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An Assessment of the Benefits of Air Pollution Control: The Case of Infant Health*

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I. INTRODUCTION

The controversy surrounding the work of Lave and Seskin [2, 3, 4] 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 [5]; Crocker *et al.* [6]), confounding (Schwing and MacDonald [7]; Chappie and Lave [8]), functional form (Smith [9]), and the impact of priors on model specification (Atkinson *et al.* [10]). Moreover, efforts to place a monetary value on the benefits of reduced pollution have also evolved. Emphasis on lost productivity (Lave and Seskin [2]) has given way to the theoretically more appealing measure of willingness to pay

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(Rosen [11]; Gerking and Stanley [12]). Cross-sectional mortality studies are not a substitute for microlevel 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'" (Chappie and Lave [8, p. 348]).

This paper incorporates many 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 United States in 1977. Our study differs from previous research in the pollution area in that it is the first 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, (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 [2] and Crocker *et al.* [6], 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 may be more hesitant to move so as not to disrupt important relationships with physicians, relatives, and friends. Thus, studies of the relationship between pollution and neonatal mortality should be less subject to the criticism that adults who die in one location may have been exposed to pollutants at other locations during their lifetimes.

II. ANALYTICAL FRAMEWORK

Following Grossman and Jacobowitz [13], Rosenzweig and Schultz [14–17], Corman and Grossman [18], Corman *et al.* [19], and Joyce [20], we

assume that parents' utility depends on their 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. 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.

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 overwhelming evidence that low birth weight (less than or equal to 2500 g or 5.5 lb) is the most important endogenous risk factor in neonatal survival outcomes. There also is considerable 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. The system of equations, which is described in detail in Joyce *et al.* [1], 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 structural neonatal mortality production function in the model is

$$1 - \pi = f_1(n, m, a, c, q, b, e). \tag{1}$$

In this equation 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), which rises as the endowment rises. The model also contains structural production functions for the probability of a light (2500 g or less) birth or the fraction of light births in a county and for the probability of a premature birth.

Substitution of these equations into (1) yields a quasi-structural neonatal mortality production function:

$$1 - \pi = f_2(n, m, a, c, q, s, x, e). \tag{2}$$

Here s is maternal cigarette smoking and x is an exogenous risk variable in birth outcomes which is measured at the county level by the number of women who are either teens or in their forties as a fraction of all women of childbearing age.

Given our interest in obtaining marginal willingness-to-pay measures (see below), we focus on the estimation of (1) and (2). 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.

In addition to the production functions, the model generates demand functions for birth weight and the five health inputs. In each of these six equations, the dependent variable is related to a vector of price and availability measures, socioeconomic characteristics that reflect command over resources and tastes, the exogenous risk measure, environmental quality, and the biological endowment. They are reduced-form equations because only exogenous variables appear on their right-hand sides.

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 poorly endowed birth outcomes and their physicians to try to offset these unfavorable prospects by choosing a different mix of inputs than other mothers (Rozenzweig and Schultz [14–17]; Harris [21]).[1] Under these circumstances, ordinary least-squares estimates of the parameters of the production function are biased and inconsistent because the inputs are

[1]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 prenatal and neonatal care for them. In addition, such women are more likely to smoke less, to abort their pregnancies, or to use contraceptive services. These incentives are present because a reduction in the endowment is equivalent to a reduction in real income. Provided all commodities in the utility function are superior, their optimal values fall. This is possible only if resources are reallocated in favor of infant health inputs. These real income-related reallocation effects are reinforced if a reduction in the endowment raises the marginal products of the infant health inputs. It follows that the effects of the inputs on infant health outcomes are understated in absolute value if the production function is estimated by ordinary least squares.

correlated with the disturbance term, which reflects in part the endowment. To circumvent this problem, production functions are obtained by two-stage least squares.

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 reduction in pollution are 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 utility level 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 [22]; Rosen [11]; Gerking and Stanley [12]). 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

$$dy = \pi_q(p/\pi_m)\,dq. \tag{3}$$

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

$$dy = \pi_q(p'/\pi_n)\,dq. \tag{4}$$

Marginal willingness-to-pay measures based on (3) and (4) are presented in section IV.[2] Particularly in the case of prenatal care, the quasi-structural production function is the most relevant one to use in the computations.

[2] The use of (3) or (4) to compute marginal willingness to pay is based on the assumption that parents equate the marginal cost of an increase in infant survival to the monetary value of its marginal utility. To the extent that some parents are willing to pay a great deal for a healthy child, marginal cost exceeds marginal benefits for them. This caveat should be kept in mind when the estimates are presented in Section IV. Note that inputs that affect arguments in the utility function in addition to infant survival cannot be used to compute willingness to pay (Rosen [11]). Therefore, abortion use, family planning use, and maternal cigarette smoking are not employed.

This is because prenatal care has its main effect on neonatal mortality via low birth weight (for example, Corman *et al.* [19]).[3]

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. The nonpollution variables are from a variety of sources and are described in detail in Corman and Grossman [18], and Corman *et al.* [19]. Table 1 contains definitions, means, and standard deviations of the variables used in this study.

The air pollution variables were taken from the EPA's SAROAD. This is an automated data processing system used by the 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, and nitrogen dioxide and weekly concentrations of lead and total suspended particulates. 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

[3]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.

TABLE 1

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

Variable	Definition
Neonatal mortality rate*	Three-year average neonatal mortality rate centered on 1977; deaths of infants less than 28 days old per 1000 live births. ($\mu_w = 8.837$, $\sigma_w = 1.595$, $\mu_b = 16.387$; $\sigma_b = 3.299$)
Teenage family planning users*[b]	Percentage of women aged 15–19 who use organized family planning clinics in 1975 ($\mu_w = 9.067$, $\sigma_w = 6.290$, $\mu_b = 24.176$, $\sigma_b = 9.656$)
Abortion rate	Three-year average state-specific resident abortion rate centered on 1976; abortions performed on state residents per 1000 women aged 15–44 ($\mu_w = 24.969$, $\sigma_w = 8.716$, $\mu_b = 24.754$, $\sigma_b = 8.602$)
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 ($\mu_w = 0.781$, $\sigma_w = 0.083$, $\mu_b = 0.594$, $\sigma_b = 0.102$)
Neonatal intensive care*	Sum of state-specific hospital inpatient days in Level II, Level III, or Levels II and III neonatal intensive care units in 1979 per state-specific 3-year average number of births centered on 1977 ($\mu_w = 0.641$, $\sigma_w = 0.385$, $\mu_b = 1.501$, $\sigma_b = 1.011$)
Cigarettes	State-specific daily number of cigarettes smoked per adult 18 years and older in 1976 ($\mu_w = 7.416$, $\sigma_w = 0.511$, $\mu_b = 7.486$, $\sigma_b = 0.350$)
Low birth weight*	Three-year average percentage of low-birth weight (2500 g or less) live births centered on 1977 ($\mu_w = 5.992$, $\sigma_z = 0.741$, $\mu_b = 13.016$, $\sigma_b = 1.228$)
High-risk women*[b]	Number of women 15–19 and 40–44 as a fraction of women 15–44 in 1975 ($\mu_w = 0.335$, $\sigma_w = 0.022$, $\mu_b = 0.350$, $\sigma_b = 0.026$)
Carbon monoxide	Four-year average carbon monoxide level for the period 1975–1978; milligrams per cubic meter ($\mu_w = 2.320$, $\sigma_w = 1.147$, $\mu_b = 2.325$, $\sigma_b = 1.063$)
Lead	Four-year average lead level for the period 1975–1978; micrograms per cubic meter ($\mu_w = 0.843$, $\sigma_w = 0.724$, $\mu_b = 0.794$, $\sigma_b = 0.617$)
Sulfur dioxide	Four-year average sulfur dioxide level for the period 1975–1978; micrograms per cubic meter ($\mu_w = 30.826$, $\sigma_w = 18.810$, $\mu_b = 34.858$, $\sigma_b = 20.326$)
Particulates	Four-year average total suspended particulates level for the period 1975–1978; micrograms per cubic meter ($\mu_w = 71.053$, $\sigma_w = 22.905$, $\mu_b = 70.366$, $\sigma_b = 17.755$)
Nitrogen dioxide	Four-year average nitrogen dioxide level for the period 1975–1978; micrograms per cubic meter ($\mu_w = 54.094$, $\sigma_w = 30.908$, $\mu_b = 53.192$, $\sigma_b = 30.878$)

[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.

was the population density (population per square mile in 1980) of each tract or division in a given county. Finally, the pollutant-specific 4-year average for the county was calculated as a simple average of the four annual averages. If data were missing for 1 or 2 years, a 3- or 2-year average was computed. If an annual average was available for a single year alone, that figure was used.[4]

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 care 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. Low birth weight, the endogenous risk factor in the structural neonatal mortality rate production function, (1), is given by a 3-year average of the percentage of births of 2500 g or less centered on 1977. The final explanatory variable represents the exogenous risk factor (x) in the quasi-structural production function, (2). 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.

[4]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, the logarithmic mean plus two standard deviations were 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 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 presented in Table 1 underscore the close correspondence between the estimated pollution levels in our data base and published figures for the United States as a whole during the period from 1975 through 1978.

B. Estimation and Functional Form

The neonatal mortality equations (1) and (2) are fitted using a two-stage least-squares procedure for the reasons discussed in Section II. In the first stage, 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, fraction of high-risk women, neonatal intensive care availability, abortion availability, organized family planning availability, maternal and infant care project availability, community health center availability, and the Medicaid program.[5] Since the neonatal survival probability (π) ranges between zero and one, we specify the production function as a logistic equation:

$$\pi = [1 + \exp(-\alpha - \beta_1 \ln q - \beta_2 \ln m - \beta_3 \ln n - \beta_4 \ln w)]^{-1}. \quad (5)$$

Here q is environmental quality, m is prenatal 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,

$$\ln[\pi/(1 - \pi)] = a + \beta_1 \ln q + \beta_2 \ln m + \beta_3 \ln n + \beta_4 \ln w, \quad (6)$$

which is called the logit function.

Given that the production function is logistic, the marginal willingness-to-pay measure [see (3) and (4)] becomes[6]

$$dy = p(\beta_1/\beta_2)(m/q)\, dq = p'(\beta_1/\beta_3)(n/q)\, dq, \quad (7)$$

where p is the price of prenatal care and p' is the price of neonatal intensive care. According to (7), marginal willingness to pay falls as the quality of the environment rises or as the pollution level 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.[7] In addition, if interactions among pollu-

[5] 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 the more detailed version of this paper.

[6] 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, $(\pi_q/\pi_m) = (\beta_1/\beta_2)(m/q)$.

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

tants are ignored, it is logical to define the level of pollution (z) as

$$z = q^{-1}. \tag{8}$$

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

$$\ln[(1 - \pi)/\pi] = -\alpha + \beta_1 \ln z - \beta_2 \ln m - \beta_3 \ln n - \beta_4 \ln w. \tag{9}$$

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

$$dy = p(\beta_1/\beta_2)m\, d \ln q = -p(\beta_1/\beta_2)m\, d \ln z = -p'(\beta_1/\beta_3)n\, d \ln z. \tag{10}$$

Equation (9) 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 [23] 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 jth 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.

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

TABLE 2

Ordinary Least-Squares and Two-Stage Least-Squares Estimates of Logit Neonatal Mortality Rate Production Functions for 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		0.87		1.17	

[a] Ln stands for natural logarithm. Asymptotic t-ratios in parentheses. The critical asymptotic t-ratios at the 5% 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% 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 for 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		0.72		1.75		2.38		0.76		1.34	

[a] See Note *a* to Table 2.
[b] Endogenous in TSLS equations.
[c] Significant at 5% level.
[d] Not significant at 5% level.

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]

	Panel A: Whites					Panel B: Blacks				
	Birth weight excluded		Birth weight included			Birth weight excluded		Birth weight included		
Explanatory variable	OLS (4-1)	TSLS (4-2)	OLS (4-3)	TSLS (4-4)	Sample size	OLS (4-5)	TSLS (4-6)	OLS (4-7)	TSLS (4-8)	Sample size
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.

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 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 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 differ from the regressions in the nonpollution 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% level.[8] Finally, the TSLS coefficients of prenatal care, abortion, and family planning are substantially larger than their OLS counterparts. This supports the argument of Rosenzweig and Schultz [14, 15] that direct correlational estimates of infant health productions functions tend to underestimate their true impact.

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% 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.

[8]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, 664 df) and 2.79 (7, 343 df), respectively.

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.

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 (10). Two 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. Both computations are based on the TSLS estimates of the quasi-structural production function.[9] 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.

The cost of prenatal care is computed as the incremental cost of obtaining care in the first trimester. 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. Since some women began care in the third trimester and some obtain no care at all, 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.[10]

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. 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

[9]As discussed in the longer version of this paper, the Wu test (Wu [24]) determines whether there is a statistically significant correlation between the right-hand-side regressors and the error term. As reported in Tables 2 and 3 of this paper, the null hypothesis of zero correlation between the health inputs and the disturbance term is rejected at the 5% level in the full-sample white regression (2-1) and at the 10% level in the black regression (3-1). Based on these results and the previous work cited, we emphasize the TSLS estimates in specifications that exclude low birth weight.

[10]This total cost includes an indirect component which incorporates the cost due to travel and waiting time.

private price or marginal cost is overestimated, although the social marginal cost is not. Under reasonable conditions, this implies that social marginal willingness to pay exceeds private marginal willingness to pay. 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 women 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% 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 (0.147 for whites and 0.093 for blacks) replace those of prenatal care in the computation of willingness to pay. The specified levels of neonatal intensive care are 0.646 white patient days per birth and 1.591 black patient days per birth. The cost per patient day in a neonatal intensive care unit is estimated at $568 in 1977. Multiplying by the race-specific fertility rates, one obtains a price of neonatal intensive care of $966 for whites and $1306 for blacks.[11]

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 women 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 nonoptimal 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.

[11] The indirect cost component of neonatal intensive care is ignored because of the ambiguity as to what they would entail.

We emphasize that the main contribution of this paper is not that we have obtained an upper-bound estimate of the benefits of a 10% 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.

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