ENVIRONMENTAL RESEARCH 66, 105–124 (1994)

Societal Benefits of Reducing Lead Exposure

JOEL SCHWARTZ

Department of Social and Preventive Medicine, University of Basal, Steinengraben 49, CH4051, Basel, Switzerland

Received July 5, 1993

While sophistication in public health research has been increasing substantially in the past few decades, sophistication in decision making about public health and environmental issues has not been increasing in parallel. Measures that are inexpensive tend to be implemented and measures that are expensive tend not to be implemented by makers of public policy. That is often independent of the degree of public health protection afforded by the measures. Understanding and addressing this pattern is crucial to the control of lead exposure of critical populations. People are still exposed to lead in our society not because anyone believes that exposure is good, but because reducing exposure costs money. Maintaining exposure also has its costs, however. It is more difficult to measure them, and they are often ignored in decision making—but they are not small, and attempts to measure them have been made. The high cost of reducing lead exposure of critical populations is the reason that progress in reducing lead-paint exposure has been minimal in the 18 years since the passage of the Lead-Based Paint Poisoning Prevention Act and that it took from the time of the initial proposal in 1973 until 1986 before lead was substantially eliminated from gasoline. In its 1986 rule making, the EPA estimated that the elimination of lead from gasoline would cost more than $500 million per year. Removing leaded paint is estimated to cost billions of dollars. The difference is that the EPA promulgated its rule of removing lead from gasoline, whereas HUD has had little success in removing leaded paint from housing. One reason that the EPA was successful in implementing such an expensive regulation was that it provided detailed estimates of the health and welfare benefits that would accrue and the monetary value of some of the benefits. The EPA cost–benefit analysis demonstrated that the monetary benefits of its regulation far exceeded the costs. That neutralized the cost issue and focused the debate over the regulation on questions of timing. A detailed benefit analysis of reducing lead in drinking water has caused the EPA to consider tighter water lead standards than initially envisioned. Despite years of concern about the consequences of leaded paint poisoning, children continue to be poisoned by leaded paint because it will cost billions of dollars to abate the hazard, and demand for these dollars has lost out to competing needs. As long as attention focuses on the costs of lead-paint abatement and ignores the costs of not abating and as long as people add up the costs of removing paint but not the costs of medical care, compensatory education, and school dropouts, substantial action is unlikely. It is possible that a detailed benefit analysis of lead-paint removal will not show that benefits exceed the costs, but we think it unlikely, given the large benefits estimated for other programs that reduce lead exposure, that a cost–beneficial removal strategy cannot be found. If no attempt is made to estimate the benefits, this strategy is less likely to be adopted. This paper cannot reasonably estimate the costs and benefits of the many measures that are available to reduce lead exposure of critical populations. It can, however, describe the methods that have been used and present a prototypical analysis that can readily be adapted to develop analyses specific to individual actions.

0013-9351/94 $6.00

INTRODUCTION TO BENEFIT ANALYSIS

This paper is not meant as a detailed discussion of the methods and issues involved in calculating the social benefits of a government policy, but rather as a brief introduction to the basic issues. Often, not all the costs and benefits of providing a good or service are captured by the participants in the transaction. For example, the damage done by air pollution is not reflected in the price of the goods made by polluting industries. Such externalities are a prime justification of government action. To estimate the monetary benefits of a policy that reduces pollution, for example, one need to assign monetary values to the impacts of the pollution. Classical economic theory assigns no inherent value to any good or service, but values it in accordance with people's willingness to pay for it. That is in contrast with the just-price theory of medieval Europe or the labor theory of value in Marxism. This analysis will be restricted to the classical approach. Where a well-functioning market (with minimal externalities) exists, the price of a good or service is taken as its value. In some cases, an object is not sold directly but is an attribute or component of other goods and services. If market prices are available for them, the value we seek can be estimated with regression techniques and what is generally referred to as hedonic-price theory. Griliches (1961) provides the classic example of valuing attributes of automobiles to determine how much of the increase in the price of automobiles is inflation and how much reflects quality change (as valued by consumers, not manufacturers). It is also possible to use survey techniques (referred to as contingent-valuation surveys) to estimate the value of a nonmarket good or service. Such research can be difficult in practice, but reasonable estimates have been obtained for some environmental goods.

A good theoretical framework has been developed for the household-production function approach. In this approach, a theoretical model of the benefits or utility that a person gains from possessions, including health, is derived. The model assumes that people allocate their money and time to maximize their utility, given a preference for goods and health. The important contribution of this model is the recognition that people engage in defensive expenditures of time (e.g., by jogging) or money (e.g., by purchasing water filters) to reduce their risk of disease. Under some simplifying assumptions, the marginal willingness to pay to avoid the risk of illness by reducing environmental exposure consists of the sum of the marginal avoided medical-care costs, avoided loss of wages, willingness to pay to avoid the disutility of illness, and the marginal reduction in defensive expenditures allowed by the reduction in exposure. Benefit approaches that include only the value of avoiding the illness (based on the observed dose–response relationship) tend to underestimate benefits because the observed dose–response relationship has been reduced by defensive expenditures. A recent study of the effects of air pollution on asthmatics in Los Angeles documented that asthmatics reacted to forecasts of high air pollution by remaining indoors. Basing benefit estimates on the observed number of asthmatic attacks would ignore the disruption in the lives of asthmatics who curtailed their activity in anticipation of exposure. Similarly, parents with children who are doing poorly in school might spend

more of their spare time in helping children with their schoolwork. The observed relationships between lead and cognitive and educational outcomes do not provide data to estimate those possible costs.

Nevertheless, most benefit analyses of government regulations and policies fall back on resource costs and human capital measures as proxies for the value of the benefits derived. For example, medical costs can be used as a proxy for the value of avoiding an illness. It must be stressed that such proxies are usually large underestimates of the true value of avoiding illness.

An early analysis by Provenzano (1980) quantified the costs of treating the severe cognitive impacts of high-dose lead exposure and found them substantial. More recent studies have focused on lower dose, but more common exposures and have tried to quantify the avoided costs associated with reduced lead exposure in several categories: medical costs, educational costs, productivity reduction, and morality-risk increase. The first broad analysis of this kind was by J. Schwartz of EPA and co-workers, who performed a cost–benefit analysis of alternative concentrations of lead in gasoline (Schwartz *et al.*, 1985). The benefits were valued by using avoided medical costs and avoided costs of compensatory education for children with blood lead concentrations over 25 μg/dl and avoided medical costs and lost wages for the cardiovascular effects expected from increases in blood pressure. In addition, the value of reduced mortality risk from cardiovascular disease was taken to be the value of reduced risk of accidental mortality implied in wage differentials (Violette and Chestnut, 1983). The analysis was reviewed and approved by the Office of Management and Budget. Numerous health effects were not monetized, but extrapolation from their study indicates that a 1 μg/dl decrease in mean blood lead concentrations in the population would produce at least $3.5 billion per year in benefits from reduced health effects of lead.

The gasoline lead benefit analysis ignored cognitive effects of exposure to lead concentrations under 25 μg/dl. Although cognitive effects at lower concentrations are smaller, they involve much larger populations. Small decrements in IQ around the mean have been linked to lower wages in numerous studies (see Barth *et al.*, 1984, for a review). The use of lost wages as a proxy for the cognitive damages of low-dose lead exposure was first suggested by Schwartz (1982). The idea was developed as part of a benefit analysis of reducing lead in drinking water (Levin, 1986). The study indicated that the lost wages avoided by lowering the mean blood lead concentration by 1 μg/dl add $1 billion per year, on an annualized basis, to the benefits estimated in the gasoline lead study. The benefits of using corrosion control to reduce lead concentrations in drinking water appeared to be over four times the cost, for example (Levin, 1986). Cost-effective approaches to reduce exposure to lead in paint, imported food, ceramics, and soil deserve intensive examination in light of these estimates of the societal costs that could be avoided.

Those methods were also used to evaluate the benefits of alternative National Ambient Air Quality Standards by EPA (1989). Both the health effects that were valued and the methods for monetizing them were reviewed by a special panel of EPA's external Clean Air Science Advisory Committee that included health sci-

entists and economists. The committee, in approving the analyses, expressed concern about underestimation caused by failure to monetize many health effects, in particular the effects of lead on birthweight, and failure to estimate willingness to pay to avoid the activity restrictions associated with illness, particularly in the case of stroke.

A more recent report for the EPA (O'Neal and McGartlund, 1989) updated and expanded on the previous studies. In particular, using data from the U.S. Public Health Service, the authors showed that infant mortality rates are not merely increased for low birthweights (less than 2500 g) but vary continuously across all birthweight categories. Hence, if lead is associated with reduced birthweight, then lead exposure would be expected to result in increased infant mortality. The relative risk is small and impossible to detect in studies as small as the prospective lead studies. However, because of the large numbers of subjects exposed, the attributable risks can be larger than the mortality risks for many environmental carcinogens. That benefit analysis was also peer-reviewed by EPA's external Science Advisory Board.

ESTIMATED BENEFITS OF A 1 μg/dl REDUCTION IN POPULATION MEAN BLOOD LEAD CONCENTRATIONS

It is beyond the scope of this report to examine specific policy alternatives for lowering blood lead concentrations in detail and to quantify their benefits. However, an estimate of the monetary benefits of reducing population mean blood lead concentrations is appropriate because it can be a major resource for analysts who wish to examine such policies and a stimulus to such examination. Presented here is a prototype analysis of the benefits of reducing the population mean blood lead concentration by 1 μg/dl, leaving the geometric standard deviation constant. Our analysis follows and expands on those cited above, incorporating new health data where appropriate. Obviously, the benefits are subject to considerable uncertainty. However, the lack of good methods for monetizing many of the health benefits of reduced lead exposure, coupled with some conservative assumptions, and the trend toward finding new and more serious health effects of lead at lower and lower concentrations make the estimates more likely low than high. Methods adopted from the earlier studies are summarized briefly below; where new categories of benefits or methods are adapted, they are described in more detail.

To estimate the effects of changes in blood lead concentrations, we need a baseline distribution. The best available data are from the second National Health and Nutrition Examination Survey (NHANES II). However, blood lead concentrations were falling rapidly during the period of the survey (1976–1980) because of the reduction in lead in gasoline (Annest *et al.*, 1983; Schwartz *et al.*, 1985; Schwartz and Pitcher, 1989). Using such a high baseline risks overestimating the benefits of a 1 μg/dl reduction. Hence, I have taken as a baseline the blood lead distribution for 1984 projected by the Agency for Toxic Substances and Disease Registry (Mushak and Crocetti, 1990).

Children with high blood lead concentrations receive medical attention, and

reducing the blood lead concentration of the population will reduce the number of children who require that attention. I have used data from Piomelli *et al.* (1984) to estimate the fraction of children in different blood lead ranges who require chelation and other treatment. The recommended treatment regimens were outlined in that paper and adopted by the Centers for Disease Control. New guidance has since been issued by CDC with a lower threshold for screening and other activity but not for chelation. The expected costs of treatment in the different ranges of blood lead concentrations have been estimated previously (Schwartz *et al.*, 1985; Levin, 1986). I have updated the costs to 1989 dollars by using the medical-care component of the Consumer Price Index. They come to $1300 per child above 25 μg/dl. Shifting the distribution of blood lead concentrations downward would lower the number of children with blood lead concentrations above 25 μg/dl by 145,000 and avoid $189 million in medical care costs.

Costs of Cognitive Damage in Children

Children with high lead concentrations are likely to show substantially decreased school performance that can require reading or speech therapy or the attention of a school psychologist. The costs of such treatment can be large. de la Burde and Choate (1975) reported a relative risk of 7 for poor academic progress and a relative risk of 4 for repeating a grade in a 3-year follow-up, for instance. Bellinger *et al.* (1984) reported data that suggested that 17% more high-lead than low-lead children were receiving daily assistance outside the classroom. Needleman *et al.* (1990) recently reported an odds ratio of 5:8 for reading disability in their high-lead group. Lyngbye *et al.* (1990) also reported an odds ratio of 4:3 for learning disability in children with circumpulpal dentin lead above 16 ppm—a significantly lower cutoff than that of the Needleman *et al.* high-lead group. On the basis of such data, previous benefit analyses have estimated that 20% of lead-poisoned children (over 25 μg/dl) would require assistance from a reading teacher, school psychologist, or other specialist for an average of 3 years, and no children with lower blood lead concentrations would require assistance. I have maintained that estimate. Costs taken from a study for the U.S. Department of Education (Kakalik, 1981), when updated to 1989 dollars with the Consumer Price Index, yield an estimated cost of $3320 per child over 25 μg/dl.

Most children with lead-related cognitive deficits do not require such educational assistance, but their losses can still be substantial in monetary terms. Impaired cognitive function can reduce the productivity of a person in society. That reduction can be used as a proxy for the cost to society of the impairment. It is clearly an underestimate, in that it puts no value on losses that are not directly related to productivity. Moreover, the neurotoxic effects of lead involve outcomes besides IQ decrements. Effects on hearing, balance, hyperactivity, and perceptual and attention disorders are more difficult to associate with a monetary loss than are IQ decrements. That does not mean that they are trivial, particularly when summed over millions of children. The lack of a benefit method should not, by default, become a judgment of significance.

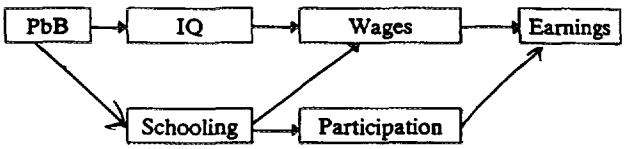

FIG. 1. A schematic diagram of the pathways by which childhood lead exposure might influence lifetime earnings.

A considerable body of literature has examined the relationship between IQ and earnings, for instance. The studies were generally parts of investigations of the economic returns resulting from schooling. It was necessary to control for IQ differences. There is usually a positive correlation between IQ and years of schooling, so failure to control for IQ could bias the estimates of the impact of schooling (Griliches, 1977). Hence, as part of the studies, the marginal impact of IQ on earnings was generally also estimated. Those studies were performed in general population samples (e.g., Project Talent) and yielded estimates of the impact of small changes in IQ near the mean, not for large decrements that led to serious impairment. The small changes are similar in magnitude to the ones associated with low-dose lead exposure. The studies have been reviewed by Barth *et al.* (1984), who found general consistency among them that suggested that a 1-point IQ deficit was associated with a 0.9% reduction in earnings, if other factors were held constant. The Barth *et al.* report was favorably reviewed by a panel of experts. Its estimates have two components: a direct effect of IQ on earnings and an indirect effect—i.e., IQ affects number of years of schooling, which affects earnings.

Such multiple pathways are usually estimated with structural equation modeling (Griliches, 1977). However, lead has CNS impact in addition to its effect on IQ. It has been associated with disturbances in auditory and visual-evoked potentials, with attention span deficits, and with minor losses in hearing and balance. Those disturbances might show up as a greater impact of lead on schooling then would be predicted from the association between lead and IQ. In any case, a direct estimate of lead's impact on schooling is to be preferred.

The potential effects of lead on lifetime earnings are illustrated more fully in Fig. 1. Lead can reduce IQ and thus result in lower wages and lower lifetime earnings. Lead can also reduce educational achievement, which has been linked to reduced wages and reduced participation in the work force, both of which, in turn, reduce lifetime earnings. Algebraically, if

$$E = P \cdot W,$$

where E = earnings, P = participation rate in the labor force, and W = wage rate, then

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

For small changes, we can compute the percentage change expected in the mean wage rate and mean work force participation rate and, given an estimate of lifetime earnings, compute the change expected. To perform the calculation for a change in mean blood lead concentration, we need the present value of lifetime earnings, estimates of the effects of lead on IQ and schooling, and estimates of the effect of IQ and schooling on work force participation and wage rates. These are discussed later.

Because I am assessing the impact of a permanent reduction in blood lead concentrations, it is not necessary to deal with the question of reversibility. To avoid double counting and complicated accounting issues, I have computed the annual benefits as the net present value of the increased earnings expected from a 1-year age cohort because of the reduction in blood lead concentrations. I have taken the cohort to be the cohort of children turning 6 years of age each year because the lead–IQ studies have generally been done in school-age children and the Bayley scores for infants used in the prenatal studies will not necessarily show the same correlation with earnings. I have taken the size of the 6-year-old cohort to be 3.9 million, about its size in the mid-1980s.

Calculating the net present value of lifetime earnings requires a number of assumptions. Dollars available in the future are less valuable than if they were available today. Viewed alternatively, a $1000 deposit generates a stream of revenue (interest payments) into the future that sum to more than $1000. The difference is the interest rate, which reflects the value to the bank of having the money today instead of in the future. The choice of the appropriate rate of discount has been contentious and is beyond the scope of this report. Conceptually, I have adopted the opportunity cost of capital approach with eventual cost recovery (Kolb and Scheraga, 1989), which we have estimated at 5% real (i.e., 5% above the inflation rate). The risk-free rate of return would yield a lower discount rate (and hence a higher benefit estimate).

It is also necessary to make assumptions about wage rates in the future. The simplest assumption is that today's distribution of earnings by age, educational concentration, and sex continues into the future or grows at a constant rate across all age, education, and sex classes. Historically, real wage increases have been about 2% per year, but that growth has fallen in the past decade. That might be an effect of the large baby-boom cohort on the labor markets, and some believe that faster wage growth will resume. I have assumed 1% real wage growth in the future from the 1987 distribution of income. That is conservative in several regards. It assumes no increase in the percentage of the work force with college educations, whereas the younger age groups in 1987 had higher educational levels. It assumes 1987's ratio of female to male earnings, whereas the ratio has increased from 0.6 to 0.7 in the past 15 years. It assumes the same rate of female participation in the work force as in 1987, whereas the participation has continued to rise and, as already noted, the assumed productivity increase is probably an underestimate.

Many adults do not participate in the work force or do not participate for all their potential working years. The largest group is women who remain at home doing housework and rearing children. Such work has value: homework has been

valued in economic studies with the use of either opportunity costs (the value of foregone income) or the market value of substitute labor for homework. The opportunity cost is usually taken as the average wage earned by persons of the same age, sex, and educational level. That is probably too high, inasmuch as the employed members of the cohorts, on the average, have more work experience, training, and more relevant education than those who remain at home. The market value of substitute labor is often too low, inasmuch as many of the substitute workers have lower educational attainment than the persons they would replace. The true value is probably between the two estimates.

Given an estimate of the value of the work, can we conclude that the impact of lower IQ on productivity is the same as for market wages? Supporting an impact on child rearing is the association between maternal IQ and child IQ, which is unlikely to be entirely hereditary. Moreover, HOME score, a measure of the quality of the home-rearing environment, has been positively correlated with both maternal IQ and child IQ in the recent lead studies. These results suggest that there is an impact, at least on the child-rearing component of home work. I have taken the value of lost productivity due to lead exposure for nonparticipants in the work force at half the value for employed workers, which I believe is a conservative estimate. Data on the present values of expected lifetime earnings under these assumptions were obtained from the U.S. Bureau of the Census. From these, I calculated a net present value discounted to age 6 of $301,000.

A recent long-term follow-up by Needleman *et al.* (1990) has provided the opportunity to estimate directly the effect of lead on schooling. Their follow-up of their original elementary school cohort after the cohort would have graduated from high school showed an odds ratio of 7:4 for the risk of dropping out of high school. Their regression coefficients for the effect of tooth lead on grade achieved estimated the current grade achieved, not the expected grade. However, some of the children were in college and were expected to attain a higher grade level. After adjusting the published results for the fact that a higher percentage of the low-tooth-lead children were attending college, I estimated about a 0.59-year difference in expected maximal grade achieved between the high- and low-lead groups. Because the Needleman *et al.* analysis used tooth lead, not blood lead, as the exposure marker, we need to convert the results. I have assumed that the differences in grade achieved and in probability of graduating from high school scale proportionately with the IQ effect. Again, note that the effect on schooling is only one of the adverse outcomes of the neurotoxicity of lead.

Estimates of the direct effect of IQ on earnings range from an earnings change of about 0.2 to 0.75% for an IQ change of 1 point (Barth *et al.*, 1984). The structural equation models of Griliches yield estimates of a little over 0.5%. Because that method has conceptual advantages and 0.5% is roughly the median estimate, I have used that value in our benefit estimate.

Studies that allow estimates of the effects of schooling on earnings are less common. In structural equation models, Chamberlain and Griliches (1977) estimated that a 1-year increase in schooling would increase wages by 6.4%. In a model with a similar specification, Olneck (1977) reported a 4.8% increase. Gril-

iches (1977) reported a 5.2% increase in a sample not restricted to brothers but a 9.8% increase if educational attainment is taken to be endogenous. All those studies used wages ascertained at one time. In a longitudinal study of 799 subjects over 8 years, Ashenfelter and Ham (1979) reported that an extra year of education increased the average wage rate during the period by 8.8%. I have taken a 6% change to be a reasonable, and slightly conservative, estimate of the effect. Using the changes in expected grade achieved from Needleman *et al.* (1990), I estimate that a lead exposure sufficient to provide a 1-point IQ deficit would reduce wages by 0.79%.

Multiplying the IQ–earnings effect by the estimated impact of a unit change in blood lead on IQ and the education–earnings effect by the estimated impact of a unit change in blood lead on grade attained gives a revised estimate of the effect of lead on child's future wage rate. Previous benefit analyses have stopped here and multiplied predicted percentage changes in wages by the net present value of the population's expected lifetime earnings. However, that assumes that lead changes only wages and does not affect participation in the workforce. But failure to graduate from high school is correlated with participation in the work force, principally through higher unemployment rates and earlier retirement ages, and lead is a strong correlate of attention-span deficits, which would also reduce work force participation. Although we have no direct data for estimating the impact of attention deficits on workforce participation, we can use the results of the Needleman *et al.* (1990) study that related lead to failure to graduate and estimate changes in earnings due to reduced workforce participation.

The 1978 Social Security Survey of Disability and Work (U.S. DHHS, 1981) obtained information on work-force participation, presence of disability or illness, age, education, and socioeconomics from a representative sample of working-age subjects. I have derived estimates of changes in work-force participation between high school graduates and nongraduates from the analysis of those data by Krupnick and Cropper (1989), which controlled for age, marital status, number of children, race, region, and other socioeconomic and medical factors. According to those data, average participation in the work force is reduced by 10.5% for persons who fail to graduate from high school. Using the odds ratio from Needleman *et al.* (1990), I estimate that a lead exposure sufficient to produce a 1-point IQ deficit would result in a 4.5% increase in the risk of failing to graduate. The mean impact is therefore a 0.47% reduction in expected earnings.

It is possible that such an analysis overcontrols for other factors in estimating the effect of schooling. For example, high school dropouts are more likely to work in occupations with a higher risk of disability (which was included as an independent variable in the regression). A more parsimonious approach is to look at the current population survey (CPS), stratified by age. I have examined the data from the 1978 CPS stratified by age groups between 25 and 65 years and found a rather consistent 20% difference in mean hours worked between persons with less than a high school education and those with high school or more. That difference combines reduced participation in the work force and reduced hours worked by participants and suggests a reduction of 0.9% in expected earnings. To be con-

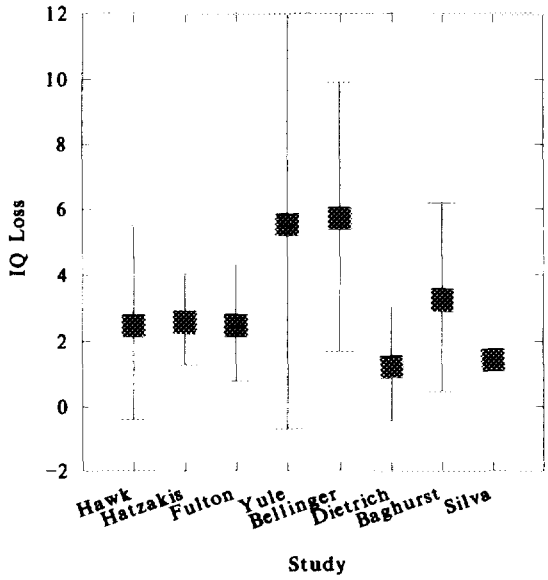

FIG. 2. The estimated effect on a child's IQ for a decrease in blood lead level from 20 to 10 μg/dl from each study in the metaanalysis. The 95% confidence intervals are also shown. [Reproduced by permission of the publisher, from Schwartz (1994).]

servative, I have used the results derived with the regressions in Krupnick and Cropper as the best estimates for our purposes.

The last estimate needed is a lead–IQ dose–response relationship. A number of recent studies have examined this relationship. A recent metaanalysis (Schwartz, 1994) of these studies reports an overall slope of 0.245 IQ points per microgram per deciliter. Figure 2 shows the estimated effect of a 10 μg/dl increase from those studies. A test for heterogeneity was insignificant, indicating that the variation in the slopes between studies was no more than would be expected, given the standard errors of the regression coefficients within the study. Using this slope coefficient, we can estimate that a permanent reduction in blood lead concentrations of 1 μg/dl will produce a net present value benefit of $1300 per child for the cohort turning 6 years of age each year, for a total benefit of $5.06 billion per year. In addition, there are probably large social benefits beyond the private benefits.

Costs of Fetal Effects of Lead

As reviewed elsewhere (NRC, 1993; Schwartz, 1992a), prenatal lead exposure has been linked to reduced gestational age, lower birth weight, and possibly fetal loss, as well as reduced performance on infant cognitive tests and reduced growth, if followed by increasing postnatal exposure. I am not estimating benefits of cognitive damage, to avoid double counting with the postnatal estimates. Low birth weight and low gestational age are strong predictors of respiratory distress

syndrome, other infant illnesses, and infant mortality, however. Moreover, the relationships are not limited to very-low-birth-weight infants. For instance, in 1983, the all-race infant mortality rate per 1000 live births was 4.0 at a gestational age of 40 weeks. At 37–39 weeks, the infant mortality rate was 5.5 per 1000, an almost 40% increase. Using data from the Linked Birth and Infant Death Record Project (National Center for Health Statistics), we can estimate the infant mortality rate as a function of gestational age. I have chosen gestational age, rather than birth weight, because the association between lead and gestational age is stronger than for birth weight. Given an estimate of the impact of lead on gestational age, which we have taken from Dietrich *et al.* (1986), we can estimate the impact of a 1 μg/dl change in maternal blood lead on gestational age, and hence infant mortality. That yields a predicted reduction of 380 deaths per year for the U.S. population—a small reduction compared with the total number of infant deaths but large in comparison with the estimated annual deaths associated with most environmental carcinogens. Lead has also been linked to stillbirths in a number of studies (Fahim *et al.*, 1976; Wibberley *et al.*, 1977); however, because the evidence is not complete and no study provides a dose-response function, I have not computed any benefits of avoiding fetal loss.

Valuing reductions in mortality is highly controversial. The U.S. Department of Transportation has used lifetime wages as a proxy—an approach also common in litigation. That approach has obvious faults. The value of reducing early mortality in retired persons and housewives is not zero, for instance. It also has the theoretical drawback of not incorporating people's willingness to pay.

Most people are willing to spend any amount of money to prevent certain death, so the willingness-to-pay approach at first seems no better. However, what is being valued is not the avoidance of certain early death in an identified subject, but a small reduction in the risk of death spread among many subjects. People make decisions that trade off small changes in the risk of premature mortality against cost all the time, for instance, when they decide how many smoke detectors to put in their homes.

Several studies have estimated willingness to pay on the basis of implicit tradeoffs between risk and dollars revealed in market transactions. Most (e.g., Smith, 1976; Thaler and Rosen, 1976; Viscusi, 1978) have used labor markets on the premise that, all else being equal, workers must receive higher wages to accept a higher risk of being injured or killed on the job. Such studies typically regress wages on risk and a variety of other explanatory variables (e.g., educational concentration, unionization, worker experience, region of the country, and working conditions). The coefficient of the accidental death rate (deaths per 10,000 workers) gives an estimate of the willingness to pay to avoid an increased risk of death of 10^{-4}. All the studies have limitations. Selection bias is a major limitation, in that the most risk-averse members of society usually are not employed in any of the (usually blue-collar) occupations studied. That leads to underestimates of the overall societal willingness to pay to avoid risk. Imperfect knowledge by the workers of the risks that they face adds considerable noise to the estimates. A review of the earlier studies (Violette and Chestnut, 1983) found estimates of the

value of a statistical life ranging from $0.5 to $7 million among the reasonable studies, with most between $1 and $5 million. The studies with low estimates were based on actuarial risk estimates derived from insurance data on broad occupational categories. The higher estimates were based on more detailed data derived from Bureau of Labor Statistics surveys, which included only work-related fatalities and provided more detailed data.

More recent studies (e.g., Marin and Psacharopoulos, 1982; Dillingham, 1985) show estimated values of $2–9 million per statistical life and, as expected, show a higher value ($9 million) for nonmanual workers. One potential problem with such an analysis is that workers' perceptions of risk can differ from their measured risk. At a minimum, that difference will introduce noise into the relationship; if the perceptions differ systematically from reality, then they can introduce bias as well. One study has shown that chemical workers' perceptions of their occupational risk were very similar to their actual risk (Viscusi and O'Connor, 1984). One labor-market study avoids the problem by using a survey of workers that collected data on their perceived risk, individual wages, etc. (Gegax *et al.*, 1991); it produced an estimate of $1.9 million per life.

An alternative approach is to measure willingness to pay through the use of survey instruments that present realistic scenarios of trade-offs between expenditures and reduced mortality risk. Jones-Lee *et al.* (1985) found average value of statistical life estimates, based on willingness to pay for measures to reduce traffic fatalities, of $1.6–4.4 million in Great Britain. Incomes are lower in Great Britain than in the United States, so one would expect somewhat higher results in the United States. A similar contingent valuation study by Gegax *et al.* (1991) yielded estimates of $2.4–3.3 million. Those studies have been reviewed extensively (Fisher *et al.*, 1989; Violette and Chestnut, 1989). All the estimates are considerably larger than the present value of lifetime wages—a conclusion that makes theoretical sense. I have taken $3 million per statistical life as the best estimate of the willingness to pay to avoid excess mortality risk. It is not obvious that the willingness to pay to avoid excess mortality of children is the same as it is for adults. However, because the number of years of potential life lost is greater for children, we have assumed that people are willing to spend at least as much to reduce risk for their children as for themselves. Under that assumption, the monetary benefit associated with reduced infant mortality is $1.14 billion per year.

Mortality is the most severe, but least common, sequela of low gestational age or low birth weight. Respiratory distress syndrome, retinopathy, and less serious illness are much more common. Reducing lead concentrations, and hence increasing gestational age, will reduce the rates of those outcomes as well. I have been unable to obtain data for estimating their occurrence as a function of gestational age. However, the Office of Technology Assessment has estimated that 5% of all births result in an admission to neonatal intensive-care units in the United States, or about 4.5 admissions per infant death. Assuming that the ratio holds constant for small changes in gestational age, reducing mean lead concentrations should result in 1700 fewer infants requiring such care. At a cost of $39,000 per case, that will produce a cost savings of approximately $67 million. I am unable to estimate

cost savings for less serious, but more common, complications of early gestational age.

Costs of Effects in Adults

The relationship between blood lead and increases in blood pressure and cardiovascular disease has been discussed in detail including a metaanalysis of 16 studies (NRC, 1993; Schwartz, 1992b; Schwartz, in press). Reducing blood lead concentrations can be expected to reduce the number of men and women with hypertension and to reduce the risk of myocardial infarction, stroke, and early death in the adult population. Substantial benefits can be expected to accrue from even minor reductions in such prevalent diseases. The general methodology we use to estimate benefits has been discussed in detail (Schwartz *et al.*, 1985; Levin, 1986; O'Neal and McGartlund, 1989). Benefits are calculated for reduced medical-care expenditures due to hypertension, myocardial infarctions, and strokes. To those are added the value of lost workdays. Excess mortality risk is valued by using the market value of risk reduction discussed above.

Estimates of reductions in the numbers of myocardial infarctions, strokes, and deaths due to reductions in blood pressure were made with the logistic risk coefficients from the Framingham study. The blood-pressure change for a change of 1 μg/dl in blood lead was taken to be one-fifth of the change in diastolic blood pressure that was associated with a 5 μg/dl change in blood lead. Cardiovascular risks depend on factors in addition to blood pressure. Because the logistic risk equations are nonlinear, the impact of a change in blood pressure varies somewhat with the magnitudes of the other factors. I have dealt with that by using a simulation model. NHANES II contains a representative sample of the U.S. population with information on all the other risk factors (smoking, age, serum cholesterol, etc.). The impact of the change in blood pressure was simulated with these data. Further details of the method are given in Schwartz *et al.* (1985).

On the basis of the models, I have estimated that a 1 μg/dl reduction in blood lead concentrations will result in approximately 3200 fewer myocardial infarctions per year, 1300 fewer strokes per year, and 3300 fewer deaths per year. In addition, 635,000 fewer persons will suffer from hypertension.

Krupnick and Cropper (1989), using data from the National Medical Care Expenditure Survey (NCHSR, 1981), have estimated medical costs of hypertension. I inflated these to 1989 dollars by using the medical-care component of the Consumer Price Index to obtain an estimate of $550 per year. The costs include those for physicians, drugs, and hospitalization. In addition, hypertensives have more bed-disability days and work-loss days than others of their age and sex. Krupnick and Cropper estimated the increase in work-loss days at 0.8 per year. Valuing those at the mean daily wage gives an estimated additional annual cost of $78. Details of the method are given in Krupnick and Cropper (1989), Schwartz *et al.* (1985), and O'Neal and McGartlund (1989). Note that this treatment values the medical costs of hypertension only for the fraction of hypertensives who take medication and so underestimates the benefits of reducing the number of cases.

Hypertensive medication has serious side effects, and the value of avoiding them would also be considerable but is not estimated here.

For myocardial infarction, I have taken the estimated medical costs from Schwartz *et al.* (1985). Those costs, derived on an incidence basis, were derived from earlier estimates of Hartunian *et al.* (1981), with some changes to reflect changes in medical technology. I have merely inflated them to reflect current prices. The estimate is $23,000 per case; this is almost certainly an underestimate, in that it does not incorporate the large increase in various surgical treatments for cardiovascular disease of the past 20 years. For example, Krupnick and Cropper (1989) have estimated costs for treatment of all ischemic heart disease to be twice the Hartunian *et al.* estimate, using data from the National Medical Care Expenditure Survey. Their data did not allow a breakdown that focused exclusively on myocardial infarction, however. In addition, Schwartz *et al.* (1985) provided estimates, again based on Hartunian *et al.* (1981), of loss of earnings because of victims' reduced participation in the work force. I have revised the estimates again to account for the broader age group (20–74) being considered here and to include women. Both changes lower the average loss of earnings, which are estimated at $21,000 per incident case. For stroke, the same method yields an average estimate of $30,000 per incident case, including wages and medical expenses.

Combining those estimates, we can expect the following benefits: $141 million in reduced costs for myocardial infarction, $39 million in reduced costs for stroke, $399 million in reduced costs for hypertension, and $9.9 billion in benefits associated with reduced mortality risk.

TABLE I

	Benefits, millions of dollars
Children	
Medical costs	189
Compensatory education	481
Earnings	5,060
Infant mortality	1,140
Neonatal care	67
Total	6,937
Adults	
Medical costs	
Hypertension	399
Heart attacks	141
Strokes	39
Lost wages	
Hypertension	50
Heart attacks	67
Strokes	19
Mortality	9,900
Total	10,215
Grand total	17,152

TOTAL BENEFIT

Combining the benefits from the categories that were monetized yields an estimate of approximately $17.2 billion dollars per year as the benefit of a 1 μg/dl reduction in blood lead concentrations across the population. The breakdown of these estimates is given in Table 1.

LIMITATIONS IN THE ANALYSIS

Table 2 lists important assumptions and the likely direction of bias. The most important limitation of the analysis is the large number of effects of lead for which no monetary value has been assigned, including reduced growth rate and stature, hyperactivity, possible hearing impairments, and reduced sense of balance, as well as the pain and suffering associated with medical treatment, poor school performance, and the profound activity limitations associated with heart attacks and strokes. Lost wages clearly do not capture the costs associated with the decreased worker satisfaction associated with lower-skill jobs. Impacts on the parents of lead-affected children have not been quantified. All those factors suggest that benefit estimates are low.

Factors that might suggest overestimates include the possibility of thresholds for some of the effects. Studies such as those of Fulton *et al.* (1987) and Schroeder and Hawk (1987), which have plotted the IQ–lead relationship across most of the range of normal exposure, do not suggest such thresholds, as is illustrated in Figs. 1 and 2. Similar remarks hold for the cardiovascular effects, although again there is a suggestion that the slopes are in fact higher at lower blood lead concentrations. The values assigned to reduced mortality risk remain controversial, but even the low estimates from wage studies suggest numbers no lower than the value of a statistical life. I have assumed that increases in blood pressure produce the expected increases in cardiovascular disease seen in all the prospective cardiovascular epidemiology studies. That might not be true. All trials of hypertensive medication have not produced the expected effect. However, such medication has substantial side effects, including disturbances in potassium metabolism, which could account for the lower than expected effect. Lower blood lead concentrations do not have such side effects; the optimal blood lead content is zero. Some studies have directly associated lead with cardiovascular disease, but not necessarily with exactly the slopes of the Framingham study. In contrast, I have assumed that lead affects cardiovascular disease only through blood pressure, whereas some animal data (Revis *et al.*, 1981) suggest that it also increases serum cholesterol.

Similarly, we cannot be sure that the earnings effect of a lead-related reduction in IQ is the same as the effect of naturally occurring reductions. The behavioral studies—which suggest reduced attention time, more time out of seat in class, etc.—imply that the effect is greater.

In general, no evidence suggests that the slopes that we have used are not equally likely to be too low and too high. Hence, the estimates of monetized benefits might be of the right general magnitude, but not right. The large number

TABLE 2

Factor	Assumption	Likely direction of bias
I. Omitted factors		
Effects of lead on		
Growth	Not monetized	Underestimate
Hearing	Not monetized	Underestimate
Cancer	Not monetized	Underestimate
Metabolic disturbances	Not monetized	Underestimate
II. Medical-care costs (children)		
<25 μg/dl	No cost	Underestimate (See Rosen bone lead data)
≥25 μg/dl	CDC-recommended protocols	Midlevel estimates
III. Cognitive costs (children)		
Special education	20% of children ≥25 μg/dl 0% of children <25 μg/dl	Underestimate; some impact on children <25 μg/dl
Lost wages		
IQ effect on wage rate	0.5%/IQ point	Midlevel
Education effect on wage rate	6%/year	Midlevel
Education effect on participation	0.45%/graduating high school	Conservative
Net present value to age 6 of expected lifetime earnings	$222,000	Underestimate: low productivity growth, high discount rate, low female participation ensured
IQ pts/μg of lead per dl of blood	0.245	Midlevel based on metaanalysis
IV. Fetal effects		
Effects on gestational age	From Dietrich	Underestimate: includes only gestational-age effect, not independent birthweight effect Overestimate: not all studies have found effect
Values of statistical life	$3,000,000	Midlevel
V. Adult effects		
Medical expenses and lost wages		
Hypertension	$628/case-year	Underestimate: assumes 1980 fraction of hypertensives taking medicine
Heart attack	$44,000	Underestimate: does not include all expenses even in 1980 (viz Krupnick and Cropper) and excludes increased use of angioplastys and bypass operations; excludes activity limitation and pain and suffering
Strokes	$30,000	Underestimate: excludes activity limitation and pain and suffering
Blood level vs blood pressure	0.2 mm Hg/μg of lead per dl of blood	Midlevel
Mortality benefits		
Adults	$3,000,000/statistical life	Midlevel
Blood pressure–mortality relationship from Framingham	Logistic risk function	Midlevel

of benefits that have not been monetized, however, gives considerable confidence that the total benefits are in fact higher.

DEVELOPING NEEDS

The major needs for the better understanding and cost effective control of lead toxicity are for better understanding of the low-dose health effects, better measurement techniques (including statistical ones) for both research and screening,

better understanding of the molecular basis of lead toxicity, better techniques for treating lead intoxication (particularly at low doses), better techniques for abating lead, and better understanding of the impacts of the various techniques.

More is known about the health effects of lead than about those of any other environmental pollutant, but the continuum of effects detected and the changing magnitude of concern leave much to be determined. It is unlikely that the effects of low-dose lead have been fully characterized, and more work is needed both to expand the range of effects studied (for example, note the recent attention to growth, as well as cognitive effects) and to find more sensitive indicators of particular effects (for example, by the use of reaction-time tests and perturbations of sense of balance, rather than full-scale IQ tests, to measure CNS impacts).

Finally, but not least, better and less expensive techniques need to be developed for abating lead-paint exposure. Lead-paint abatement has been common for decades in cities with screening programs, and knowledge of the problem of contamination due to poor cleanup and technique is widespread, but basic scientific studies estimating the effects of different approaches on blood lead concentrations, controlling for appropriate covariates, have yet to be done. Reliable estimates of even the mean change in blood lead produced by standard techniques, as a function of housing and other characteristics, are unavailable. Under those circumstances, it is hardly surprising that innovative technologies have yet to be developed. Given the high cost of current techniques and the lack of funds to implement them, studies such as those just mentioned are critical to reducing the lead-paint hazard.

REFERENCES

Annest, J. L., Pirkle, J. L., Makuc, D., Neese, J. W., Bayse, D. D., and Kovar, M. G. (1983). Chronological trend in blood lead levels between 1976 and 1980. *N. Engl. J. Med.* **308,** 1373–1377.

Ashenfelter, O., and Ham, J. (1979). Education, unemployment, and earnings. *J. Polit. Econ.* **87,** S99–S116.

Barth, M. C., Janny, A. M., III, Arnold, F., and Sheiner, L. (1984). "A Survey of the Literature Regarding the Relationship between Measures of IQ and Income." EPA Contract 68-01-6614. ICF, Washington, DC.

Bellinger, D., Needleman, H. L., Bromfield, R., and Mintz, M. (1984). A followup study of the academic attainment and classroom behavior of children with elevated dentine lead levels. *Biol. Trace Elem. Res.* **6,** 207–223.

CDC (Centers for Disease Control) (1991). "Preventing Lead Poisoning in Young Children: A Statement by the Centers for Disease Control—October 1991." CDC, U.S. Department of Health and Human Services, Atlanta, GA.

CDC (Centers for Disease Control) (1985). "Preventing Lead Poisoning in Young Children: A Statement by the Centers for Disease Control, January 1985," DHHS Publ. No. 99-2230. CDC, U.S. Department of Health and Human Services, Atlanta, GA.

Chamberlain, G., and Griliches, Z. (1977). More on brothers. *In* "Kinometrics: Determinants of Socioeconomic Success Within and Between Families" (P. Taubman, Ed.), pp. 97–124. North-Holland, Amsterdam.

de la Burde, B., and Choate, M. S., Jr. (1975). Early asymptomatic lead exposure and development at school age. *J. Pediatr.* **87,** 638–642.

Dietrich, K. N., Krafft, K. M., Bier, M., Succop, P. A., Berger, O., and Bornschein, R. L. (1986). Early effects of fetal lead exposure: Neurobehavioral findings at 6 months. *Int. J. Biosoc. Res.* **8,** 151–168.

Dillingham, A. E. (1983). The influence of risk variable definition on value-of-life estimates. *Econ. Inquiry* **23**, 277–294.

EPA (U.S. Environmental Protection Agency) (1989). "Review of the National Ambient Air Quality Standards for Lead: Assessment of Scientific and Technical Information." EPA-450/02-89/022. Office of Air Quality Planning and Standards. Research Triangle Park, NC.

Fahim, M. S., Fahim, Z., and Hall, D. G. (1976). Effects of subtoxic lead levels on pregnant women in the state of Missouri. *Res. Commun. Chem. Pathol. Pharmacol.* **13**, 309–331.

Fisher, A., Chestnut, L. G., and Violette, D. M. (1989). The value of reducing risks of death: A note on new evidence. *J. Policy Anal. Manage.* **8**, 88–100.

Fulton, M., Raab, G., Thomson, G., Laxen, D., Hunter, R., and Hepburn, W. (1987). Influence of blood lead on the ability and attainment of children in Edinburgh. *Lancet* **1**, 1221–1226.

Gegax, D., Gerking, S., and Schulze, W. (1991). Perceived risk and the marginal value of safety. *Rev. Econ. Stat.* **73**, 589–596.

Griliches, Z. (1961). Hedonic price indexes for automobiles: An econometric analysis of quality change. *In* "The Price Statistics of the Federal Government, No. 73, General Series," pp. 173–196. National Bureau of Economic Research, New York.

Griliches, Z. (1977). Estimating the returns to schooling: Some econometric problems. *Econometrica* **45**, 1–22.

Hartunian, N. S., Smart, C. N., and Thompson, M. S. (1981). "The Incidence and Economic Costs of Major Health Impairments: A Comparative Analysis of Cancer, Motor Vehicle Injuries, Coronary Heart Disease and Stroke." Heath, Lexington, MA.

Hatzakis, A., Kokkevi, A., Katsouyanni, K., Maravelias, K., Salaminlos, F., Kalandidi, A., Koutselinis, A., Stefanis, K., and Trichopoulos, D. (1987). Psychometric intelligence and attentional performance deficits in lead-exposed children. *In* "International Conference on Heavy Metals in the Environment" (S. E. Lindberg and T. C. Hutchinson, Eds.), Vol. 1, pp. 204–209. CEP Consultants, Edinburgh.

Hatzakis, A., Kokkevi, A., Maravelias, C., Katsouyanni, K., Salaminlos, F., Kalandidi, A., Koutselinis, A., Stefanis, K., and Trichopoulos, D. (1989). Psychometric intelligence deficits in lead-exposed children. *In* "Lead Exposure and Child Development: An International Assessment." (M. A. Smith, L. D. Grant, and A. I. Sors, Eds.), pp. 211–223. Kluwer Academic, Netherlands.

Hawk, B. A., Schroeder, S. R., Robinson, G., Otto, D., Mushak, P., Kleinbaum, D., and Dawson, G. (1986). Relation of lead and social factors to IQ of low SES children: A partial replication. *Am. J. Ment. Defic.* **91**, 178–183.

Jones-Lee, M. W., Hammerton, M., and Phillips, P. R. (1985). The value of safety: Results of a national sample survey. *Econ. J.* **95**(377), 49–72.

Kakalik, J. S. (1981). "The Cost of Special Education." Rand Note N-1792-ED for the U.S. Department of Education. Rand Corp, Santa Monica, CA.

Kolb, J. A., and Scheraga, J. D. (1989). Discounting the benefits and costs of environmental regulations. *J. Policy Anal. Manage.* **9**, 381–390.

Krupnick, A. J., and Cropper, M. (1989). "The Social Costs of Chronic Heart and Lung Disease." Paper QE 89-16. Resources for the Future, Washington, DC.

Lansdown, R., Yule, W., Urbanowicz, M.-A., and Hunter, J. (1986). The relationship between blood-lead concentrations, intelligence, attainment and behaviour in a school population: The second London study. *Int. Arch. Occup. Environ. Health* **57**, 225–235.

Levin, R. (1986). "Reducing Lead in Drinking Water: A Benefit Analysis." U.S.E.P.A., Washington, DC.

Lyngbye, T., Hansen, O., Trillingsgaard, A., Beese, J., and Grandjean, P. (1990). Learning disabilities in children: Significance of low-level lead-exposure and confounding factors. *Acta Paediatr. Scand.* **79**, 352–360.

Marin, A., and Psacharopoulos, G. (1982). The reward for risk in the labor market: Evidence from the United Kingdom and a reconciliation with other studies. *J. Polit. Econ.* **4**, 827–853.

Mushak, P., and Crocctti, A. F. (1990). Methods for reducing lead exposure in young children and other risk groups: An integrated summary of a report to the U.S. Congress on childhood lead poisoning. *Environ. Health Perspect.* **89**, 121–124.

National Research Council (NRC) (1993). "Measuring Lead Exposure in Infants, Children, and Other Sensitive Populations." National Academy Press, Washington, DC.

Needleman, H. I., Schell, A., Bellinger, D., Leviton, A., and Allred, E. N. (1990). The long-term effects of childhood exposure to low doses of lead: An 11-year follow-up report. *N. Engl. J. Med.* **322**, 83–88.

O'Neal, K., and McGartlund, A. (1989). "Monetized Benefits of Regulating Sewage Sludge Use and Disposal. Final Report for the U.S. Environmental Protection Agency." Abt Associates, Cambridge, MA.

Olneck, M. R. (1977). On the use of sibling data to estimate the effects of family background, cognitive skills, and schooling: Results from the Kalamazoo brothers study. *In* "Kinometrics: Determinants of Socioeconomic Success Within and Between Families." (P. Taubman, Ed), pp. 125–162. North-Holland, Amsterdam.

Piomelli, S., Rosen, J. F., Chisolm, J. J., and Graef, J. W. (1984). Management of childhood lead poisoning. *J. Pediatr.* **105**, 523–532.

Provenzano, G. (1980). The social costs of excessive lead exposure during childhood. *In* "Low Level Lead Exposure: The Clinical Implications of Current Research." (H. L. Needleman, Ed.), pp. 299–315. Raven, New York.

Revis, N. W., Zinsmeister, A. R., and Bull, R. (1981). Atherosclerosis and hypertension induction by lead and cadmium ions: An effect prevented by calcium ion. *Proc. Natl. Acad. Sci. USA* **78**, 6494–6498.

Schroeder, S. R., and Hawk, B. (1987). Psycho-social factors, lead exposure, and IQ. *In* "Toxic Substances and Mental Retardation: Neurobehavioral Toxicology and Teratology. Monographs of the American Association on Mental Deficiency, No. 8." (S. R. Schroeder, Ed.), pp. 97–137. American Association on Mental Deficiency, Washington, DC.

Schroeder, S. R., Hawk, B., Otto, D. A., Mushak, P., and Hicks, R. E. (1985). Separating the effects of lead and social factors on IQ. *Environ. Res.* **38**, 144–154.

Schwartz, J. (1993). Beyond LOELs, p values, and vote counting: Methods for looking at the shapes and strengths of associations. *Neurotoxicology* **14**, 237–246.

Schwartz, J. (1994). Low-level exposure and children's IQ: A meta-analysis and search for a threshold. *Environ. Res.* **65**, 42–55.

Schwartz, J. Lead, blood pressure, and cardiovascular disease in men. *Arch. Environ. Health*, in press.

Schwartz, J. (1982). "The Benefits of Reducing Gasoline Lead Exposure: Memo to Docket." U.S.E.P.A.

Schwartz, J. (1992a). Low level health effects of lead—growth development and neurological disturbances. *In* "Human Lead Exposure" (H. Needleman, Ed.) CRC Press, Boca Raton, FL.

Schwartz, J. (1992b). Lead, blood pressure, and cardiovascular disease. *In* "Human Lead Exposure." (H. Needleman, Ed.). CRC Press, Boca Raton, FL.

Schwartz, J., and Pitcher, H. (1989). The relationship between gasoline lead and blood lead in the United States. *J. Off. Stat.* **5**, 421–431.

Schwartz, J., Pitcher, H., Levin, R., Ostro, B., and Nichols, A. L. (1985). "Costs and Benefits of Reducing Lead in Gasoline: Final Regulatory Impact Analysis." EPA-230/05-85/006. U.S. E.P.A., Washington, DC.

Smith, R. S. (1976). "The Occupational Safety and Health Act: Its Goals and its Achievements." American Enterprise Institute for Public Policy Research, Washington, DC.

Thaler, R., and Rosen, S. (1976). The value of saving a life: Evidence from the labor market. *In* "Household Production and Consumption" (N. E. Taleckji, Ed.), pp. 265–298. Columbia Univ. Press, New York.

U.S. DHHS (1981). "The 1978 Social Security Survey of Disability and Work." Social Security Administration, Washington, DC.

Violette, D. M., and Chestnut, L. (1983). "Valuing Reductions in Risks: A Review of the Empirical Estimates. Environmental Benefits Analysis Series." EPA-230/05-83/003. U.S.E.P.A., Washington, DC.

Viscusi, W. K. (1978). Labor market valuations of life and limb: Empirical evidence and policy implications. *Public Policy* **26**(3), 359–386.

Viscusi, W. K., and O'Connor, C. J. (1984). Adaptive responses to chemical labeling: Are workers Bayesian decision makers. *Am. Econ. Rev.* **74**, 942–956.

Wibberley, D. G., Khera, A. K., Edwards, J. H., and Rushton, D. I. (1977). Lead levels in human placentae from normal and malformed births. *J. Med. Genet.* **14**, 339–345.

Yule, W., Lansdown, R., Millar, J. B., and Urbanowicz, M.-A. (1981). The relationship between blood lead concentrations, intelligence, and attainment in a school population: A pilot study. *Dev. Med. Child Neurol.* **23**, 567–576.

Yule, W., Urbanowicz, M.-A., Lansdown, R., and Millar, I. (1984). Teachers' ratings of children's behaviour in relation to blood lead levels. *Br. J. Dev. Psychol.* **2**, 295–305.