits of measures to improve environmental health are often difficult to measure and value. Dollar values are difficult to place on adverse health effects. In the case of lead and impaired neurodevelopment, reduction in cognitive ability is the most easily measured outcome, but behavioral outcomes may be as important. Even ignoring other important outcomes of lead exposure, the valuation of cognitive ability is sufficient to demonstrate the substantial

magnitude of economic gains resulting from reducing the amount of lead in the environment to which people become exposed. Further, from the broader perspective of children's health and the environment, the specific example of lead in the environment raises the question of measuring the economic and health effects from other environmental health risks to which American children continue to be exposed.

## References and Notes


1. Pirkle JL, Brody DJ, Gunter EW, Kramer RA, Paschal DC, Flegal KM, Matte TD. The decline in blood lead levels in the United States. The National Health and Nutrition Examination Surveys. JAMA 272:284–291 (1994).
2. Pirkle JL, Kaufmann RB, Brody DJ, Hickman T, Gunter EW, Paschal DC. Exposure of the U.S. population to lead, 1991–1994. Environ Health Perspect 106:745–750 (1998).
3. National Research Council. Measuring Lead Exposure in Infants, Children, and Other Sensitive Populations. Washington, DC:National Academy Press, 1993.
4. Bellinger DC. Interpreting the literature on lead and child development: the neglected role of the "experimental system." Neurotoxicol Teratol 17:201–212 (1995).
5. Soong WT, Chao KY, Jang CS, Wang JD. Long-term effect of increased lead absorption on intelligence of children. Arch Environ Health 54:297–301 (1999).
6. Schwartz J. Societal benefits of reducing lead exposure. Environ Res 66:105–124 (1994).
7. CDC. National Report on Human Exposure to Environmental Chemicals. Atlanta:Centers for Disease Control and Prevention, 2001. Available: http://www.cdc.gov/nceh/dls/report/ [cited 8 July 2001].
8. Annest JL, Pirkle JL, Makuc D, Neese JW, Bayse DD, Kovar MG. Chronological trend in blood lead levels between 1976 and 1980. N Engl J Med 308:1373–1377 (1983).
9. Centers for Disease Control and Prevention. Blood lead levels in young children—United States and selected states, 1996–1999. MMWR 49:1133–1137 (2000).
10. Schwartz J. Low-level lead exposure and children's IQ: a meta-analysis and search for a threshold. Environ Res 65:42–55 (1994).
11. Pocock SJ, Smith M, Baghurst P. Environmental lead and children's intelligence: a systematic review of the epidemiological evidence. Br Med J 309:1189–1197 (1994).
12. Wasserman GA, Liu X, Lolacono NJ, Factor-Litvak P, Kline JK, Popovac D, Morina N, Musabegovic A, Vrenezi N, Capuni-Paracka S, et al. Lead exposure and intelligence in 7-year-old children: the Yugoslavia Prospective Study. Environ Health Perspect 105:956–962 (1997).
13. Lanphear BP, Dietrich K, Auinger P, Cox C. Cognitive deficits associated with BLLs < 10 µg/dL in US children and adolescents. Public Health Rep 115:521–529 (2000).
14. Tong S, Baghurst P, McMichael A, Sawyer M, Mudge J. Lifetime exposure to environmental lead and children's intelligence at 11–13 years: the Port Pirie cohort study. Br Med J 312:1569–1575 (1996).
15. Schwartz J. Beyond LOEL's, p values, and vote counting: methods for looking at the shapes and strengths of associations. Neurotoxicology 14:237–246 (1993).
16 Bellinger DC, Stiles KM, Needleman HL. Low-level lead exposure, intelligence and academic achievement: a long-term follow-up study. Pediatrics 90:855–861 (1992).
17. Lanphear BP, Canfield RL, Henderson CR, Cory-Slechta DA, Cox C. Environmental exposure to lead and children's intelligence at blood lead concentrations below 10 micrograms per deciliter [Abstract]. Pediatr Res 49(4):16A (2001).
18. Cawley J, Heckman J, Lochner L, Vytlacil E. Understanding the role of cognitive ability in accounting for the recent rise in the economic return to education. In: Meritocracy and Economic Inequality (Arrow K, Bowles S, Durlaf S, eds). Princeton:Princeton University Press, 2000;230–265.
19. Griliches Z. Estimating the returns to schooling: some econometric problems. Econometrica 45:1–22 (1977).
20. Needleman HL, Schell A, Bellinger D, Leviton A, Allred EN. The long-term effects of exposure to low doses of lead in childhood. An 11-year follow-up report. N Engl J Med 322:83–88 (1990).
21. Krupnick AJ, Cropper M. The social costs of chronic heart and lung disease. Paper QE 89-16. Washington, DC:Resources for the Future, 1989.
22. Salkever DS. Updated estimates of earnings benefits from reduced exposure of children to environmental lead. Environ Res 70:1–6 (1995).
23. Neal DA, Johnson WR. The role of premarket factors in black-white wage differences. J Polit Econ 104:869–895 (1996).
24. Bureau of Labor Statistics and Bureau of the Census. Annual Demographic Survey, March Supplement. Detailed Person Income (P60 Package). Available: http://ferret.bls.census.gov/macro/032001/perinc/toc.htm [cited 1 October 2001].
25. Lesser JA, Zerbe RO. Discounting procedures for environmental (and other) projects. A comment on Kolb and Scheraga. J Policy Anal Manage 13:140–156 (1994).
26. Gold MR, Siegel JE, Russell LB, Weinstein MC, eds. Cost-effectiveness in Health and Medicine. New York:Oxford University Press, 1996.
27. Grosse SD. Appendix: productivity loss tables. In: Prevention Effectiveness. A Guide to Decision Analysis and Economic Evaluation, (Haddix AC, Teutsch SM, Corso, PA, eds). 2nd ed. New York:Oxford University Press, 2002. In press.
28. Nevin R. How lead exposure relates to temporal changes in IQ, violent crime, and unwed pregnancy. Environ Res 83:1–22 (2000).
29. Flynn JR. IQ trends over time: intelligence, race, and meritocracy. In: Meritocracy and Economic Inequality (Arrow K, Bowles S, Durlaf S, eds). Princeton, NJ:Princeton University Press, 2000;35–60.
30. Thorndike R, Hagan E. CogAT Research Handbook. Chicago, IL:Riverside Publishing Co., 1997.