NBER WORKING PAPER SERIES

AS ASSESSMENT OF THE BENEFITS OF AIR POLLUTION CONTROL: THE CASE OF INFANT DEATH

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Working Paper No. 1928

NATIONAL BUREAU OF ECONOMIC RESEARCH
1050 Massachusetts Avenue
Cambridge, MA 02138
May 1986

Working Paper #1928
May 1986

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ABSTRACT

This paper contains estimates of the impacts of air pollutants on race-specific neonatal mortality rates based on data for heavily populated counties of the U.S. in 1977. Unlike previous research in this area, these estimates are obtained from a well specified behavioral model of the production of health, which is estimated with the appropriate simultaneous equations techniques. The results suggest that sulfur dioxide is the dominant air pollutant in newborn survival outcomes. There is also evidence that an increase in sulfur dioxide raises the neonatal mortality rate by raising the percentage of low-birth weight births. Based on marginal-willingness-to-pay computations, we estimate that the benefits of a 10 percent reduction in sulfur dioxide levels range between $54 million and $1.09 billion in 1977 dollars.

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AN ASSESSMENT OF THE BENEFITS OF AIR POLLUTION CONTROL: THE CASE OF INFANT HEALTH

Theodore J. Joyce, Michael Grossman, and Fred Goldman*

I. Introduction

The controversy surrounding the work of Lave and Seskin (1970, 1973, 1977) on the negative effect of air pollution on aggregate mortality has continued unabated for well over a decade. The debate has sharpened the focus of succeeding research by calling attention to such issues as simultaneity (Gerking and Schulze 1981; Crocker et al. 1979), confounding (Schwing and MacDonald 1976; Lave and Chappie 1982), functional form (Smith 1976), and the impact of priors on model specification (Atkinson, Crocker, and Murdock 1985). Moreover, efforts to place a monetary value on the benefits of reduced pollution have also evolved. Emphasis on lost productivity (Lave and Seskin 1977) has given way to the theoretically more appealing measure of willingness to pay (Rosen 1981; Gerking 1983). Cross-sectional mortality studies are not a substitute for micro-level data based on individually monitored exposure. Nevertheless, a well-structured ecological study can provide evidence of potentially causal relationships between pollution and mortality. In addition, aggregate studies remain a practical means of "observing large numbers of people and environments in less than 'real time.'" (Lave and Chappie 1982, p. 348).

This paper is an attempt to incorporate many of the insights from past studies of pollution and mortality in order to measure the impact of air pollution on race-specific neonatal mortality rates (deaths within the first 27 days of life per thousand live births) across heavily populated counties

in the U.S. in 1977. Our study differs from previous research in the pollution area in that it is the first one that simultaneously (1) adopts a behavioral model of the production and demand for health, (2) employs a health indicator (neonatal mortality) that has a well documented relation to a set of medical and nonmedical inputs,[1] (3) recognizes that the inputs are endogenous variables and therefore uses the proper econometric techniques to obtain the best available estimates from which willingness-to-pay figures can most justifiably be approximated, (4) uses more recent data than Lave and Seskin (1977) and Crocker et al. (1979), and (5) performs an ecological analysis on a larger sample than has previously been attempted. Since the data are more recent they are more relevant to present policy discussions. Moreover, the recent automation of the Environmental Protection Agency's Storage and Retrieval of Aerometric Data (SAROAD) system has improved the accuracy with which data on the pollutants are collected and measured.

The advantages of focusing on neonatal mortality in research on the health effects of air pollution are also worth stressing. The infant mortality rate (deaths of infants within the first 364 days of life per thousand live births) is approximately equal to the mortality rate of 55 to 64 year olds and much higher than age-specific death rates of persons between the ages of 1 and 54. The neonatal mortality rate is twice as large as the postneonatal mortality rate (deaths of infants between the ages of 28 and 364 days per thousand live births). Moreover, accidents, which clearly are not related to pollution, are an important cause of postneonatal deaths. Expectant mothers are unlikely to move during their

pregnancies. Thus, studies of the relationship between pollution and neonatal mortality are not subject to the criticism that adults who die in one location may have been exposed to pollutants at other locations during their lifetimes.

In spite of the differences between our research and prior work in this area, previous research provides a basis for applying our framework to study the impacts of air pollution on infant health. Lave and Seskin (1973) fit dose-response functions in which the neonatal mortality rate (not race-specific) for 117 Standard Metropolitan Statistical Areas (SMSAs) in 1960 or 1961 is the dependent variable. These rates are positively related to total suspended particulate and sulfate concentrations in their ordinary least squares multiple regressions in 1960, and they are positively related to particulate concentrations in 1961. The 1960 sulfate effect and the 1961 particulate effect are statistically significant. Lave and Seskin (1977) use the same data set to study the effects of air pollution on total infant mortality (deaths of infants within the first 364 days if life), race-adjusted infant mortality, and race-specific infant mortality. In general, the particulate regression coefficient is positive and statistically significant, while the sulfate coefficient is positive and insignificant. Futhermore, in a smaller sample of SMSAs, in which additional pollutants were also examined, sulfur dioxide evidences a statistically significant association with infant mortality. Crocker et al. (1979), in a sample of 60 U.S. cities in 1970, report a positive and significant effect of sulfur dioxide concentrations in the air on deaths from early infant diseases (not race-specific), most of which occur within the

first 27 days of life. The particulate coefficient in their regression is positive and not significant, and the nitrogen dioxide coefficient is negative and not significant. Mendelsohn and Orcutt (1978) find that nitrates and particulates have a statistically significant effect on white male infant mortality rates but no effect on female rates across 404 county groups in 1970. These results are not consistent with their primary finding that sulfates are positively related to mortality among adults and that the effect increases with age.

II. Analytical Framework

Following Grossman and Jacobowitz (1981), Rosenzweig and Schultz (1981, 1982, 1983a, 1983b), Corman and Grossman (1985), Corman, Joyce, and Grossman (1985), and Joyce (1985), we assume that the parents' utility function depends on their own consumption, the number of births, and the survival probability of each of their offspring (assumed to be the same for each infant in a given family). Both the number of births and the survival probability are endogenous variables. In particular, the survival probability production function depends upon such endogenous inputs as the quantity and quality of medical care, maternal cigarette smoking, the use of abortion services, and the use of family planning services.[2] In addition, the production function is affected by the reproductive efficiency of the mother, including the unobserved biologically endowed probability that her infant will survive the first month of life, and other aspects of her efficiency in household production. Finally, the production function depends on the quality of the environment as reflected by the levels of various pollutants.[3]

Maximization of the parents' utility function subject to production and resource constraints generates a demand function for survival in which the survival probability or its complement, the neonatal mortality rate, is related to input prices (whose direct and indirect cost components are negatively related to input availability), efficiency, income, environmental quality, and tastes. The interaction between the survival demand and production function determines demand functions for medical care and other endogenous inputs. These demand functions depend on the same set of variables as the demand function for survival. Environmental quality is a relevant argument in the input demand functions because high levels of pollution may, for example, induce individuals to obtain larger quantities of preventive or curative medical care or to smoke less.

The preceding ideas are formalized in a structural equations model that incorporates the relationship between neonatal mortality and its two most proximate determinants — low birth weight and prematurity. In particular, there is an overwhelming amount of evidence that low birth weight (less than or equal to 2,500 grams or 5.5 pounds) is the most important endogenous risk factor in neonatal survival outcomes (for example, Harris 1982; Lewit 1983; Institute of Medicine 1985). There also is a considerable amount of evidence that prematurity, reflected by gestational ages of 36 weeks or less, is the most important and most proximate endogenous risk factor in birth weight outcomes (for example, Taffel 1980; Rosenzweig and Schultz 1981, 1982, 1983b; Harris 1982; Lewit 1983). The system of equations is designed to obtain estimates of the direct and indirect (through low birth weight) effects of five basic health inputs and air

pollution on neonatal mortality. These inputs are prenatal medical care, perinatal and neonatal care, the use of abortion services, the use of contraceptive services, and maternal cigarette smoking. The equations in the model have meaningful interpretations both at the family level and at the county level. The latter is the unit to which the empirical analysis in this paper pertains.

The basic model consists of the following nine equations:

$$1 - \pi = f_1(n, m, a, c, q, b, e) \qquad (1)$$

$$b = f_2(m, a, c, q, s, g, e) \qquad (2)$$

$$g = f_3(m, a, c, q, r, e) \qquad (3)$$

$$r = f_4(a, c, x, e) \qquad (4)$$

$$n = f_5(p, y, q, x, e) \qquad (5)$$

$$m = f_6(p, y, q, x, e) \qquad (6)$$

$$a = f_7(p, y, q, x, e) \qquad (7)$$

$$c = f_8(p, y, q, x, e) \qquad (8)$$

$$s = f_9(p, y, q, x, e) \; . \qquad (9)$$

Equations (1), (2), (3), and (4) are production functions, while equations (5)-(9) are input demand functions. In equation (1) the probability that an infant dies within the first month of life or the neonatal mortality rate at the county level ($1-\pi$, where π is the survival probability) is shown as a vector of perinatal and neonatal care inputs (n), a vector of prenatal medical inputs (m), the use of abortion services (a), the use of contraceptive services (c), environmental quality (q, which rises as the level of air pollution falls), the probability that the infant is born light (b), and the infant's biological endowment (e).[4] In equation (2) the

probability of a light (2,500 grams or less) birth or the fraction of light births in a county is a function of the variables in equation (1) except neonatal care and also depends on maternal cigarette smoking (s) and the probability that the birth is premature (g, which represents the probability that gestational age is less than 37 weeks). In equation (3) the probability of a premature birth or the county-level fraction of such births is related to the variables in equation (2) except cigarette smoking and also an endogenous risk factor in prematurity such as mother's age at birth (r).[5] In equation (4) the endogenous risk factor is expressed as a function of its determinants, including an observed exogenous risk variable (x, a measure of which is specified in Section III). In equations (5)-(9) the inputs depend on a vector of price and availability measures (p), socioeconomic characteristics that reflect command over resources and tastes (y), the exogenous risk measure, environmental quality, and the biological endowment. Each of the nine equations contains an unspecified disturbance term (u_i, i=1,..., 9) that is uncorrelated with $u_j (j \neq i)$. In addition each u_i is uncorrelated with the set of right-hand side variables in the equation that contains it.

The four production functions are structural equations because they show relationships among endogenous variables. Substitution of the input demand functions and equations (3) and (4) into (1) and (2) yields demand functions for survival and birth weight:

$$1 - \pi = f_{10}(p, y, q, x, e) \qquad (10)$$

$$b = f_{11}(p, y, q, x, e) \; . \qquad (11)$$

These are reduced form equations because only exogenous variables appear on

their right-hand sides. Together with the input demand functions, they constitute the reduced form of the model.

Given our interest in obtaining marginal-willingness-to-pay measures (see below), we focus on the estimation of the structural neonatal mortality rate production function (1) and the quasi-structural mortality production function obtained by substituting equations (2), (3), ankd (4) into equation (1):

$$1 - \pi = f_{12}(n, m, a, c, q, s, x, e). \quad (12)$$

This procedure enables us to calculate the direct and indirect (through low birth weight) effects of the basic health inputs and environmental quality on neonatal mortality.

If the infant's biological endowment (e) were an observed variable, unbiased estimates of the production function could be obtained by ordinary least squares. Since this is not the case, the endowment must be treated as one component of the disturbance term in each equation. Hence, our model generates a recursive system of equations whose disturbance terms may be correlated. In particular, although the researcher has no information about the endowment, the mother and her physician have at least some information about it. This information is likely to lead mothers with poor endowed birth outcomes and their physicians to try to offset these unfavorable prospects by choosing a different mix of inputs than other mothers (Rosenzweig and Schultz 1981, 1982, 1983a, 1983b; Harris 1982).

To be specific, mothers with poor endowments have incentives to seek prenatal care earlier in their pregnancies than other women, and their physicians are likely to obtain larger amounts of neonatal care for them. In

addition, such women are more likely to smoke less, to abort their pregnancies, or to use contraceptive services. Under these circumstances, ordinary least squares estimates of the parameters of the production function are biased and inconsistent because the inputs are correlated with the disturbance term, which reflects in part the endowment. In particular, the effects of the inputs on favorable infant health outcomes are understated.[6]

To circumvent the above problem, production functions are obtained by two-stage least squares. In the first stage of this procedure, the input demand functions and the reduced form birth weight equation are fitted with explanatory variables that are uncorrelated with the endowment by assumption. In the second stage the predicted values of the inputs and low birth weight rather than the actual values are used as regressors.

It should be noted that the biases that arise when equation (12) is estimated by ordinary least squares are likely to be more severe than the biases that arise when equation (1) is estimated in a similar manner. This is because equation (1) includes birth weight, which may be a very useful proxy for the infant's endowed probability of survival. Put differently, it is possible that the endowment has no effect or a smaller effect on neonatal mortality with low birth weight held constant. We explore this proposition in empirical tests discussed in Sections III and IV.

Certain restrictions must be imposed to insure that each equation in the system satisfies rank and order conditions for identification. The most important restrictions are: (1) neonatal care has no impact on low birth weight; (2) cigarette smoking affects neonatal mortality only through its effect on low birth weight; (3) prematurity affects mortality only

through low birth weight; and (4) cigarette smoking has no effect on prematurity. With regard to the first restriction, decisions to use neonatal care services are made after birth, and low birth weight causes more use of these services.[7] For the other assumptions, there is considerable supporting evidence.[8]

To allocate scarce resources among competing goals, policy makers require information about the dollar values of the potential health benefits associated with improvements in environmental quality (reductions in pollution). The benefits of a small (incremental) reduction in pollution is given by marginal willingness to pay, defined as the amount of income that must be taken from an individual to leave him as well off as previously (to hold his level of utility constant) when the level of pollution declines. Marginal willingness to pay can be obtained directly from the health production function and is independent of the demand function (Freeman 1979; Rosen 1981; Gerking 1983; Harrington and Portney 1983). It is given by the marginal product of environmental quality in the production function multiplied by the ratio of the price of an endogenous health input such as prenatal medical care utilization to the marginal product of that input. The ratio just mentioned coincides with the marginal cost of producing health.

In the context of our model, the marginal willingness to pay (dy, defined to be positive) for an increase in environmental quality of dq is

$$dy = \pi_q(p/\pi_m)dq. \qquad (13)$$

Here a subscript denotes a partial derivative, π_q is the marginal product of environmental quality (the increase in the survival probability caused

by a small increase in environmental quality), and π_m is the marginal product of prenatal care. In equilibrium the ratio of the price of an input used solely to produce infant health to its marginal product is the same for all inputs. Therefore, if p' is the price of neonatal care and π_n is its marginal product, an alternative estimate of marginal willingness to pay is given by

$$dy = \pi_q(p'/\pi_n)dq. \qquad (14)$$

Marginal-willingness-to-pay measures based on equations (13) and (14) are presented in Section IV.[9] Particularly in the case of prenatal care, the quasi-structural production function is the most relevant one to use in the computations. This is because prenatal care has its main effect on neonatal mortality via low birth weight (for example, Corman, Joyce, and Grossman 1985).[10]

Note that in a somewhat more general model where other aspects of health in addition to the survival of neonates enters the utility function, the formula for marginal willingness to pay given by equation (13) or (14) would be the correct theoretical construct if and only if environmental quality, prenatal care, and neonatal care affected neonatal mortality outcomes alone. However, if environmental quality affects the health of children, adolescents, and adults and other aspects of their well being such as cognitive development, then we understate willingness to pay. Finally, if medical care also affects those outcomes, which is plausible since they are known to depend on birth weight and health in the first year of life (for example, Edwards and Grossman 1979; Shakotko, Edwards, and Grossman 1981), the direction of the bias is indeterminant. These caveats should be kept in mind when the estimates are presented.

III. Empirical Implementation

A. Measurement of Variables

The neonatal mortality rate production functions are estimated with a data set that pertains to the 677 most populated counties of the United States. These are counties with a population of at least 50,000 persons in 1970.[11] We have constructed this data base from a variety of sources and have described it in detail in Corman and Grossman (1985), Corman, Joyce, and Grossman (1985), and Joyce, Grossman, and Goldman (1986). Table 1 contains definitions, means, and standard deviations of the variables used in this study.

The mortality production functions focus on the neonatal mortality rate as opposed to the postneonatal mortality rate or the total infant death rate. This strategy is adopted because most neonatal deaths are caused by congenital anomalities, prematurity, and complications of delivery. These conditions are more sensitive to improved prenatal, perinatal, and neonatal care than are the infectious diseases and accidents that contribute to postneonatal mortality. Moreover, accidents clearly are not related to pollution, and expectant mothers are unlikely to move during their pregnancies.

Separate regressions are fitted for white and black outcomes. Black infant health levels are much lower than white levels. For example, in 1977 the black neonatal mortality rate was roughly twice as large as the white rate. In a non-race-specific regression, one would enter the percentage of black births to control for race differences. But this variable would be highly correlated with the use of prenatal care and other inputs.

Table 1

Definition, Means, and Standard Deviation of Variables[a]

Variable	Definitions
Neonatal mortality rate*	Three-year average neonatal mortality rate centered on 1977; deaths of infants less than 28 days old per 1,000 live births. (μ_w = 8.837, σ_w = 1.595, μ_b = 16.387; σ_b = 3.299)
Teenage family planning users*[b]	Percentage of women aged 15-19 who used organized family planning clinics in 1975 (μ_w = 9.067, σ_w = 6.290, μ_b = 24.176, σ_b = 9.656)
Abortion rate	Three-year average state-specific resident abortion rate centered on 1976; abortions performed on state residents per 1,000 women aged 15-44 (μ_w = 24.969, σ_w = 8.716, μ_b = 24.754, σ_b = 8.602)
Prenatal care*	Three-year average fraction of live births for which prenatal care began in the first trimester (first three months) of pregnancy centered on 1977 (μ_w = .781, σ_w = .083, μ_b = .594, σ_b = .102)
Neonatal intensive care*	Sum of state-specific hospital inpatient days in Level II, or Level III, or Levels II and III neonatal intensive care units in 1979 per state-specific three-year average number of births centered on 1977 (μ_w = .641, σ_w = .385, μ_b = 1.501, σ_b = 1.011)
Cigarettes	State-specific daily number of cigarettes smoked per adult 18 years and older in 1976 (μ_w = 7.416, σ_w = .511, μ_b = 7.486, σ_b = .350)
Low birth weight*	Three-year average percentage of low-birth weight (2,500 grams or less) live births centered on 1977 (μ_w = 5.992, σ_w = .741, μ_b = 13.016, σ_b = 1.228)
High risk women*[b]	Number of women 15-19 and 40-44 as a fraction of women 15-44 in 1975. (μ_w = .335, σ_w = .022, μ_b = .350, σ_b = .026)
Carbon monoxide	Four-year average carbon monoxide level for the period 1975-1978; milligrams per cubic meter (μ_w = 2.320, σ_w = 1.147, μ_b = 2.325, σ_b = 1.063)
Lead	Four-year average lead level for the period 1975-1978; micrograms per cubic meter (μ_w = .843, σ_w = .724, μ_b = .794, σ_b = .617)

Table 1 (continued)

Variables	Definition
Sulfur dioxide	Four-year average sulfur dioxide level for the period 1975-1978; micrograms per cubic meter (μ_w = 30.826, σ_w = 18.810, μ_b = 34.858, σ_b = 20.326)
Particulates	Four-year average total suspended particulates level for the period 1975-1978; micrograms per cubic meter (μ_w = 71.053, σ_w = 22.905, μ_b = 70.366, σ_b = 17.755)
Nitrogen dioxide	Four-year average nitrogen dioxide level for the period 1975-1978; micrograms per cubic meter (μ_w = 54.094, σ_w = 30.908, μ_b = 53.192, σ_b = 30.878)

[a]An asterisk (*) next to a variable means that it is race-specific. All variables are county-specific unless otherwise indicated. The symbols μ_w, σ_w, μ_b, and σ_b denote the white mean, the white standard deviation, the black mean, and the black standard deviation, respectively. Means and standard deviations are weighted by the race-specific total number of births in the period 1976-1978. With the exception of the pollutants, the white data pertain to 677 counties, and the black data pertain to 357 counties. The pollution measures pertain to the first five subsamples for each race defined in the text. The number of counties in each subsample is indicated in Tables 2, 3, and 4.

[b]Variable is available for whites and nonwhites as opposed to whites and blacks.

By fitting race-specific regressions, multicollinearity is reduced, and the coefficients of the inputs and the pollutants are allowed to vary between races. Moreover, in preliminary regressions we tested and rejected the hypothesis that slope coefficients but not intercepts are the same for whites and blacks.

Counties are our units of observation rather than Standard Metropolitan Statisitical Areas (SMSAs) or states because counties tend to be more homogeneous with respect to key variables such as income, schooling, medical resources, and pollution. Some counties are so sparsely populated, however, that people may receive medical care outside the county. Also, small counties, with few births, experience large fluctuations in birth rates simply due to random movements. The problems with county data are reduced by including only counties with a population of at least 50,000 persons in 1970. A county must also have at least 5,000 blacks for inclusion in the black data base. There are 677 counties in the white sample and 357 counties in the black sample. The counties in the white data set accounted for approximately 80 percent of the white population of the U.S. in 1970, and the counties in the black data set accounted for a similar percentage of the black population of the U.S. in that year. In addition to selecting large counties, we attenuate random elements by employing a three-year average of the race-specific neonatal mortality rate for the period 1976-1978 as the dependent variable.

The air pollution variables were taken from the Environmental Protection Agency's Storage and Retrieval of Aerometric Data (SAROAD). This is an automated data processing system used by EPA for the storage of

data concerning concentrations of the six criteria air pollutants in the atmosphere: carbon monoxide, lead, sulfur dioxide, total suspended particulates, nitrogen dioxide, and ozone. These are pollutants for which National Ambient Air Quality Standards (NAAQS) have been established as part of the Clean Air Act. All major cities and one-third of all counties have at least one monitoring station, although a number of stations are not operative, and many others do not monitor all the pollutants. In preliminary regressions (not shown in Section IV), ozone never had a positive and statistically significant coefficient. Therefore, it was discarded from the set of pollutants.

The raw data in SAROAD take the form of hourly concentrations of carbon monoxide, sulfur dioxide, nitrogen dioxide and weekly concentrations of lead and total suspended particulates.[12] To obtain county-specific estimates of pollution the following algorithm was used. Counties in the data base were divided into census tracts or minor civil divisions in counties with no census tracts. Exposure measures for these smaller units by pollutant and year were computed as weighted (by the number of readings) averages of the monitor averages. Then county averages were obtained as weighted averages of the tract or the division averages, where the weight was the population density (population per square mile in 1980) of each tract or division in a given county. Finally, the pollutant-specific four-year average for the county was calculated as a simple average of the four annual averages.[13] If data were missing for one or two years, a three- or two-year average was computed. If an annual average was available for a single year alone, that figure was used.[14]

Complete data on the five pollutants are available for 102 of the 677 counties in the white sample and for 86 of the 357 counties in the black sample. Instead of limiting our production functions to these two subsamples, we define six subsamples for each race. The first five include counties in which a given pollutant measure is available, regardless of whether data on the other four pollutants are present. Each of the five subsamples is used to estimate a production function in which the pollutant at issue alone and the health inputs are included as regressors. The sixth subsample includes counties with data on all pollutants and is used to fit a production function in which the five pollutants are entered as explanatory variables. It is worth noting that the means and standard deviations of the neonatal mortality rate and the health inputs are very similar in the seven samples for each race (the total sample and the six subsamples).

The five basic inputs used in the production of infant health are neonatal intensive use, prenatal care use, the abortion rate, the use of family planning clinics by teenagers, and adult per capita smoking. Except for smoking, all the inputs should be negatively related to the measures of infant health described above.[15] Low birth weight, the endogenous risk factor in the structural neonatal mortality rate production function [equation (1)] is given by a three-year average of the percentage of births of 2,500 grams or less centered on 1977. The final explanatory variable represents the exogenous risk factor (x) in the quasi-structural production function [equation (12)]. For this variable, we use the number of women who are either teens or in their forties as a fraction of all women of childbearing age in 1975. These are the age groups considered most at risk for negative birth outcomes.

B. Estimation and Functional Form

The neonatal mortality equations (1) and (12) are fitted using a two-stage least squares procedure for the reasons discussed in Section II. Specifically, the unobserved health endowment, which is captured in the error term of the production function, is believed to be correlated with the use of the health inputs. Rosenzweig and Schultz (1982) refer to this problem as population "heterogeneity." Such "heterogeneity" causes ordinary least squares (OLS) estimates to be biased and inconsistent. If income and input availability measures are uncorrelated with the health endowment, however, these variables can serve as instruments in a two-stage least squares (TSLS) estimation procedure.

We test for the significance of the correlation between the production function residuals and the health inputs, using Wu's T_2 statistic (Wu 1973) as described by Nakamura and Nakamura (1981). If the null hypothesis of zero correlation between the error term and the regressors is not rejected, then OLS is an appropriate technique. For this reason, we perform OLS as well as the two-stage least squares technique on equations (1) and (12). A comparison of Wu statistics for equations (1) and (12) allows us to examine whether birth weight is a reasonable proxy for the health endowment.

In the first stage of our two-stage estimation procedure, birth weight, prenatal care, neonatal intensive care, abortion, and organized family planning use are predicted on the basis of the pollutants, female schooling, female poverty levels, the fraction of high-risk women, neonatal intensive care availability, abortion availability, organized family planning availability, maternal and infant care project availability, com-

munity health center availability, and the Medicaid program. With the exception of the pollutants, these right-hand side variables are similar to those used in Corman and Grossman's (1985) reduced form estimates. Predicted values of the five endogenous variables are then entered into the neonatal mortality equations. We do not estimate values for the smoking variable in a first stage because the smoking variable was already estimated on the basis of income, price, education, age, sex and race, as described in Joyce, Grossman, and Goldman (1986). Our procedure is based on the reasonable assumption that prenatal and neonatal input availability measures have zero coefficients in the cigarette demand function.[16]

Since the neonatal survival probability (π) ranges between zero and one, we specify the production function as a logistic equation:

$$\pi = [1 + \exp(-\alpha-\beta_1 \ln q-\beta_2 \ln m-\beta_3 \ln n-\beta_4 \ln w)]^{-1}. \qquad (15)$$

Here q is environmental quality, m is prenatal medical care, n is neonatal intensive care, w is a vector of additional explanatory variables, ln stands for natural logarithm, and the disturbance term is suppressed. By solving for the logarithm of the odds of survival relative to death $[\pi/(1-\pi)]$, one transforms the logistic function into a linear equation:

$$\ln[\pi/(1-\pi)] = a + \beta_1 \ln q + \beta_2 \ln m + \beta_3 \ln n + \beta_4 \ln w, \qquad (16)$$

which is called the logit function. The logit coefficient β_1, for example, shows the percentage change in the odds of survival for a one percent change in the quality of the environment. Given that the production function is logistic, the marginal-willingness-to-pay measure [see equations (13) and (14)] becomes[17]

$$dy = p(\beta_1/\beta_2)(m/q)dq = p'(\beta_1/\beta_3)(n/q)dq, \qquad (17)$$

where p is the price of prenatal care and p' is the price of neonatal intensive care. According to equation (17), marginal willingness to pay falls as the quality of the environment rises or as the level of pollution falls. Moreover, willingness to pay rises as prenatal care or neonatal intensive care rises. These extremely appealing theoretical properties are the result of the assumption that the logit function is linear in the logarithms of m, q, and n, and justify this assumption.[18] In addition, if interactions among pollutants are ignored, it is logical to define the level of pollution (z) as

$$z = q^{-1}. \tag{18}$$

Since we take $\ln[(1-\pi)/\pi]$ as the dependent variable, the final form of the equation to be fitted is:

$$\ln[(1-\pi)/\pi] = -\alpha + \beta_1 \ln z - \beta_2 \ln m - \beta_3 \ln n - B_4 \ln w. \tag{19}$$

Note that the marginal willingness to pay for a percentage increase in environmental quality (dln q) is exactly the same as the marginal willingness to pay for a percentage reduction in pollution (dln z):

$$dy = p(\beta_1/\beta_2)m \, d\ln q = -p(\beta_1/\beta_2)m \, d\ln z = -p'(\beta_1/\beta_3)n \, d\ln z. \tag{20}$$

Equation (19) is estimated with all explanatory variables except the percentage of teenagers who use organized family planning services and the fraction of high-risk women in natural logarithms. The former variable is entered in arithmetic form because it equals zero in certain counties, while the latter is entered in arithmetic form because it is not an endogenous health input. The logit coefficients of prenatal care, abortion, family planning, and neonatal intensive care are expected to be negative, and the coefficients of the pollutants, cigarette smoking, low birth

weight, and high-risk women are expected to be positive. Maddala (1983) shows that a weighted least squares procedure should be employed to fit a logit model to grouped data. The weights are given by $[B_j\pi_j(1-\pi_j)]^{1/2}$, where B_j is the number of race-specific births in the j^{th} county. Results obtained with such alternative function forms as the linear and log-linear models do not differ in a qualitative sense from those presented in Section IV.[19]

IV. Results

Ordinary least squares (OLS) and two-stage least squares (TSLS) estimates of the white and black logit neonatal mortality rate production functions are contained in Tables 2 and 3. Panel A in each table pertains to the full white and black samples. No pollution measures are included in the four regressions in panel A. The first two regressions (2-1, 2-2, 3-1, and 3-2) exclude the endogenous risk factor of low birth weight while the ensuing two (2-3, 2-4, 3-3 and 3-4) include the percentage of low-birth weight births as an explanatory variable. Panel B of Tables 2 and 3 pertains to the white and black sulfur dioxide samples. We present detailed results for the sulfur dioxide sample because, unlike the other four pollutants, sulfur dioxide has a consistently negative and statistically significant impact on newborn survival. As shown below, this is true regardless of whether sulfur dioxide is the sole pollutant or one of five in the production function. As with Panel A, Panel B contains OLS and TSLS estimates of the production functions with and without the endogenous risk factor. Finally, Panel C of Tables 2 and 3 presents the production function estimates with all five pollutants. Panel C follows the same format as

TABLE 2

Ordinary Least Squares and Two-Stage Least Squares Estimates of Logit Neonatal Mortality Rate Production Functions
Whites[a]

Explanatory Variable	Panel A: Full Sample				Panel B: Sulfur Dioxide Sample				Panel C: Five Pollutant Sample			
	OLS (2-1)	TSLS (2-2)	OLS (2-3)	TSLS (2-4)	OLS (2-5)	TSLS (2-6)	OLS (2-7)	TSLS (2-8)	OLS (2-9)	TSLS (2-10)	OLS (2-11)	TSLS (2-12)
Teenage family planning users*[b]	-0.002	-0.007	-0.003	-0.009	-0.001	0.0004	-0.002	-0.003	-0.001	0.003	-0.004	-0.004
	(-1.54)	(-2.55)	(-3.13)	(-4.40)	(-0.94)	(0.12)	(-1.61)	(-1.52)	(-0.48)	(0.55)	(-1.71)	(-1.03)
Ln abortion rate[b]	-0.074	-0.088	-0.061	-0.077	-0.086	-0.121	-0.055	-0.071	-0.110	-0.141	-0.064	-0.104
	(-4.04)	(-3.43)	(-3.49)	(-3.11)	(-3.32)	(-3.30)	(-2.23)	(-2.01)	(-2.04)	(-1.84)	(-1.35)	(-1.43)
Ln prenatal care*[b]	-0.328	-0.491	-0.130	-0.171	-0.319	-0.516	-0.077	0.056	-0.294	-0.493	-0.160	0.288
	(-5.64)	(-5.97)	(-2.26)	(-1.41)	(-3.83)	(-4.31)	(-0.96)	(0.40)	(-1.36)	(-1.46)	(-0.88)	(0.83)
Ln neonatal intensive care*[b]	-0.047	-0.028	-0.052	-0.001	-0.068	-0.147	-0.063	-0.060	-0.064	-0.109	-0.042	-0.086
	(-2.12)	(-0.48)	(-2.47)	(-0.02)	(-2.23)	(-1.86)	(-2.17)	(-0.88)	(-1.18)	(-0.83)	(-0.90)	(-0.70)
High risk women*	0.742	0.445	--	--	0.233	0.817	--	--	-0.539	0.327	--	--
	(2.32)	(1.10)	--	--	(0.49)	(1.31)	--	--	(-0.61)	(0.29)	--	--
Ln cigarettes[b]	0.493	0.469	--	--	0.511	0.496	--	--	0.431	0.417	--	--
	(5.63)	(5.20)	--	--	(4.64)	(4.38)	--	--	(2.11)	(2.12)	--	--
Ln low birth weight*[b]	--	--	0.571	0.615	--	--	0.611	0.820	--	--	0.864	1.387
	--	--	(10.20)	(3.36)	--	--	(7.54)	(4.89)	--	--	(5.05)	(3.66)
Ln sulfur dioxide	--	--	--	--	0.035	0.038	0.030	0.029	0.073	0.077	0.047	0.018
	--	--	--	--	(3.68)	(3.79)	(3.33)	(2.97)	(3.31)	(3.31)	(2.33)	(0.62)
Ln particulates	--	--	--	--	--	--	--	--	0.060	0.044	0.015	0.014
	--	--	--	--	--	--	--	--	(0.86)	(0.55)	(0.23)	(0.18)
Ln nitrogen dioxide	--	--	--	--	--	--	--	--	-0.022	-0.024	-0.003	0.023
	--	--	--	--	--	--	--	--	(-1.23)	(-1.29)	(-0.20)	(1.02)
Ln carbon monoxide	--	--	--	--	--	--	--	--	-0.016	-0.024	-0.036	-0.049
	--	--	--	--	--	--	--	--	(-0.38)	(-0.54)	(-0.99)	(-1.16)
Ln lead	--	--	--	--	--	--	--	--	-0.050	-0.036	0.012	0.093
	--	--	--	--	--	--	--	--	(-1.07)	(-0.67)	(0.28)	(1.42)
Constant	-4.296	-3.334	-4.967	-4.742	-4.247	-3.512	-5.374	-6.265	-4.139	-3.426	-5.494	-8.263
	(-12.90)	(-7.52)	(-15.76)	(-5.74)	(-9.70)	(-6.22)	(-11.71)	(-7.13)	(-3.53)	(-2.00)	(-5.18)	(-3.78)
R^2	0.119		0.195		0.166		0.249		0.246		0.381	
F	15.14	15.35	32.53	16.00	8.20	8.09	16.02	10.54	2.67	2.60	5.59	4.01
Sample size	677	677	677	677	297	297	297	297	102	102	102	102
Wu test F	3.43		2.79		2.08		1.07		.87		1.17	

Notes to Table 2

[a]Ln stands for natural logarithm. Asymptotic t-ratios in parentheses. The critical asymptotic t-ratios at the 5 percent level are 1.64 for a one-tailed test and 1.96 for a two-tailed test. In this table and the others that contain regression results, the F-ratio associated with each regression is significant at the 1 percent level unless otherwise indicated. An asterisk (*) next to a variable means it is race specific. Each pollution measure is the natural logarithm of the county-specific geometric mean.

[b]Endogenous in TSLS equations.

TABLE 3

Ordinary Least Squares and Two-Stage Least Squares Estimates of Logit Neonatal Mortality Rate Production Functions
Blacks[a]

Explanatory Variable	Panel A: Full Sample				Panel B: Sulfur Dioxide Sample				Panel C: Five Pollutant Sample			
	OLS (3-1)	TSLS (3-2)	OLS (3-3)	TSLS (3-4)	OLS (3-5)	TSLS (3-6)	OLS (3-7)	TSLS (3-8)	OLS (3-9)	TSLS (3-10)	OLS (3-11)	TSLS (3-12)
Teenage family planning users*[b]	-0.002	-0.007	-0.002	-0.006	-0.003	-0.007	-0.003	-0.008	-0.005	-0.006	-0.004	-0.004
	(-1.37)	(-2.38)	(-2.11)	(-2.48)	(-1.76)	(-2.47)	(-2.20)	(-2.65)	(-2.04)	(-1.66)	(-1.79)	(-1.21)
Ln abortion rate[b]	-0.034	-0.098	-0.042	-0.076	-0.079	-0.211	-0.050	-0.177	-0.238	-0.337	-0.099	-0.235
	(-0.91)	(-1.61)	(-1.51)	(-1.74)	(-1.41)	(-2.40)	(-1.24)	(-2.66)	(-2.52)	(-2.52)	(-1.45)	(-1.72)
Ln prenatal care*[b]	-0.039	-0.249	0.004	-0.054	0.012	-0.162	0.062	-0.095	-0.048	0.028	0.189	0.298
	(-0.66)	(-2.06)	(0.07)	(-0.39)	(0.16)	(-1.09)	(0.90)	(-0.59)	(-0.35)	(0.09)	(1.50)	(0.76)
Ln neonatal intensive care*[b]	-0.069	-0.064	-0.079	-0.082	-0.103	-0.093	-0.099	-0.097	-0.090	-0.095	-0.112	-0.115
	(-2.43)	(1.19)	(-3.01)	(-1.69)	(-2.87)	(-1.49)	(-3.04)	(-1.64)	(-1.79)	(-1.11)	(-2.65)	(-1.46)
High risk women*	-0.028	-0.278	--	--	0.252	-0.260	--	--	1.636	1.169	--	--
	(-0.06)	(-0.48)	--	--	(0.38)	(-0.33)	--	--	(1.38)	(0.91)	--	--
Ln cigarettes[b]	0.254	0.353	--	--	-0.006	0.002	--	--	-1.061	-0.784	--	--
	(0.99)	(1.36)	--	--	(-0.02)	(0.01)	--	--	(-1.63)	(-1.41)	--	--
Ln low birth weight*[b]	--	--	0.838	0.668	--	--	0.857	0.286	--	--	1.082	0.701
	--	--	(8.05)	(7.84)	--	--	(6.37)	(0.84)	--	--	(5.29)	(1.34)
Ln sulfur dioxide	--	--	--	--	0.04	0.049	0.030	0.045	0.068	0.077	0.029	0.053
	--	--	--	--	(2.73)	(2.59)	(2.05)	(2.50)	(2.25)	(2.41)	(1.14)	(1.62)
Ln particulates	--	--	--	--	--	--	--	--	-0.052	-0.096	-0.009	-0.070
	--	--	--	--	--	--	--	--	(-0.50)	(0.83)	(-0.09)	(-0.65)
Ln nitrogen dioxide	--	--	--	--	--	--	--	--	0.006	0.010	-0.009	0.002
	--	--	--	--	--	--	--	--	(0.29)	(0.46)	(-0.56)	(0.10)
Ln carbon monoxide	--	--	--	--	--	--	--	--	-0.029	-0.028	0.008	-0.009
	--	--	--	--	--	--	--	--	(-0.47)	(-0.45)	(0.16)	(-0.16)
Ln lead	--	--	--	--	--	--	--	--	-0.014	-0.005	-0.001	0.002
	--	--	--	--	--	--	--	--	(-0.25)	(-0.08)	(-0.03)	(0.03)
Constant	-4.249	-3.187	-6.029	-5.155	-3.967	-2.579	-6.350	-3.767	-1.372	-1.585	-7.219	-6.065
	(-6.26)	(-3.66)	(-15.48)	(-3.62)	(-3.90)	(-2.15)	(-12.25)	(-2.65)	(-0.79)	(-0.84)	(-7.21)	(-1.93)
R^2	0.033		0.182		0.076		0.239		0.219		0.394	
F	2.04[c]	2.70	15.63	4.11	2.21[c]	2.06[c]	9.87	2.80	1.88[c]	1.58[d]	4.88	1.95[c]
Sample Size	357	357	357	357	196	196	196	196	86	86	86	86
Wu test F	2.24		.72		1.75		2.38		.76		1.34	

[a]See note a to Table 2.

[b]Endogenous in TSLS equations.

[c]Significant at 5 percent level.

[d]Not significant at 5 percent level.

Panels A and B.

An overview of the results in Panel A of Tables 2 and 3 indicates that all the health inputs have the anticipated signs. In particular, in the specifications that omit the percentage of low-birth weight births, the logit coefficients of teenage family planning use, abortion, prenatal care, and neonatal intensive care are negative, while the logit coefficient of cigarette smoking is positive. In the case of whites, 14 out of the 18 coefficients are significant at the 5 percent level. With respect to the blacks, 13 of 18 coefficients have t-ratios greater than one. In most cases the magnitudes and significance levels of the input coefficients do not vary dramatically among the three samples (Panels A, B, and C). A Chow test was used to determine whether the neonatal mortality regressions in the samples that contain pollutants differed from the regressions in the non-pollution samples. The null hypothesis of no difference was never rejected when comparing the four sulfur dioxide regressions with the regressions from counties that have no sulfur dioxide monitors. When the same test was performed with the five pollutant sample, the null hypothesis of no difference was rejected in two out of the four cases at the 5 percent level.[20]

A comparison of the TSLS and OLS estimates reveals that the TSLS coefficients of prenatal care, abortion, and family planning are substantially larger than the corresponding OLS coefficients. These findings reflect decisions by women with unobserved poor endowed birth outcomes to initiate care earlier in their pregnancies than other women. In addition, such women are more likely to abort their pregnancies and use organized family

planning services. The existence of these relationships biases OLS estimates of the production function parameters of prenatal care, abortion, and family planning toward zero. Furthermore, the result that this difference is greater for blacks underscores the argument by Rosenzweig and Schultz (1983a) that OLS and other direct correlational estimates of prenatal care's effect on early infant deaths may be seriously underestimating its true impact on infant health.

Based on the Wu test, the null hypothesis of zero correlation between the health inputs and the disturbance term is rejected at the 5 percent level in the full-sample, white regressions (2-1, 2-3). In the case of blacks, the null hypothesis is rejected at the 10 percent level in regression (3-1). In the smaller samples which contain data on pollutants, the null hypothesis can only be rejected once (regression 3-7). It is noteworthy that in the full sample the Wu statistic falls when birth weight is held constant. Similar results were obtained by Rosenzweig and Schultz (1982, 1983b), Corman, Joyce, and Grossman (1985) and Joyce (1985). This suggests that in the estimation of infant health production functions the relevant intermediate birth outcome may be an effective proxy for endowed health. Based on this result and the previous work just cited, we emphasize the TSLS estimates in specifications that exclude the percentage of low-birth weight births and the OLS estimates in the equations that include this risk factor.

The results from including sulfur dioxide as an exogenous determinant of neonatal mortality are presented in Panel B of Tables 2 and 3. In all eight specifications the coefficient of sulfur dioxide is significant at

the 5 percent level or better. Each of the other four pollutants was also treated as an exogenous determinant of neonatal mortality. Table 4 presents the coefficients and t-ratios of each pollutant estimated by OLS as well as TSLS for both the white and black samples. These results reveal that carbon monoxide and total suspended particulates have the most consistently negative impact on early infant survival. However, when all five pollutants are employed as regressors, sulfur dioxide is the only significant predictor of neonatal mortality (Panel C, Tables 2 and 3).

The environmental quality effects in Panel C of Tables 2 and 3 are our most refined estimates because they include the full set of pollutants. The conclusion that sulfur dioxide is the dominant pollutant should be interpreted with some caution due to the intercorrelation among the five measures. Nevertheless, this result is consistent with the regressions that consider each pollutant one at a time, for the logit coefficients of sulfur dioxide are the only ones that are always significant at conventional levels. Note that the positive and significant sulfur dioxide coefficients in regressions (2-10), (2-11), and (3-10) are not artifacts of the negative coefficients of some of the other pollutants. In specifications not shown, we deleted the pollutants with negative coefficients in Panel C. The magnitude and significance of sulfur dioxide was unaltered.

There is also evidence that sulfur dioxide impacts on early infant deaths by raising the percentage of low-birth weight births. Comparing regressions 2-6 with 2-7 and 3-6 with 3-7, one sees that the inclusion of low birth weight lowers the white sulfur dioxide coefficient by 21 percent and lowers the black sulfur dioxide coefficient by 39 percent. The same

Table 4

Pollution Coefficients from Ordinary Least Squares Estimates and Two Stage Least Squares Estimates of Logit Neonatal Mortality Rate Production Functions, Each Pollutant Entered Individually[a]

Explanatory Variable	Panel A: Whites					Panel B: Blacks				
	Birth Weight Excluded		Birth Weight Included		Sample Size	Birth Weight Excluded		Birth Weight Included		Sample Size
	OLS (4-1)	TSLS (4-2)	OLS (4-3)	TSLS (4-4)		OLS (4-5)	TSLS (4-6	OLS (4-7)	TSLS (4-8)	
Ln carbon monoxide	0.023	0.031	0.020	0.029	246	0.036	0.034	0.031	0.041	171
	(1.25)	(1.54)	(1.19)	(1.56)		(1.22)	(1.02)	(1.45)	(1.44)	
Ln particulates	0.023	0.017	0.019	0.019	604	0.103	0.103	0.067	0.057	328
	(1.42)	(1.01)	(1.28)	(1.18)		(2.87)	(2.70)	(2.01)	(1.51)	
Ln nitrogen dioxide	-0.006	-0.007	0.013	0.022	172	0.009	0.022	-0.001	0.0004	130
	(-0.51)	(-0.62)	(1.22)	(1.85)		(0.66)	(1.34)	(-0.96)	(0.002)	
Ln lead	0.001	0.027	0.007	0.022	260	-0.003	0.002	0.002	0.006	179
	(0.05)	(1.23)	(0.40)	(1.10)		(-0.08)	(0.35)	(0.09)	(0.20)	

[a]t-ratios in parentheses.

comparison in the five pollutant sample reveals an even greater decline (regressions 2-10 with 2-11 and 3-10 with 3-11). Nevertheless, since the decrease in the magnitude of the sulfur dioxide coefficient is less than fifty percent, the direct effect of this pollutant on neonatal mortality is greater than its indirect effect through low birth weight in every case but one.

In sum, the significance of our results is underscored by model which generated them. By including measures of prenatal care and neonatal care as well as abortion, we have controlled for three health inputs that have a well-documented relationship to neonatal mortality. Moreover, these same inputs have experienced dramatic increases in use during the 1970's, a period during which the decline in neonatal mortality accelerated substantially. Stated in Bayesian terms, we posses strong priors for the inclusion of these inputs (Atkinson, Crocker, and Murdock 1985). Furthermore, by examining the impact of pollution on mortality holding the percentage of low-birth weight births constant, we have effectively controlled for the major socioeconomic risk factors (such as births to teenagers and unmarried mothers) that operate through birth weight. In other words, the possibility that the association between sulfur dioxide and early infant deaths represents a spurious relationship due to the omission of other health inputs or risk factors is unlikely.

We conclude this paper by calculating the marginal willingness to pay for declines in sulfur dioxide levels that result in increases in neonatal survival rates. The computations are based on equation (20). Two alternative estimates are presented. In the first the marginal cost of

improving infant health outcomes is given by the ratio of the price of prenatal care to its marginal product. In the second marginal cost is given by the ratio of the price of neonatal intensive care to its marginal product. For reasons spelled out in Section II and the preceding portion of this section, both computations are based on the TSLS estimates of the quasi-structural production function. The logit coefficients are taken from the sulfur dioxide sample rather than from the five pollutant sample because the former is larger than the latter and because its input coefficients do not differ significantly from those of the full sample.

When prenatal care is used to measure the marginal cost of improving the probability of infant survival, five pieces of information are required to obtain marginal willingness to pay. These are the logit coefficient of environmental quality (β_1), the logit coefficient of prenatal care (β_2), the specified level of prenatal care (m), the percentage increase in environmental quality divided by 100 (dln q) or the percentage decline in pollution (-dln z) divided by 100, and the price of prenatal care (p). For whites, β_1 equals .038, and β_2 equals .516. For blacks, β_1 equals .049, and β_2 equals .162. The specified value of prenatal care is the mean in the sulfur dioxide sample: .784 in the case of whites and .599 in the case of blacks. Since the marginal-willingness-to-pay formula pertains to small changes, a 10 percent reduction in sulfur dioxide concentrations for each race is considered (dln q = .1).

If all expectant mothers began prenatal care in the first or second trimester of pregnancy, the price or cost of starting care in the first trimester approximately equals the total cost of three physician visits.

In turn total cost is given by the direct (money) cost of the visits (the payment to the physician) and the indirect cost (transportation cost plus the sum of travel and waiting time multiplied by the opportunity cost of time). Most physicians charge more for an initial visit than for a follow-up visit. Fee information provided by the American Medical Association (1984) reveals that obstetricians/gynecologists, who are the primary suppliers of prenatal care, follow this pricing policy. Since a woman who begins care in the second trimester must have an initial visit, we multiply the price of a follow-up office visit to an obstetrician/gynecologist -- $17 in 1977 -- by three visits to obtain a direct cost component of $51.[21]

Colle and Grossman (1978) estimate the indirect cost of a pediatric care visit at $3 in 1970. This is the sum of round-trip travel time and waiting time multiplied by the hourly wage rate of a mother who works or the potential hourly wage of a mother who does not work.[22] Based on trends in the hourly wage rate between 1970 and 1977, the comparable figure for the latter year amounts to $5 per visit. Since Colle and Grossman ignore transportation cost, we expand the indirect cost per visit to $7 based on data given by Goldman and Grossman (1978). When multiplied by three visits, this gives an indirect cost of $21. Consequently, the cost of beginning prenatal care in the first trimester rather than in the second trimester is $72 in 1977 dollars.

Along the same lines, the cost of beginning care in the first trimester rather than the third trimester amounts to the total cost of six follow-up visits: $144 in 1977 dollars. The cost of beginning care in the first trimester rather than obtaining no care at all involves the total cost of an

initial visit and eleven follow-up visits. The AMA data indicate that the direct cost of an initial visit was $29 in 1977. Therefore, cost of this option comes to $276.

For each race, the total cost of beginning care in the first trimester is defined as a weighted average of the cost of each of the three options. The weight applied to, for example, the first option is the race-specific number of births in which prenatal care began in the second trimester divided by the race-specific number of births with no prenatal care in the first trimester. This gives a total cost of $94 for whites and $100 for blacks.

In the model outlined in Section II, parents in a given family allocate the same quantity of resources to each birth. Therefore, the figures just given are multiplied by the race-specific total fertility rates in 1977 -- 1.7 children for white women and 2.3 children for black women.[23] This yields a price of prenatal care equal to $160 for whites and $230 for blacks. To the extent that prenatal care is financed by private health insurance and Medicaid, the private price or marginal cost is overestimated, although the social marginal cost is not. Under reasonable conditions, this implies that social marginal willingness on pay exceeds private marginal willingness to pay.[24] We focus on the former measure.

Based on the above data, the social marginal willingness to pay of a typical white woman between the ages of 15 and 44 or her husband comes to $1 and the corresponding figure for a typical black woman between the ages of 15 and 44 comes to $4. In 1977 there were 34 million white woman in childbearing ages in the 677 countries in our data base and 5 million black

women. Collectively, these women or their families would have been willing to pay $54 million (social marginal willingness to pay) in 1977 dollars for the improved neonatal survival prospects associated with a 10 percent reduction in sulfur dioxide concentrations.

When neonatal intensive care is used to measure the marginal cost of raising the probability of infant survival, the logit coefficients of neonatal intensive care (.147 for whites and .093 for blacks) replace those of prenatal care in the computation of willingness to pay. The specified levels of neonatal intensive care are .646 white patient days per birth and 1.591 black patient days per birth. Budetti et al. (1981) report that the cost per case in a Level II or III neonatal intensive care unit was $8,000 in 1978 and that the average length of stay in such a unit was 13 days. Based on trends in the medical care component of the Consumer Price Index between 1977 and 1978, the cost per patient day in a neonatal intensive care unit was $568 in 1977. Multiplying by the race-specific fertility rates, one obtains a price of neonatal intensive care of $966 for whites and $1,306 for blacks.[25]

The above data generate much larger values of social marginal willingness to pay than those based on prenatal care. To be specific, the social marginal willingness to pay of a typical white woman comes to $16 and that of a typical black woman comes to $110. The collective marginal-willingness-to-pay-figure of $1.09 billion is approximately 20 times larger than the $54 million sum associated with prenatal care.

The divergence between the two estimates of marginal willingness to pay suggests that the same infant survival probability could be produced at

lower cost by spending less on neonatal intensive care and more on prenatal care. This does not necessarily mean that pregnant women or their physicians are selecting a non-optimal input mix. Given that neonatal intensive care is financed to a very large extent by private health insurers, the Federal government, and other third parties, the present input mix may well be optimal (cost minimizing) from a private point of view. Of course, it may also be the case that the differential impacts of neonatal care and prenatal care on health outcomes beyond the first month of life justify the current allocation of resources.

We want to emphasize that the main contribution of this paper is not that we have obtained an upper-bound estimate of the benefits of a 10 percent reduction in sulfur dioxide levels of $1.09 billion in 1977 dollars and a lower-bound estimate of $54 million. Instead, the main contribution is that these estimates are obtained from a well specified behavioral model of the production of health, which has been estimated with the appropriate simultaneous equations techniques. They stand in sharp contrast to existing figures that apply extraneous estimates of the value of life or the earnings foregone by premature death to dose-response functions obtained by ordinary least squares.

FOOTNOTES

*Research for this paper was supported Cooperative Agreement CR 811041 with the U.S. Environmental Protection Agency. We are indebted to Linda Edwards, Bart Ostro, Paul Portney, Paul Taubman, and especially George Provenzano for helpful comments and suggestions. We are also indebted to James Capel, Theodore Johnson, and Roy Paul of PEI Associates, Inc. for providing us with air pollution data from SAROAD. We wish to thank Frank Chaloupka and Naci Mocan for research assistance. This paper has not undergone the review accorded official NBER publications; in particular, it has not been submitted for approval by the Board of Directors. Any options expressed are those of the authors and not those of EPA or NBER.

[1]For a summary of the literature and the most recent findings, see the set of articles in the Section I of Bracken (1984).

[2]The amount of time worked by the mother during pregnancy and her use of alcohol are excluded from the theoretical model because of lack of data. Rosenzweig and Schultz (1981, 1982, 1983b) exclude the number of months the mother worked while pregnant from their final estimates of infant health production functions because its coefficient was insignificant in preliminary regressions. In a recent report on low birth weight, the Institute of Medicine (1985) concludes that the association between maternal alcohol use and unfavorable birth outcomes is much less uniform than that between maternal cigarette smoking and these outcomes. Heavy alcohol consumption during pregnancy raises the risk of delivering a baby with fetal alcohol syndrome. The empirical evidence is less clear, however, with respect to the effects of moderate or light alcohol use.

[3]Following the literature on the estimation of dose-response functions by economists and epidemiologists, we treat pollution as exogenous. In a more complete model this variable would have an endogenous component because the amount of exposure to multimedia pollutants and the ingestion of these pollutants depend on decisions with regard to location, diet, and occupation. In addition, at a more aggregate level, state and local governments in areas with high initial mortality and morbidity rates and high pollution levels may allocate resources to reducing these levels. Note that there exist theoretical and empirical micro-epidemiological studies in which pollution is viewed as endogenous (for example, Spengler et al. 1981; Duan 1982), but these studies do not contain estimates of dose-response functions.

[4]Note that an increase in e is associated with a more favorable endowment.

[5]Other endogenous risk factors in the prematurity production function include parity and legitimacy status of the birth. These factors are not incorporated into the model because we do not estimate the prematurity production function, as explained below. Clearly, we do not ignore the risk indicators just cited because they are caused to a large extent by abortion and contraceptive services at the county level.

[6]In the case of cigarette smoking, the detrimental impact of this input is understated in absolute value.

[7]Given the advanced state of perinatal science, infants requiring neonatal intensive care services may be identified prior to birth. Even in this situation low birth weight still causes neonatal intensive care, and

causality from use to birth weight can be ruled out.

[8]The U.S. Department of Health and Human Services (1980) summarizes numerous studies supporting restriction (2). Restriction (3) is consistent with research of the Institute of Medicine (1985) and Harris (1982). Restriction (4) is supported by Rosenzweig and Schultz (1982, 1983b). In part prematurity is excluded from equation (1) because gestational age is difficult to measure and was not reported on the birth certificates of a number of states during the period of our empirical analysis. If equation (1) is viewed as the one that is obtained by replacing g and r by their determinants, then cigarette smoking must be excluded from the prematurity equation. This is necessary for the quasi-structural mortality and birth weight production functions to satisfy the rank condition. These equations are

$$1 - \pi = d(n, m, a, c, q, b, x, e)$$

$$b = b(m, a, c, q, s, x, e)$$

If s enters the equation for $1 - \pi$, the rank condition is satisfied for the birth weight production function but is violated for the mortality production function. Empirical estimates of the above mortality function (not shown) do not differ from the ones presented in Section IV, which exclude x.

[9]Inputs that affect arguments in the utility function besides infant survival cannot be used to compute willingness to pay (Rosen 1981). Therefore, abortion use, family planning use, and maternal cigarette smoking are not employed.

[10]Conceptually, neonatal care has no causal impact on birth weight.

Therefore, if birth weight is an ideal measure of the endowment, there are two equivalent estimates of the neonatal care coefficient. One is obtained from the structural production function fitted by ordinary least squares. The second is obtained from the quasi-structural production function fitted by two-stage least squares. In the latter case the use of instrumental variables corrects for the correlation between neonatal care use and the disturbance term, while in the former case the inclusion of low birth weight accomplishes the same goal.

[11]One county with a population of at least 50,000 persons in 1970 was eliminated from the sample because it was the only such county characterized as an isolated rural county with no incorporated place with a population of at least 2,500 persons in 1970. In addition, the District of Columbia was excluded because of difficulty in defining its relevant market area. In particular, many nonresidents use its sophisticated neonatal intensive care hospitals, and these facilities are not likely to be widely available to its relatively large black population. A second reason for excluding the District of Columbia is that Stanley K. Henshaw, who estimates resident abortion rates for the Alan Guttmacher Institute, informed us that figures for the District of Columbia are very unreliable.

[12]Certain monitors in SAROAD, called source monitors, are located very near to factories and other large sources of industrial pollution. Exposure readings from these monitors are misleading indicators of the environmental quality of a typical resident of the county in question. Consequently, source monitors were not used in the development of pollution measures at the county level. There are no zeros in the SAROAD data.

Instead, each pollutant has a minimum detectible level. If readings fall below this point, then the observation is given the value of its particular minimum detectible level.

[13]We experimented with a second algorithm that did not divide counties into census tracts or minor civil divisions and did not employ population density as a weighting factor. In this algorithm county averages were obtained directly as weighted (by the number of readings) averages of the monitor averages. Our results were unaltered. For a more detailed discussion, see Joyce, Grossman, and Goldman (1986).

[14]Two types of averages resulted from the algorithm: an arithmetic average and a logarithmic average (the antilogarithm of which gives the geometric mean). In addition the logarithmic standard deviation was obtained. Since pollution measures typically have a log-normal distribution (for example, Tanner, D'Ottavia, and Gorber 1978), the logarithmic mean plus two standard deviations was used to test and reject the hypothesis that the maximum pollution level is a more important determinant of neonatal mortality than the mean level. The logarithmic means are employed in the production functions employed in Section IV, but the estimated effects are similar to those obtained with the logarithms of the arithmetic means. The arithmetic means of the pollutants are presented in Table 1 to underscore the close correspondence between the estimated pollution levels in our data base and published figures for the U.S. as a whole during the period from 1975 through 1978 (for example, Bureau of the Census 1983, Table 355).

[15]We focus on teenage family planning use because neonatal death rates

associated with births to teenage mothers are substantially higher than births to women beyond the age of 20 (for example, Joyce 1985). Moreover, Forrest (1980) finds that the use of organized family planning services by teenagers has a sizable negative effect on teenage birth rates. It follows that the use of family planning services by teenagers may have a larger impact on neonatal mortality than the use of these services by older women.

[16]Although cigarette consumption is labeled as an endogenous variable in Section IV, it should be noted that the same variable is used in OLS and two-stage estimation procedures.

[17]The marginal products of environmental quality and medical care are

$$\pi_q = \beta_1 \pi(1-\pi) q^{-1}$$

$$\pi_m = \beta_2 \pi(1-\pi) m^{-1} .$$

Hence,

$$(\pi_q/\pi_m) = (\beta_1/\beta_2)(m/q) .$$

[18]Put differently, the assumption guarantees that an isoquant between m and q or between n and q us convex to the origin.

[19]In preliminary regressions we experimented with measures pertaining to population density, climate, and the percentage of employed persons who work in manufacturing industries. The inclusion of these regressors had very minor impacts on the coefficients of the pollutants and the health inputs. Therefore, they are excluded from the regressions in Section IV.

[20]The two cases in which the null hypothesis was rejected were the white specification including low birth weight and the black specification excluding low birth weight. The F-statistics were 2.16 (6,665 df) and 2.79 (7,343 df), respectively.

[21]The reader is cautioned that obstetricians typically charge pregnant women a flat fee for prenatal visits and the delivery of the child, rather than a fee for each prenatal visit. This suggests that the AMA fee data pertain to visits to obstetricians/gynecologists for services other than prenatal care. Nevertheless, it is reasonable to assume that the real resource cost of a prenatal care visit is similar to the cost of a gynecological visit. It also is reasonable to assume that an obstetrician will charge a lower flat fee to a woman who begins prenatal care after the first trimester.

[22]The potential wage is estimated from a race-specific regression of the natural logarithm of the hourly wage rate of working mothers on mother's years of formal schooling, mother's years of experience in the labor market, years of experience squared, and the number of children in the family. Colle and Grossman present separate indirect cost estimates for whites and blacks, but the two figures are very similar.

[23]The total fertility rate is the number of children a woman would have in her lifetime if, at each year of age, she experienced the birth rate occurring in the specified year. The total fertility rate of blacks indicated in the text actually pertains to nonwhites.

[24]Price is multiplied by the quantity of prenatal care in the marginal-willingness-to-pay formula (20). Therefore, social marginal willingness to pay exceeds private marginal willingness to pay if the price elasticity of demand for prenatal care is less than one in absolute value. While there are no estimates of this parameter, Ghez and Grossman (1980) summarize a number of studies in which the price elasticity of demand for

physician visits by adults or children is less than one. Note that social marginal willingness to pay is not necessarily overstated if social marginal cost is multiplied by the observed quantity of prenatal care in equation (20). This depends on whether the observed quantity exceeds, equals, or falls short of the socially optimal quantity. For a detailed discussion of the latter concept, see Ghez and Grossman (1980).

[25]The indirect cost component of neonatal intensive care is ignored. To be sure, a mother with an infant in a neonatal intensive care unit will spend much more time in the hospital than otherwise. But she probably would not have returned to her usual activities even if her infant had not been hospitalized. It should be noted, however, that the psychic cost of the hospitalization clearly is substantial.

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