Birth Outcome Production Function in the United States

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ABSTRACT

This paper contains the first infant health production functions that simultaneously consider the effects of a variety of inputs on race-specific neonatal mortality rates. These inputs include the use of prenatal care, neonatal intensive care, abortion, federally subsidized organized family planning clinics, maternal and infant care projects, community health centers, and the WIC program. We place major emphasis on two-stage least squares estimation. Our results underscore the qualitative and quantitative importance of abortion, prenatal care, neonatal intensive care, and the WIC program in black and white birth outcomes.

I. Introduction

Neonatal mortality rates declined sharply in the U.S. between 1964 and 1982—from 17.9 deaths of infants less than one month old per thousand live births to 7.7 deaths per thousand live births (National Center for Health Statistics 1985). For whites, the mortality rate fell by 4.9 percent

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Research for this paper was supported by Grant Number 5 RO1 HD16316 from the National Institute of Child Health and Human Development to the NBER. The authors thank the following for providing data: Kathleen Bajo of Ross laboratories; Richard Bohrer, Edward Duffy, Joann Gephart, and Robert Nelson of the Bureau of Health Care Delivery and Assistance, DHHS; Stephen M. Davidson of the Northwestern University Program in Hospital and Health Services Administration; Gary Davis of the American Hospital Asso-
[Submitted October 1985; accepted September 1986]

per year (annually compounded), and for blacks, the rate of decline was 4.2 percent per year. During this period there were significant changes in a number of the important health inputs related to neonatal mortality. Federally subsidized programs such as family planning clinics, community health centers (CHCs), maternal and infant care (M and I) projects, the Special Supplemental Food Program for Women, Infants and Children (WIC), and Medicaid were either expanded or implemented for the first time. In addition, the use of prenatal care grew, and there were numerous medical advances resulting in high technology neonatal intensive care units. Finally, during this period, abortion was legalized.

Since the beginning of 1981, however, budget cutbacks by the Reagan Administration have curtailed the rates of growth of such poverty-related programs as WIC, M and I projects, community health centers, subsidized family planning clinics, and Medicaid. When inflation is taken into account, the absolute sizes of some of these programs declined in real terms. The cutbacks coincide with a slowing in the decline of mortality rates, especially for blacks. For instance, from 1981 to 1982, the black neonatal mortality rate fell by 2.2 percent, and the white rate fell by 4.2 percent. These developments have caused some persons to attribute the deceleration in the rate of decline in neonatal mortality to the Reagan Administration's policies (for example, Miller 1985).

The purpose of this study is to examine the relationship between the utilization of the health inputs mentioned above and race-specific neonatal mortality in a production function context. Although a number of studies have treated subsets of these inputs,[1] none have entered them together in a multivariate birth outcome equation estimated with data covering a large percentage of all births in the U.S. We use the estimates to gain insights into the causes of the rapid reduction in neonatal mortality since 1964 and the deceleration in the rate of decline in the early 1980s. Our empirical analysis

ciation; Jacqueline D. Forrest and Stanley K. Henshaw of the Alan Guttmacher Institute; and Letty Wunglueck of the Health Care Financing Administration. They also thank Peter Budetti, Karen Davis, Jacqueline D. Forrest, Stanley K. Henshaw, Salih Neftci, Chris Robinson, David Salkever, T. Paul Schultz, John Strauss, and an anonymous referee for helpful comments and suggestions and Emil Berendt for research assistance. A longer version of this paper with more detailed descriptions of the data and the analytical model is available from the authors. 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 opinions expressed are those of the authors and not those of NICHD or NBER.

1. For example, see Williams 1974, 1979; Grossman and Jacobowitz 1981; Goldman and Grossman 1982; Hadley 1982; Harris 1982; Paneth et al. 1982; Rosenzweig and Schultz 1981, 1982, 1983a, 1983b; Lewit 1983, Kotelchuck et al. 1984. No study entered all variables together in a multivariate birth outcome equation estimated with data covering a large percentage of all births in the U.S.

is based on a cross-section of U.S. counties in 1977, with the neonatal mortality rate (deaths of infants within the first 27 days of life per thousand live births) as the principal birth outcome, and with the incidence of low birth weight (2,500 grams or less) as an intermediate outcome. This allows us to examine the extent to which prenatal inputs operate directly on mortality and also allows us to examine their indirect effects on mortality rates through low birth weight.

In models developed by Harris (1982) and Rosenzweig and Schultz (1982, 1983a, 1983b), mothers with poor endowed birth outcomes will attempt to offset these unfavorable prospects by utilizing more health inputs. Thus, the decision to use the health inputs may not only affect the outcome, but the potential outcome may also affect utilization. We adopt two strategies to account for this potential reverse causality. First, major emphasis is placed on two-stage least squares estimates of the production function with prenatal care, abortion, and neonatal intensive care as endogenous (choice) variables. Second, we control for the health endowment directly, using the incidence of low birth weight as a proxy for the endowment. If birth weight is, in fact, an appropriate endowment measure, the production functions can be estimated using ordinary least squares. We then test for the appropriateness of this proxy variable approach.

II. Analytical Framework

Following Corman and Grossman (1985) and the theoretical literature that they cite, we assume that the parents' utility function depends on their consumption, the number of births, and the survival probability of each birth (which does not vary among births 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, nutrition, and the own time of the mother. 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.

The preceding ideas are formalized in a structural equations model that incorporates the relationship between neonatal mortality and its most proximate determinant—low birth weight. 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. The system of equations, which is described in detail in Corman, Joyce, and Grossman (1985), is designed to obtain esti-

mates of the direct and indirect (through low birth weight) effects of five basic health inputs. 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

$$(1) \quad \pi = f_1(n, m, a, c, b, e).$$

In this equation the probability that an infant dies within the first month of life or the neonatal mortality rate at the county level (π) 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), the probability that the infant is born light (b), and the infant's biological endowment (e, which rises as the endowment rises). The full model also contains structural production functions for the probability of a light (2,500 grams 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 Equation (1) yields a quasi-structural neonatal mortality production function:

$$(2) \quad \pi = f_2(n, m, a, c, s, x, e).$$

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. By estimating both Equations (1) and (2), we are able to calculate the direct and indirect (through low birth weight) effects of the basic health inputs.

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, 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 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). 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. To circumvent this problem, production functions are estimated by two-stage least squares.

It should be noted that the biases that arise when Equation (2) 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 on neonatal mortality with low birth weight held constant. A test of this proposition is discussed in the next section.

The model underscores the need to treat the abortion rate as an endogenous variable. This is both because abortion availability and the underlying frequency distribution of health endowments (the distribution that would be observed in the absence of abortion) vary among counties. Suppose that availability varies but the underlying frequency distribution does not. Since less healthy fetuses are more likely to be aborted, the actual health endowment of infants born in counties with high abortion rates will exceed the health endowment of infants born in counties with low abortion rates. Consequently, an expansion in the abortion rate will lower the fraction of light births. If the underlying frequency distribution of endowments varies among counties, abortion rates will be higher in counties with low underlying endowments, all other things the same, and the correlation between the abortion rate and the actual endowment is reduced and may even be negative.

Based on evidence to date, it is anticipated that abortion, contraceptive services, and prenatal care services will have their primary effect on neonatal mortality through low birth weight (for example, Institute of Medicine 1985). These inputs, however, may have sizable direct effects on newborn survival if other relevant inputs are lacking. For instance, we have no measures of the quality of prenatal and perinatal care. If these variables are positively correlated with abortion, contraception, and prenatal care, the latter inputs can have negative impacts on birth weight-specific neonatal mortality. This should be kept in mind when interpreting the results.[2]

2. We use a two-stage least squares procedure to purge the observed health inputs of their correlation with the endowments. This is based on the assumption that the reduced form determinants of input utilization are not correlated with the endowment. If the reduced form regressors are correlated with the unmeasured inputs, the use of two-stage least squares does not eliminate relationships between measured and unmeasured inputs.

III. Empirical Implementation

A. Data and Measurement of Variables

We have constructed the data base from a variety of sources which are described in detail in Corman and Grossman (1985) and Corman, Joyce, and Grossman (1985). Counties are the units of observation because they are the smallest units for which aggregate data are available. Some counties are so small, however, that people may receive medical care outside the county. Also, small counties experience large fluctuations in birth rates simply due to random movements. These problems are reduced by including only large counties. Our sample includes counties with at least 50,000 persons in 1970, and for black regressions, at least 5,000 blacks. The 677 counties in the white regressions and 357 counties in the black regressions account for about 80 percent of the white and black populations in the U.S. in 1970.[3] In addition to selecting large counties, we attenuate random elements by employing three-year averages of the race-specific neonatal mortality rate and the percentage of low-birth weight births, weighting regressions by the square root of the race-specific total number of births.

Separate regressions are fitted for white and black birth outcomes, rather than a nonrace-specific equation which enters the percentage of black births. By fitting race-specific equations, we circumvent a possible source of multicollinearity, if race and input usage are correlated. Moreover, in preliminary regressions we tested and rejected the hypothesis that slope coefficients but not intercepts are the same for whites and blacks.

Table 1 contains definitions, means, and standard deviations of the variables used in this study. The key inputs at issue in this paper are prenatal care, abortion, organized family planning clinic services, maternal and infant care (M and I) projects, community health centers (CHCs), WIC, and neonatal intensive care. All of these measures are expected to have negative regression coefficients in the neonatal mortality rate production function. Additional risk factors such as smoking and women in high-risk age groups are expected to have positive coefficients. Note that the use of M and I

3. 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, Washington, D.C. was excluded because of difficulty of defining its relevant market area for neonatal intensive care. For all other counties, we defined the state as the relevant neonatal intensive care area. Since this was impossible for D.C., we eliminated the observation. A second reason for excluding it is that Stanley K. Henshaw, who estimates resident abortion rates by states for the Alan Guttmacher Institute, informed us that figures for the states are very reliable but those for D.C. are very unreliable. Note that we use state-specific resident abortion rates in our analysis because county-specific resident abortion rates are not available. Note also that, although 84 percent of all births in Washington, D.C. were black, D.C. black births accounted for less than 2 percent of all black births in the sample of 358 black counties.

projects and CHCs is combined in one measure—Bureau of Community Health Services (BCHS) project use—because the BCHS (renamed the Bureau of Health Care Delivery and Assistance in 1982) is the agency within the U.S. Department of Health and Human Services that has overall administrative responsibility for both M and I projects and CHCs.

B. Estimation

Neonatal intensive care units are aimed at low-birth weight births, and community health centers, organized family planning clinics and the WIC program are aimed at the poor. It follows that the impact of the use of neonatal intensive care on neonatal mortality is larger, the larger the fraction of low-birth weight births. Also, the impact of the use of inputs provided by public programs is larger the larger the fraction of poor women.[4] To account for these effects, we interact the neonatal intensive care measure with the race-specific fraction of low-birth weight births. Under the assumption of equal use rates by white and black light neonates, the resulting variable can be interpreted as the race-specific number of inpatient days in neonatal intensive care units per birth. Similarly, the WIC and BCHS program measures are interacted with the race-specific fraction of women aged 15 to 44 with family income less than 200 percent of the poverty level, and the teenage family planning measure is interacted with the race-specific fraction of women 15 to 19 with family income less than 200 percent of the poverty level. The interacted teenage family planning measure gives the race-specific number of teenage users as a percentage of all race-specific teenagers. Under the assumption of equal use rates by poor white and black women, the other interacted poverty variables can be interpreted as the race-specific number of users of a given program per thousand race-specific women aged 15 to 44.

The neonatal mortality Equations (1) and (2) are fitted using a two-stage least squares procedure for the reasons discussed in Section II. 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).[5] If the null hypoth-

4. For a more detailed explanation of this proposition see Corman, Joyce, and Grossman (1985).

5. Wu's T_2 statistic can be used to test whether a set of right-hand-side regressors is correlated with the residuals. The following description is based on the discussion by Nakamura and Nakamura (1981). Assume the following model:

(F-1) $D = HB + X_1C + u$ (structural equation)

(F-2) $H = X_1P_1 + X_2P_2 + V = XP + V$ (reduced form equation),

where D is an $N \times 1$ vector of observations on the neonatal mortality rate; H is an $N \times G$ matrix of stochastic health inputs which may also include birth weight; X_1 is an $N \times K_1$ matrix of

Table 1
Definitions of Variables[a]

Variable Name	Definition
Neonatal mortality*	Three-year average neonatal mortality rate centered on 1977; deaths of infants less than 28 days old per 1,000 live births ($\mu_w = 8.837, \sigma_w = 1.595, \mu_b = 16.387, \sigma_b = 3.299$)
Low birth weight*	Three-year average percentage of low-birth weight (2,500 grams or less) live births centered on 1977 ($\mu_w = 5.992, \sigma_w = .741, \mu_b = 13.016, \sigma_b = 1.228$)
Prenatal care*[e]	Three-year average percentage of live births for which prenatal care began in the first trimester (first three months) of pregnancy centered on 1977 ($\mu_w = 78.111, \sigma_w = 8.290, \mu_b = 59.359, \sigma_b = 10.236$)
Abortion	Three-year average state-specific resident abortion rate centered on 1976; abortions performed on state residents per 1,000 women aged 15–44 in the state ($\mu_w = 24.969, \sigma_w = 8.716, \mu_b = 24.754, \sigma_b = 8.603$)
Teen family planning*[b]	Percentage of women aged 15–19 with family income less than 200 percent of the poverty level in 1975 who use organized family planning services in 1975 ($\mu_w = 35.747, \sigma_w = 25.265, \mu_b = 44.613, \sigma_b = 17.966$)
BCHS Projects[c]	Sum of maternity patients in maternal and infant care (M and I) projects and female users aged 15–44 of community health centers (CHCs) in

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 1976–78.
b. Variable is available for whites and nonwhites as opposed to whites and blacks.
c. Since numerator of this variable is not race-specific, denominator also is not race-specific.
d. Variable is available for nonblacks and blacks as opposed to whites and blacks.

Table 1 (*continued*)

Variable Name	Definition
	1976 per 1,000 women aged 15–44 with family income less than 200 percent of the poverty level in 1975; numerator termed Bureau of Community Health Services (BCHS) female project users (μ_w = 37.808, σ_w = 153.103, μ_b = 54.929, σ_b = 141.517)
WIC (maternal nutrition program)	State-specific number of eligible pregnant women served by the Special Supplemental Food Program for Women, Infants, and Children (WIC program) per 1,000 state-specific eligible women in 1980 (μ_w = 262.894, σ_w = 77.983, μ_b = 267,931, σ_b = 74.089)
Neonatal intensive care[c]	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 low-birth weight births centered on 1977 (μ_w = 10.709, σ_w = 5.817, μ_b = 11.538, σ_b = 7.395)
Smoking	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 = .351)
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)
Poverty*[d]	Fraction of women aged 15–44 with family income less than 200 percent of the poverty level in 1980 μ_w = .266. σ_w = .877, μ_b = .549, σ_b = .936)

e. In counties where prenatal care was unknown for more than 50 percent of births, we consider the value as missing. Prenatal care data were missing for 83 counties in the white sample and 45 counties in the black sample. For counties with known values, prenatal care was estimated on the basis of the percentage of women employed, the percentage of women with at least a high school education, and the percentage of poor women. The coefficients were then used to generate values for the unknown counties. Note that the coefficients of production functions estimated by a two-step procedure, one which did not repredict prenatal care for the unknown counties, were almost identical to the coefficients presented in Section IV.

esis 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 techniques, on Equations (1) and (2). A comparison of Wu statistics for Equations (1) and (2) allows us to examine whether birth weight is a reasonable proxy for the health endowment. It should be noted that in both OLS and TSLS estimates of the production functions, we use a linear function form[6] and use weights appropriate for aggregate data.

In the first stage of our two-stage estimation procedure, birth weight, prenatal care, abortion, and neonatal intensive care use are predicted on the basis of variables that are used in Corman and Grossman's (1985) reduced form neonatal mortality estimates. Predicted values of these four endogenous variables are then entered into the neonatal mortality rate equations. The reduced form regressors are as follows: the race-specific fraction of high-risk women; the race-specific fraction of women aged 15 through 44 with family income less than 200 percent of the poverty level; the race-specific fraction of women 15 through 49 with at least a high school education; the number of hospitals with Level II or Level III neonatal intensive care units per thousand women aged 15 through 44 (neonatal intensive care availability); the number of abortion providers (public hospitals, private hospitals, nonhospital clinics, and office based physicians) per thousand women aged 15 through 44 (abortion availability); the number of organized family planning clinics per thousand women aged 15 through 44 with family income less than 200 percent of the poverty level (family planning availability); the number of BCHS projects per thousand women aged 15 through 44 with family income less than 200 percent of the poverty level (BCHS project availability); a dichotomous variable that equals one if a county is in a state

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exogenous health determinants; X_2 is an $N \times K_2$ matrix of demand determinants; u and V are $N \times 1$ and $N \times G$ matrices of disturbances; and B, C, P_1, P_2 are $G \times 1, K_1 \times 1, K_1 \times G$, and $K_2 \times G$ matrices of unknown constants. The test of whether $E(Hu) = 0$, where E is the expected value operator, can be expressed as a test of whether u and V are correlated $[E(Vu) = 0]$. Following Hausman's (1978) derivation, Equation (F-1) is rewritten as:

(F-3) $D = HB + X_1C + V\alpha + u$

Wu's T_2 test can be expressed as a test of whether $\alpha = 0$. This is a straightforward F-test on a set of linear restrictions. Acceptance of the null hypothesis that $E(Vu) = 0$ is equivalent to the restriction that $\alpha = 0$.

6. We choose a linear rather than a logistic functional form because linear coefficients are more easily interpreted. Maddala (1983, 30) argues that a linear form is appropriate for large, aggregate probability samples such as ours. Preliminary results obtained with logistic and log-linear specifications did not differ in a qualitative sense from those presented in Section III. The linear specification rules out an investigation of the optimal input mix (the combination of inputs that minimizes the cost of producing a given level of infant health), but this is not the focus of our empirical research.

that covers prenatal care for all first-time pregnancies under Medicaid to financially eligible women; a dichotomous variable that equals one if a county is in a state that covers prenatal care for first-time pregnancies under Medicaid only if no husband is present or if the husband is present but unemployed and not receiving unemployment compensation; a dichotomous variable that equals one if a county is in a state that covers prenatal care for first-time pregnancies under Medicaid only if no husband is present;[7] the likelihood that the newborn care received by the infant of a low-income woman will be financed by Medicaid measured by a dichotomous variable that equals one if a county is in a state in which Medicaid pays for newborn care under the mother's Medicaid number or does not pay for care under the mother's number but allows pregnant women to register their unborn children with Medicaid; and the average annual Medicaid payment per adult recipient in AFDC families. All of these variables are county-specific except for the neonatal intensive care and Medicaid measures which are state-specific.[8]

Ideally, in our two-stage procedure, we would treat all right-hand variables in Equation (1) and all variables except the fraction of high-risk women in Equation (2) as endogenous. Doing so, however, would create severe problems of multicollinearity and would tax the data to an inordinate degree. The public program input measures are all treated as exogenous in the estimation procedure. This procedure can be justified because these programs are used by poor women as opposed to all women, and the programs are relatively new. Joyce's (1985) empirical estimates of input demand functions suggest that differences in their use among counties are governed to a large extent by differences in their availability. Technically, our procedure is analogous to viewing the capital input in a firm's production function as fixed in the short run by varying among firms for historical reasons.[9] 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 Corman, Joyce, and

7. The omitted category for the three variables that characterize the eligibility of first-time pregnant women for prenatal care under Medicaid pertains to states that cover no first-time pregnancies because their AFDC programs do not recognize "unborn children."

8. Note that in the case of abortion and neonatal intensive care, both availability and use measures are present. In each case own availability is a powerful prediction of own use. It has the largest *t*-ratio in the input demand function and therefore the highest partial correlation coefficient with the dependent variable. These results strengthen our confidence in the abortion and neonatal intensive care availability measures.

9. We do not employ public program use measures to predict prenatal care, abortion, neonatal intensive care, and birth weight in the first stage. Instead, public program availability measures are employed, except in the case of WIC where there is no availability measure. Production functions obtained with public program use measures as first stage predictors were similar to those presented in Section IV.

Grossman (1985). Our procedure is based on the reasonable assumption that prenatal and neonatal input availability measures have zero coefficients in the cigarette demand function.[10]

Rosenzweig and Wolpin (1986) point out that it may be inappropriate to treat the availability of government funded programs such as BCHS projects and organized family planning as exogenous. They show that the government may allocate program resources systematically across areas on the basis of health endowments, thus making availability measures of such inputs endogenous. They present empirical evidence in favor of this proposition using data on the distribution of family planning and health facilities among villages in one province in the Philippines, although they do not estimate health production functions. In a separate paper, Rosenzweig and Wolpin (1984) also present evidence that women with low health endowments migrate to areas where program availability is high based on the use of a maternal and child health program in a small village in Colombia.

To examine the sensitivity of our estimated production functions to the potential endogeneity of BCHS and family planning availability, we deleted these two variables from the set of reduced form regressors. The resulting production functions were very similar to those presented in Section IV. This suggests that the endogeneity of input availability may not be a problem in our data, particularly because BCHS projects and organized family planning clinics are funded in part by state and local governments. It may be more difficult to allocate resources across areas based on health endowments if more than one level of government is involved in the decision-making process. Note that the rules governing the eligibility of low-income women for Medicaid financing of prenatal care and newborn care were made solely by the states in 1977. This reduces the possibility of significant correlations between the Medicaid variables and the health endowment. Moreover, Rosenzweig and Wolpin's results pertain to less developed countries. Whether they generalize to a developed country such as the U.S. and to subpopulations such as teenagers is unknown.

IV. Results

Ordinary least squares (OLS) and two-stage least squares (TSLS) estimates of the black and white neonatal mortality production functions are presented in Table 2. The first set of regressions for each race (A1, A2, B1, and B2) excludes the endogenous risk factor of low birth weight. As discussed in Section I, the substitution of low birth weight by its

10. 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.

structural determinants yields what we have termed the quasi-structural production function. The remaining regressions show the direct effect of an input on neonatal mortality by holding constant the percentage of low-birth weight births.

The TSLS regression coefficients of prenatal care, abortion, and neonatal intensive care are substantially larger in absolute value than their corresponding OLS coefficients in the quasi-structural regressions (A1, A2, B1, and B2). The fact that this difference is greater for blacks is a noteworthy result for as Rosenzweig and Schultz (1983a) have noted, 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. For instance, the prenatal care coefficients estimated by OLS in regressions (A1) and (B1) suggest that prompt initiation of prenatal care is more effective in lowering neonatal mortality among whites than it is for blacks. The TSLS estimates reveal just the opposite [regressions (A2) and (B2)]. This implies that remedial behavior among black pregnant women may be an important response to information regarding the health of the fetus.

Based on the Wu test, the null hypothesis of zero correlation between the health inputs and the disturbance term can only be accepted when the percentage of low-birth weight births is held constant [regressions (A3), (A4), (B3), and (B4)]. This suggests that the unobserved health endowment is effectively controlled for by this endogenous risk factor. Consequently, the TSLS coefficients should be used to measure the total effect of an input on neonatal mortality, whereas the OLS estimates are appropriate when gauging an input's direct effect. Hence, further discussion of the results will focus on regressions (A2), (B2), (A3), and (B3).

All four of the aforementioned equations are significant at the 1 percent level, as indicated by F-values. For whites, in the TSLS estimate of the quasi-structural model (A2), all coefficients have their predicted signs, and five of the eight are highly significant. For the comparable black estimates (B2), seven of the eight coefficients have predicted signs, although not as many of the variables have strong significance levels. In the white OLS equation holding birth weight constant (A3), all coefficients have correct signs and have *t*-values greater than one in absolute value. In the comparable black equation (B3), all coefficients have correct signs, although fewer are significant as compared to the white equation. Altogether, the model works well in predicting variations in neonatal mortality rates based on medical program usage.

The effects of WIC, abortion, and prenatal care on race-specific neonatal mortality fall in absolute value when birth weight is held constant [see regressions (A2), (A3), (B2), and (B3)]. For blacks, the abortion coefficient falls 65 percent. Nevertheless, its risk-specific effect is still greater than the

Table 2
Regression Results[a]

	Whites				Blacks			
	A1	A2	A3	A4	B1	B2	B3	B4
	OLS	*TSLS*	*OLS*	*TSLS*	*OLS*	*TSLS*	*OLS*	*TSLS*
Constant	7.478	9.831	7.223	5.512	17.913	24.929	4.647	8.600
	(4.98)	(5.60)	(7.24)	(1.78)	(2.97)	(3.60)	(1.87)	(1.04)
Teen family planning*	−.021	−.011	−.025	−.014	−.024	−.025	−.029	−.024
	(−2.01)	(−1.03)	(−2.64)	(−1.32)	(−1.26)	(−1.27)	(−1.66)	(−1.37)
Maternal nutrition program (WIC)*	−.002	−.006	−.002	−.004	−.004	−.009	−.001	−.008
	(−.78)	(−2.34)	(−1.12)	(−1.46)	(−.99)	(−2.19)	(−.44)	(−2.06)
Neonatal intensive care*[b]	−.096	−.467	−.219	−1.176	−.306	−.475	−.356	−.772
	(−.63)	(−.93)	(−1.48)	(−2.26)	(−1.73)	(−.89)	(−2.22)	(−1.60)
Abortion[b]	−.029	−.033	−.025	−.021	−.044	−.085	−.030	−.044
	(−4.19)	(−3.57)	(−3.72)	(−2.13)	(−1.53)	(−2.01)	(−1.51)	(−1.58)

Prenatal care*[b]	−.045	−.076	−.024	−.016	−.030	−.117	−.008	−.026
	(−5.40)	(−5.14)	(−2.99)	(−.79)	(−1.69)	(−2.81)	(−.49)	(−.56)
BCHS projects*	.0003	−.0002	−.002	−.001	−.002	−.001	−.006	−.002
	(.23)	(−.13)	(−1.96)	(−.82)	(−.77)	(−.42)	(−2.37)	(−.96)
Smoking[b]	.535	.555			.600	1.045		
	(4.65)	(4.76)			(1.06)	(1.86)		
High risk women*	5.843	7.366			−4.263	−13.138		
	(2.05)	(2.50)			(−.52)	(−1.33)		
Low birth weight*[b]			.781	1.046			1.121	1.026
			(9.50)	(3.78)			(8.39)	(2.18)
R-squared	.108		.184		.036		.195	
F	10.16[c]	9.78[c]	21.51[c]	9.35[c]	1.64[e]	2.66[c]	12.05[c]	3.55[c]
WU test F	2.61[d]		1.57[e]		3.86[c]		.19[e]	

a. Asymptotic t-ratios in parentheses. The critical t-ratios at the 5 percent level are 1.64 for a one-tailed test and 1.96 for a two-tailed test. An asterisk next to a variable means it is race-specific.
b. Endogenous in TSLS equations.
c. Significant at the 1 percent level.
d. Significant at the 5 percent level.
e. Not significant at the 5 percent level.

corresponding white one, although its significance level is only 10 percent.[11] WIC and prenatal care reduce neonatal mortality solely by reducing the percentage of low-birth weight births. Put differently, the estimated effects are zero once the risk factors are held constant. For whites, the WIC coefficient falls by 67 percent, the abortion coefficient falls by 24 percent, and the prenatal care coefficient falls by 70 percent when the risk factor is included in the set of regressions. In spite of these reductions, the prenatal care and abortion coefficients retain their significance at the 1 percent level.

The above results imply that expansions in prenatal care use lower risk-specific death rates for whites but not for blacks. These findings suggest that the quality of prenatal care is positively related to the quality and quantity of perinatal and newborn care received by white mothers. The absence of this relationship for black women is plausible since the early initiation of prenatal care by these women is a recent phenomenon. For instance, in 1969, 72 percent of white women and 43 percent of black women started their prenatal care in the first trimester of pregnancy (Taffel 1978). The corresponding figures in 1977 were 77 percent of white women and 59 percent of black women (see Table 1).

In the case of abortion, the above results suggest that the process of fetal selection encouraged by abortion may be improving the survivability of risk-specific births as well as reducing the incidence of low birth weight. The former effect may be the result of births being better planned or "more wanted." That is, births that are not averted may receive more of the unmeasurable inputs such as better nutrition and higher quality care that enhance the health and survivability of newborns of a given birth weight.

The sizable risk-specific BCHS project use and family planning use coefficients reflect positive relationships between these inputs and the percentage of low-birth weight births. The BCHS result suggests that poor women who obtain prenatal care from M and I projects or community health centers probably do not start to receive care until fairly late in their pregnancies. Moreover, these women may have poorly endowed birth outcomes. Consequently, the receipt of care from BCHS projects does not lower the incidence of low birth weight, but it appears to raise the quantity and quality of perinatal and newborn care.

The same argument may apply for organized teenage family planning use. Chamie et al. (1982) report that counties that serve a high proportion of women at risk of pregnancy are more likely to provide gynecological and prenatal care than counties that serve a smaller proportion of such women. Jones, Namerow, and Philliber (1982) find that more than half of the first-time clients of a large metropolitan family planning clinic previously

11. Statements concerning statistical significance in the text are based on one-tailed tests except when the estimated effect has the "wrong sign." In this case two-tailed tests are used.

had been pregnant. In short, a rise in the proportion of low-income women who use organized family planning services may be indicative of a population that has been integrated into a network of prenatal and perinatal care. The births to these women may still be problematic (that is, premature or light), but with better support and care their infants are more likely to survive.

One way to gauge the magnitudes of the estimated relationships between infant health inputs and outcomes is to apply the relevant coefficients to national trends in the inputs between 1964 and 1977. This exercise allows us to shed light on the sources of the rapid decline in the U.S. neonatal mortality rate starting in 1964 by computing the contribution of each input to the downward trend in neonatal mortality. The extrapolations end in 1977 because the regressions pertain to that year. In the period at issue the white neonatal mortality rate declined by 7.5 deaths per thousand live births, from 16.2 to 8.7, or by 46 percent. The black neonatal mortality rate declined by 11.5 deaths per thousand live births, from 27.6 to 16.1, or by 42 percent.

The results of estimating the implied changes in white and black neonatal mortality rates due to trends in the inputs are shown in Panels A and B of Table 3, respectively. The direct effect is obtained from the OLS neonatal mortality rate production function that includes birth weight as a regressor. The total effect is obtained from the TSLS estimate of the quasi-structural production function. Subtraction of the former from the latter yields the indirect effect. Thus, the indirect effect shows the reduction in the neonatal mortality rate due to an increase in one of the health inputs between 1964 and 1977 that operates via a reduction in the percentage of low-birth weight births.[12]

For whites, the statistical analysis "explains" 25 percent of the decline in neonatal mortality. The increase in abortion makes the largest contribution to the decline (0.8 births per thousand live births) followed by prenatal care and WIC (0.4 deaths per thousand live births each) and neonatal intensive care (0.1 deaths per thousand live births). Prenatal care and WIC each have a substantial indirect effect which accounts for approximately two-thirds of the total effect of the input in question.

For blacks, the statistical analysis explains 56 percent of the decline in neonatal mortality. As in the case of whites, abortion makes the largest

12. Consider the following model:

$$d = \alpha_0 + \alpha_1 x + \alpha_2 k$$
$$k = \beta_0 + \beta_1 x,$$

where k is the percentage of low-birth weight births and x is any input. If Δx is the change in x between 1964 and 1977, then the direct effect is $\alpha_1 \Delta x$, the indirect effect is $\beta_1 \alpha_2 \Delta x$, and the total effect is $(\alpha_1 + \beta_1 \alpha_2)\Delta x$. Note that the indirect effect of neonatal intensive care is restricted to be zero because conceptually it is not a cause of low birth weight.

Table 3
Contribution of Selected Factors to Reductions in Neonatal Mortality Rates, 1964–77[a]

Factor	Panel A: Whites[b]			Panel B: Blacks[c]		
	Direct	Indirect	Total	Direct	Indirect	Total
Organized family planning	.191	−.107	.084	.610	−.084	.526
WIC	.143	.282	.425	.148	1.182	1.330
BCHS project use	.022	−.020	.002	.182	−.152	.030
Neonatal intensive care	.140	—	.140	.534	—	.534
Abortion	.624	.200	.824	.743	1.366	2.109
Prenatal care	.137	.297	.434	.133	1.816	1.949
Total explained reduction[d]			1.9			6.5
Percentage explained			25.3			56.5

a. Reduction in deaths per 1,000 live births. Negative sign denotes a predicted increase.
b. Direct effect from regression (A3). Total effect from regression (A2). Subtraction of the former from the latter gives the indirect effect.
c. Direct effect from regression (B3). Total effect from regression (B2). Subtraction of the former from the latter gives the indirect effect.
d. Rounded to one decimal.

contribution (2.1 deaths per thousand live births) followed by prenatal care (1.9 deaths per thousand live births), WIC (1.3 deaths per thousand live births), and neonatal intensive care and organized family planning (0.5 deaths per thousand live births each). Prenatal care, WIC, and abortion have sizable indirect effects, and in each case the indirect effect is larger than the direct one.

Some caution should be exercised in interpreting the results in Table 3 because an increase in abortion use, for example, due to an increase in abortion availability is likely to cause organized family planning use to fall. Put differently, these computations do not provide the reduced form effects that are required to evaluate fully the contributions to reductions in neonatal mortality between 1964 and 1977 of the increased availability of the inputs considered here. Nevertheless, they do provide insight with regard to the role of the expansion in the use of one input with all other inputs held constant. Caution also should be exercised because the results pertain to the actual benefits in terms of neonatal mortality of increases in the inputs in the period considered rather than to the potential benefits of future expansions. Note, however, that a ranking of the magnitude of the effect of each input

based on a 10 percent increase in its 1977 value is very similar to the ranking presented in Table 3.

A final caveat is that, although our production functions include a measure of the quantity of neonatal intensive care, they exclude a measure of its quality. Even if the state-of-the-art in neonatology is fixed in the cross-section, clearly it is not fixed over time. In light of the rapid advances in perinatal and neonatal science since 1964, we undoubtedly understate the growth in a comprehensive measure of the neonatal intensive care input.[13]

It is notable that practically all the black regression coefficients in Table 2 and all the estimated black effects in Table 3 exceed the corresponding white coefficients or white effects. This is a key finding because it suggests that the inputs at issue have the potential to reduce the excess mortality rate of black babies, an important goal of public health policy in the U.S. for a number of years. It also is notable that the combined contribution of abortion, prenatal care, and neonatal intensive care to the reduction in black neonatal mortality (4.5 deaths per thousand live births) exceeds the combined contribution of WIC, BCHS project use, and organized family planning use (1.8 deaths per thousand live births). This is an important result because the first three inputs are used by all segments of the population, while the last three are used by the poor. It implies that blacks may benefit more from developments that affect neonatal mortality rates in all segments of the population than from programs that are targeted at the poor.

V. Discussion

Our results underscore the qualitative and quantitative importance of abortion and prenatal care services in black and white birth outcomes. We find that black neonatal mortality rates are more sensitive to the use of these basic health inputs than are white neonatal mortality rates. We also present evidence with respect to the potential importance of neonatal intensive care in the determination of neonatal mortality rates, particularly for blacks. Neonatal intensive care ranks fourth in importance behind prenatal care, abortion, and WIC in explaining declines in both white and black neonatal mortality between 1964 and 1977. Given the absence of cross-sectional or time-series indexes of the quality of care, the impact of neonatal intensive care undoubtedly is understated. Clearly the

13. In addition to the caveats mentioned in the text, it should be noted that the contribution of abortion may be overstated because all states had laws that outlawed abortion except when it was necessary to preserve a pregnant woman's life in 1964. Many illegal abortions were performed in that period. If illegal abortions affected neonatal mortality, we overstate the abortion effect in Table 4.

development of more comprehensive measures of this input deserves high priority on an agenda for future research.

These results provide suggestive, although far from definitive, explanations of the slowdown in the downward trend in neonatal mortality in the early 1980s. The abortion rate of white women reached a peak in 1980 and was stable between 1980 and 1981. The abortion rate of black and other nonwhite women peaked in 1977 and declined every year since then with the exception of 1980 (Bureau of the Census 1984). The percentage of white women who began prenatal care in their first trimester of pregnancy fell between 1980 and 1981, and the percentage of black women who began prenatal care in their first trimester fell between 1981 and 1982 (Ingram, Makuc, and Kleinman 1986). The introduction and diffusion of new techniques in neonatology slowed appreciably in the late 1970s and early 1980s (McCormick 1985).

The role of public policy in the above developments is not clearcut. In part the recent trend in abortion may reflect the end of the diffusion of a relatively new contraceptive technique. In part it also may reflect the Hyde Amendment which has banned Federal funding of abortions under Medicaid, except in cases when the woman's life was in danger, since September 1977 (except for the months of February through August 1980). Medicaid cutbacks may have made it more difficult for pregnant low-income women to initiate prenatal care in the first trimester, although the recession of 1981–82 also may have played a role. Our results identify the use of the WIC program as a much more important determinant of black neonatal mortality than the use of CHCs, M and I projects, or organized family planning services. Declines or modest increases in the percentage of poor black pregnant women serviced by WIC may have retarded the rate of decline in black neonatal mortality, but definitive recent trends in this statistic are not available. In summary, more research is required to provide a fuller explanation of the behavior of the U.S. neonatal mortality rate since 1980. Our study represents a useful first step in this process.

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