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BIRTH OUTCOME PRODUCTION FUNCTIONS IN THE U.S.

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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. The empirical analysis is based on a cross section of U.S. counties in 1977, and the incidence of low birth weight (2,500 grams or less) is employed as an intermediate outcome. This allows us to examine the extent to which prenatal inputs operate directly on neonatal mortality and also allows us to examine their indirect effects on mortality rates through low birth weight. Since mothers with poor endowed birth outcomes will attempt to offset these unfavorable prospects by utilizing more health inputs, major emphasis is placed on two-stage least squares estimates of the production function. 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.

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BIRTH OUTCOME PRODUCTION FUNCTIONS IN THE U.S.

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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.[1] 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.

The purpose of this study is to examine the effects of the utilization of these important health inputs on race-specific neonatal mortality. Specifically, we use a production function approach to examine the relationship between health outcomes and the use of these important health inputs. Although a number of studies have treated subsets of these inputs,[2] none has entered them together in a multivariate birth outcome equation estimated with data covering a large percentage of all births in the U.S.

Our empirical analysis 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 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.

I. Analytical Framework

Following Corman and Grossman (1985) and the theorectical 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.

Maximization of the parents' utility function subject to production and resource constraints generates a demand function for survival in which the survival probability or its complement, the neonatal mortality rate, is related to input prices (whose direct and indirect cost components are negatively related to input availability), efficiency, income, and tastes. The interaction between the survival demand and the production functions determines demand functions for medical care and other endogenous inputs. These demand functions depend on the same set of variables as the demand function for survival.

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

system of equations is designed to obtain estimates of the direct and indirect (through low birth weight) effects of five basic health inputs on neonatal mortality. These inputs are prenatal medical and nonmedical care (including appropriate nutrition), perinatal and neonatal care, the use of abortion services, the use of contraceptive services, and maternal cigarette smoking.[3] Although the equations in the model have meaningful interpretations at the family level, the empirical analysis pertains to county-level data for the year 1977. Differences between micro effects and those at a more aggregate level are noted where relevant.

The basic model consists of the following nine equations:

$$d = f_1(n, m, a, c, b, e) \qquad (1)$$

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

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

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

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

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

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

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

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

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

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

$$d = f_{10}(p, y, x, e) \qquad (10)$$

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

Corman and Grossman (1985) estimated the reduced form neonatal mortality rate equation (10) using the county data base described in Section II. Here we focus on the estimation of the structural neonatal mortality rate production function (1) and the quasi-structural mortality production function obtained by substituting equations (2), (3), and (4) into equation (1):

$$d = f_{12}(n, m, a, c, s, x, e) . \quad (12)$$

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

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

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

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

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

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

Certain restrictions must be imposed to insure that each equation in the system satisfies rank and order conditions for identification. The most important restrictions are: (1) neonatal care has no impact on low birth weight; (2) cigarette smoking affects neonatal mortality only through its effect on low birth weight; (3) prematurity affects mortality only through low birth weight; and (4) cigarette smoking has no effect on prema-

turity. With regard to the first restriction, decisions to use neonatal care services are made after birth, and low birth weight causes more use of these services.[7] For the other assumptions, there is considerable supporting evidence.[8]

It should be noted that certain prenatal inputs may have different interpretations, depending on whether a micro data set or aggregate data set is used. Differences arise at the county level because abortion availability, for example, varies among counties and because the underlying frequency distribution of health endowments (the distribution that would be observed in the absence of abortion) also varies. 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.[9] 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. This underscores the need to treat the abortion rate as an endogenous variable.

Some persons would question the inclusion of the use of abortion services, prenatal care services, and contraceptive services in the basic neonatal mortality rate production function given by equation (1). These persons would argue that the inputs just mentioned operate on the observed

neonatal mortality rate solely by lowering the fraction of light births, which is entered in the equation. This is a plausible proposition in the presence of complete measures of the quantity and quality of perinatal and neonatal care. But if such measures are absent or incomplete, as they are in our data, and are positively correlated with abortion, prenatal care, and contraception, the latter inputs can have negative impacts on birth weight-specific neonatal mortality. For instance, consider women who have a relatively high propensity to identify and abort defective fetuses. Subsequent prenancies of such women are likely to be better planned ("more wanted") and to receive larger quantitites of unmeasured inputs that improve birth outcomes.

II. Empirical Implementation

A. Data and Measurement of Birth Outcomes

We have constructed the data base from a variety of sources which are described in detail in Corman and Grossman (1985). Briefly, neonatal deaths by race for the years 1976 through 1978 were taken from the National Center for Health Statistics (NCHS) Mortality Tape. Births by race for those years were obtained from the NCHS Natality Tape. White and black births by weight and month in which prenatal care began (one characteristic at a time as opposed to conditional distributions) for the same three-year period were also taken from the Natality Tape. Information on abortion rates and on the use of organized family planning services was acquired from the Alan Guttmacher Institute (the technical assistance division of Planned Parenthood). Hospital inpatient days in neonatal intensive care

units were derived from the American Hospital Association and from Ross Laboratories. Socioeconomic characteristics of counties were taken from the Area Resource File.

The analysis focuses on the neonatal mortality rate as opposed to the postneonatal rate. This is because many postneonatal deaths are due to infectious diseases and accidents, causes which are insensitive to most prenatal inputs. Separate regressions are fitted for white and black birth outcomes, rather that a non-race 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.

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.[10] In addition to selecting large counties, we attenuate random elements by employing three-year averages of the race-specific neonatal mortality rate and percentage of low-birth weight births, weighting regressions by the square root of the race-specific total number of births.

B. Measurement of Right-Hand Side Variables

Whenever possible, county- and race-specific variables are employed in the regressions. Race-specific variables are denoted by an asterisk. Table 1 contains definitions of the variables, and Table 2 contains their means and standard deviations. When possible, variables pertain to one or more years in the 1975-1978 period. The neonatal intensive care input pertains to 1979 and the WIC input and the poverty variable pertain to 1980. In these cases the assumption is made that the 1975-1978 measure is highly correlated with the one actually used. Table 2 distinguishes between raw and interaction variables, discussed below.

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.

The use of prenatal medical care services is given by a three-year average of the percentage of live births for which prenatal care began in the first trimester (first three months) of pregnancy. This measure is more desirable than the number of prenatal care physician visits for two reasons. First, the latter variable is mechanically related to the length of the pregnancy. Also, the decision to initiate prenatal care is made by

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
Low Birth Weight*	Three-year average percentage of low-birth weight (2,500 grams or less) live births centered on 1977
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
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
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
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 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
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
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

(continued on next page)

Table 1 (concluded)

Variable Name	Definition
Smoking	State-specific daily number of cigarettes smoked per adult 18 years and older in 1976
High Risk Women*[b]	Number of women 15-19 and 40-44 as a fraction of women 15-44 in 1975
Poverty*[d]	Fraction of women aged 15-44 with family income less than 200 percent of the poverty level in 1980

[a]An asterisk (*) next to a variable means that it is race-specific. All variables are county-specific unless otherwise indicated.

[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.

[e]In counties where prenatal care was unknown for over 50 percent of births, we considered 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 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 III.

Table 2

Means and Standard Deviations of Variables[a]

Variable	Raw Variable		Interacted Variable[b]	
	Mean (1)	Standard Deviation (2)	Mean (1)	Standard Deviation (2)
		WHITES		
Neonatal Mortality*	8.837	1.595		
Low Birth Weight*	5.992	.741		
Prenatal Care*	78.111	8.290		
Abortion	24.969	8.716		
Teen Family Planning*	35.747	25.265	9.067	6.290
BCHS Projects*[c]	37.808	153.103	10.770	48.149
WIC (Maternal Nutrition Program)*[c]	262.894	77.983	70.836	33.111
Neonatal Intensive Care*[d]	10.709	5.817	.641	.385
Smoking	7.416	.511		
High Risk Women*	.335	.022		
Poverty*	.266	.877		
		BLACKS		
Neonatal Mortality*	16.387	3.299		
Low Birth Weight*	13.016	1.228		
Prenatal Care*	59.359	10.236		
Abortion	24.754	8.603		
Teen Family Planning*	44.613	17.966	24.176	9.656
BCHS Projects*[c]	54.929	141.517	30.277	69.440
WIC (Maternal Nutrition Program)*[c]	267.931	74.089	147.825	51.259
Neonatal Intensive Care*[d]	11.538	7.395	1.501	1.011
Smoking	7.486	.351		
High Risk Women*	.350	.026		
Poverty*	.549	.936		

[a] An asterisk denotes a race-specific variable. The white data pertain to 677 counties, while the black data pertain to 357 counties. Means and standard deviations are weighted by the race-specific total number of births in the period 1976-1978.

[b] Family Planning, BCHS Projects, and WIC variables are interacted with the race-specific fraction of poor women (Poverty) in the county. The Neonatal Intensive Care variable, which is state-specific, is interacted with the race-specific and state-specific fraction of low-birth weight births.

[c] Raw variable is not race-specific. Interacted variable is race-specific under the assumption of equal use rates by white and black poor women.

[d] Raw variable is not race-specific. Interacted variable is race-specific under the assumption of equal use rates by white and black light neonates.

the mother, while the number of visits given that care has begun is determined more by medical protocol.

The use of abortion services is reflected by a three-year average of the state-specific resident abortion rate (abortions performed on state residents per thousand women aged 15 to 44 in the state). Abortion rates are centered on 1976 rather than 1977 because Grossman and Jacobowitz's (1981) estimates suggest that abortions performed in the first half of a given year affect the neonatality mortality rates in the second half of the year.

Organized family planning use is given by the percentage of teenagers with family income less than 200 percent of the poverty level in 1975 who used organized family planning clinics in 1975. The denominator pertains to poor women because the relevant public program is aimed at the poor. The numerator refers to teens for two reasons. First, race-specific usage data were only available for this age group. Second, research indicates that use of family planning services by teenagers may have larger impacts on neonatal mortality than use of these services by older women.[11]

The use of the maternal and infant care program and the community health center program is given by the sum of maternity patients in M and I projects and female users aged 15 to 44 of community health centers in 1976 per thousand poor women aged 15 to 44 in 1975. Note that we, again, use poor women in the denominator since we are referring to programs aimed at the poor. This variable is termed BCHS (Bureau of Community Health Services) project use because the Bureau of Community Health Services (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.

There is a possibility that BCHS project use may be a causal determinant of the use of prenatal care services by low-income women. Thus, the inclusion of both variables in the same regression may mask the effect of the latter variable on infant health outcomes. This is not a serious problem because only 20 percent of the counties in the white regressions and 30 percent of the counties in the black regressions had at least one BCHS project in 1976. More importantly, there is a strong rationale for including both variables in the production function because prenatal care services delivered to low-income women as reflected by BCHS project use may have a different impact on birth outcomes than prenatal care services delivered to other women. In addition, BCHS project users may receive more and better obstetrical and newborn care than other poor women.

The nutritional adequacy of diets of pregnant low-income women is measured by the number of state-specific eligible pregnant women served by the WIC program in 1980 per thousand state-specific eligible women also in 1980. The use of neonatal intensive care services is measured by the sum of the state-specific number of hospital inpatient days in Level II and Level III neonatal intensive care units in 1979 divided by a state-specific three-year average of the number of low-birth weight births centered on 1977.[12] The denominator pertains to light neonates because they are the primary users of the input at issue. We use a state-specific variable because many states have developed either informal or formal regional referral networks for ill neonates, suggesting that the market area for

this care is larger than the county.

Our smoking variable, the state-specific daily number of cigarettes smoked per adult 18 years and older in 1976, was taken from Lewit (1982) who estimated it from his micro-level study of the demand for cigarettes with Coate (Lewit and Coate 1982). Specifically, Lewit and Coate used the 1976 Health Interview Survey to estimate micro demand functions for cigarettes based on income, price, educaton, age, sex and race. Their procedure capitalizes on cross-sectional differences in the price of cigarettes primarily due to differences in state excise tax rates. Lewit applied the coefficients of the fitted demand functions to state means of the independent variables to arrive at the figures used here. The advantage of Lewit's variable over the readily-available tax-paid sales per state is that his measure adjusts for the substantial "bootlegging" of cigarettes at both the individual and the group level. Because of this smuggling, data from tax-paid sales underestimate consumption in high-tax states and overestimate it in low-tax states.

Our final variable represents the exogenous risk factor, x, in equation (12). For this variable, we use the number of women who are either teens or in their forties as a fraction of all women of childbearing age. These are the age groups considered most at risk for negative birth outcomes.

C. 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 is the

fraction of low-birth weight births. Also, the use of inputs provided by public programs is larger the larger is the fraction of poor women.[13] 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.[14]

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

endowment, however, these variables can serve as instruments in a two-stage least squares (TSLS) estimation procedure.

We test for the significance of the correlation between the production function residuals and the health inputs, using Wu's T_2 statistic (Wu 1973) as described by Nakamura and Nakamura (1981). If the null hypothesis of zero correlation between the error term and the regressors is not rejected, then OLS is an appropriate technique. For this reason, we perform OLS as well as the two-stage least squares techniques, on equations (1) and (12). A comparison of Wu statistics for equations (1) and (12) allows us to examine whether birth weight is a reasonable proxy for the health endowment. It should be noted that in both OLS and TSLS estimates of the production functions, we use a linear function form[15] 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 female schooling, female poverty levels, the percentage of high-risk women, neonatal intensive care availability, abortion availability, organized family planning availability, BCHS project availability, and the Medicaid program. These right-hand variables are similar to those used in Corman and Grossman's (1985) reduced form estimates. Predicted values of these four endogenous variables are then entered into the neonatal mortality equations.

Ideally, in our two-stage procedure, we would treat all right-hand variables in equations (1) and all variables except the percentage of high-risk women in equation (12) 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 but varying among firms for historical reasons.[16] 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 above. Our procedure is based on the reasonable assumption that prenatal and neonatal input availability measures have zero coefficients in the cigarette demand function.[17]

III. 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 3. 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 structural determinants yields what we have termed the quasi-structural production function. The remaining regressions show the direct effect of an input on

Table 3

Regression Results[a]

	Whites				Blacks			
	A-1	A-2	A-3	A-4	B-1	B-2	B-3	B-4
	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^2	.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.

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. Further evidence to this effect is that the difference between the TSLS and OLS coefficients is reduced appreciably, and even eliminated, in the structural as opposed to the quasi-structural regressions. 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.

A comparison of the OLS coefficients of neonatal intensive care use estimated with and without birth weight held constant [regressions (A1), (A3), (B1), and (B3)] is instructive because neonatal intensive care is the one input that conceptually has no causal impact on low birth weight. The comparison reveals that the white and black coefficients rise in absolute value by a factor of 2.3 and 1.2, respectively, when birth weight is held constant. These results are consistent with the interaction model developed in Section II (see footnote 13). The failure to control for low birth weight biases the OLS estimate of the neonatal intensive care parameter since light infants are more likely to be place in a neonatal intensive care unit and also are more likely to die.

Note, however, that the replacement of low birth weight with its endogenous determinants and the use of instrumental variables to correct for the correlation between neonatal intensive use and the disturbance term should yield an estimated coefficient that is equivalent to the one obtained by OLS in the specification including low birth weight. We tested whether the difference between the neonatal intensive care coefficients in these two specifications was significantly different from zero. For each race, the null hypothesis of no difference was easily accepted. The t-statistic was .48 for whites and .22 for blacks.[18]

Based on the Wu test, further discussion of the results will focus on regressions (A2), (B2), (A3), and (B3). All four equations are significant at the 1 percent level, as indicated by F-values. For whites, in the 2SLS

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 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 corresponding white one, although its significance level is only 10 percent.[19] 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 lowers 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 2).

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." As mentioned in Section I, births which are not averted may receive more of the unmeasurable inputs such as better nutrition and higher quality care that enhance the healthiness 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 poor 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 Philiber (1982) find that more than half of the first-time clients of large metropolitan family planning clinic previously 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.[20] 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

Table 4

Contribution of Selected Factors to Reductions in Neonatal Mortality Rates, 1964-1977[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.

of Table 4, 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.[21]

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 (.8 births per thousand live births) followed by prenatal care and WIC (.4 deaths per thousand live births each) and neonatal intensive care (.1 deaths per thousand live births). Prenatal care and WIC each has 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 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 (.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 4 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 4.

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.[22]

It is notable that practically all the black regression coefficients in Table 3 and all the estimated black effects in Table 4 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.

At the same time, the results do not imply that the construction and subsidization of additional neonatal intensive care units has a more favorable benefit-cost ratio than an expansion in BCHS project use for blacks, if, for example, these were competing programs. The direct effect of BCHS project use on black neonatal mortality (.2 deaths per thousand live births) is smaller than the direct effect of neonatal intesive care (.5 deaths per thousand live births). But the projects probably are cheaper to construct and maintain than sophisticated neonatal intensive care units. This suggests that BCHS projects may be attractive vehicles to lower birth weight-specific black mortality rates. Their benefit-cost ratio would be even more favorable if techniques could be developed to enable them to reduce the percentage of low-birth weight births.[23]

IV. 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 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 absense of cross-sectional or time-series indexes of the quality of care, the impact of neonatal intensive care undoubtedly is understated. Clearly the development of more comprehensive measures of this input deserves high priority on an agenda for future research.

Between 1964 and 1982 the white neonatal mortality rate fell by 4.9 percent per year (annually compounded), and the black neonatal mortality rate fell by 4.2 percent per year. Recent rates of decline have fallen short of these approximately two-decade historical trend values, especially for blacks. For instance, from 1981 to 1982, the black neonatal mortality rate fell by 2.2 percent, and the white rate fell 4.2 percent.[24]

Since the beginning of 1981, 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. 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).

Our results provide suggestive, although far from definitive, explanations of the slowdown in the downward trend in neonatal mortality. 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 (National Center for Health Statistics various years). 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 1981-1982 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.

FOOTNOTES

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[1]The 1964 figure was taken from the Bureau of the Census (1984). The 1982 figure was obtained from Arthur Horn of the Division of Vital Statistics of the National Center for Health Statistics. It replaces pre-

liminary estimates published in a variety of places.

[2]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.

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

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

[5]Other endogenous risk factors in the prematurity production function include parity and legitimacy status of the birth. These factors are not incorporated into the model because we do not estimate the prematurity production function, as explained below. Clearly, we do not ignore the risk

indicators just cited because they are caused to a large extent by abortion and contraceptive services at the county level.

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

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

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

$$d = d(n, m, a, c, b, x, e)$$

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

If s enters the equation for d, the rank condition is satisfied for the birth weight production function but is violated for the mortality production funtion. Empirical estimates of the above mortality function (not

shown) do not differ from the ones presented in Section III, which exclude x. Joyce (1985) estimates equations (1)-(3) and explores the role of prematurity in infant health outcomes using a subset of the counties employed in this paper.

[9]In general the use of abortion services is related to such risk characteristics as mother's age, parity, and marital status, which in turn are related to the endowment.

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

[11]Neonatal death rates associated with births to teenage mothers are substantially higher than births to women beyond the age of 20 (for example, Joyce 1985). Moreover, Forrest (1980) finds that the use of organized family planning services by teenagers has a sizable negative effect on teenage birth rates.

[12]Patient days in Level I units are excluded from the numerator because these units do not provide specialized state-of-the-art services. No

distinction is drawn between Level II and Level III patient days due to definitional problems in the available data.

[13]Let d_{1j} be the neonatal mortality rate of light babies in the j^{th} county, and let d_{2j} be the mortality rate of normal-weight babies. As an identity,

$$d_j = k_j d_{1j} + (1-k_j)d_{2j} ,$$

where d_j is the observed neonatal mortality rate and k_j is the fraction of light births. Ignoring other inputs, specify production functions for d_{1j} and d_{2j} as follows:

$$d_{1j} = \alpha_0 + \alpha_1 n_j + u_{1j}$$
$$d_{2j} = \beta_0 + u_{2j} .$$

In these equations n_j is neonatal intensive care use per low-birth weight birth and u_{1j} and u_{2j} are disturbance terms. Because birth weight-specific death rates are not available at the county level, substitute the last two equations into the first one:

$$d_j = \beta_0 + (\alpha_0-\beta_0)k_j + \alpha_1 k_j n_j + v_j ,$$

where $v_j = k_j u_{1j} + (1-k_j)u_{2j}$. Hence,

$$(\partial d_j/\partial n_j) = \alpha_1 k_j$$
$$(\partial^2 d_j/\partial n_j \partial k_j) = \alpha_1 .$$

For a similar argument with respect to poverty programs, see Grossman and Jacobowitz (1981).

[14]The interacted family planning variable is obtained by dividing the race-specific number of teenage family planning users by the race-specific number of women aged 15 to 19. The raw family planning variable is obtained by assumming the race-specific fraction of women aged 15 to 19

with family income less than 200 percent of the poverty level equals the race-specific fraction of women aged 15 to 44 with family income less than 200 percent of the poverty level. The raw family planning variable is shown in Table 2 for illustrative purposes; it is not used to create the interacted variable.

[15]We choose a linear rather than a logistic functional form because linear coefficients are more easily interpreted. Maddala (1983, p. 30) argues that a linear form is appropriate for large, aggregate probability samples such as ours.

[16]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 III.

[17]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.

[18]To calculate the standard error of the difference between the two coefficients, we made use of Hausman's (1978) result that the variance of the difference between two estimates of the same coefficient is equal to the sum of the variances. This holds if both estimates are consistent and one of them is efficient under the null hypothesis of no difference.

[19]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.

[20]The sources for the initial year and terminal year values of the inputs in the extrapolations and the assumptions that underlie these values are available in the Appendix to this paper, which is available on request.

[21]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.

[22]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 in the same manner as legal abortions, we overstate the abortion effect in Table 4. It is plausible, however, that differences in illegal abortion rates among states and counties in 1964 had much weaker impacts on neonatal mortality than differences in legal abortion rates in 1977. This is because it probably was more difficult for low-income women, who undoubtedly have poorer endowed birth outcomes than other women, to obtain illegal abortions.

[23]Similar comments apply to the direct effect of organized family

planning, although here some caution is required because the main purpose of organized family planning clinics is to deliver contraceptive services.

[24]Neonatal mortality rates for years after 1982 are not used here because they are preliminary.

REFERENCES

Bureau of the Census. Statistical Abstract of the United States. Washington, D.C.: U.S. Government Printing Office, 1984.

Chamie, Mary, et al. "Factors Affecting Adolescents' Use of Family Planning Clinics." Family Planning Perspectives, 14, No. 3 (May/June 1982).

Corman, Hope, and Grossman, Michael. "Determinants of Neonatal Mortality Rates in the U.S.: A Reduced Form Model." Journal of Health Economics, 4, No. 3 (December 1985).

Forrest, Jacqueline D. "Exploration of the Effects of Organized Family Planning Programs in the United States on Adolescent Fertility." Final Report, Contract No. NO1-HD-82844 from the National Center of Child Health and Human Development to the Alan Guttmacher Institute, October 1980.

Goldman, Fred, and Grossman, Michael. "The Impact of Public Health Policy: The Case of Community Health Centers." National Bureau of Economic Research Working Paper No. 1020, November 1982.

Grossman, Michael, and Jacobowitz, Steven. "Variations in Infant Mortality Rates Among Counties of the United States: The Roles of Public Policies and Programs." Demography, 18, No. 4 (November 1981).

Hadley, Jack. More Medical Care, Better Health? Washington, D.C.: Urban Institute, 1982.

Harris, Jeffrey E. "Prenatal Medical Care and Infant Mortality." In Economic Aspects of Health, edited by Victor R. Fuchs. Chicago: University of Chicago Press for the National Bureau of Economic Research, 1982.

Hausman, J. "Specification Tests in Econometrics." Econometrica, 46, No. 6 (November 1978).

Institute of Medicine. Preventing Low Birth Weight. Washington, D.C.: National Academy Press, 1985.

Jones, Judith B.; Namerow, Pearila B.; and Philliber, Susan. "Adolescents' Use of a Hospital-Based Contraceptive Program." Family Planning Perspectives, 14, No. 4 (July/August 1982).

Joyce, Theodore J. "A Structural Model of Birth Outcomes in the U.S." Ph.D. dissertation, City University of New York Graduate School, 1985.

Kotelchuck, Milton, et al. "WIC Participation and Pregnancy Outcomes: Massachusetts Statewide Evaluation Project." American Journal of Public Health , 74, No. 10 (October 1984).

Lewit, Eugene M. and Coate, Douglas. "The Potential for Using Excise Taxes to Reduce Smoking." Journal of Health Economics, 1, No. 2 (August 1982).

Lewit, Eugene M. "The Demand for Prenatal Care and the Production of Healthy Infants." In Volume III of the Annual Series of Research in Human Capital and Development, edited by Ismail Sirageldin, David Salkever, and Alan Sorkin. Greenwich, Connecticut: JAI Press, Inc., 1983.

Lewit, Eugene M. "The Interstate Market for Smuggled Cigarettes." Presented at the annual meetings of the American Economic Association, New York, New York, December 1982.

Maddala, G.S. Limited-Dependent and Qualitative Variables in Econometrics. Cambridge, England: Cambridge University Press, 1983.

McCormick, Marie C. "The Contribution of Low Birth Weight to Infant Mortality and Childhood Morbidity." The New England Journal of Medicine, 312, No. 2 (January 10, 1985).

Miller, C. Arden. "Infant Mortality in the U.S." Scientific American, 253, No. 1 (July 1985).

Nakamura, A. and Nakamura, M. "On the Relationship Among Several Specification Error Tests Presented by Durbin, Wu and Hausman." Econometrica, Vol. 49, No. 6 (November 1981).

National Center for Health Statistics. Advance Report of Final Natality Statistics. U.S. Department of Health and Human Services, Public Health Service, various years.

Paneth, Nigel, et al. "Newborn Intensive Care and Neonatal Mortality in Low-Birth Weight Infants." New England Journal of Medicine, 307 (1982).

Rosenzweig, Mark R., and Schultz, T. Paul. "Education and Household Production of Child Health." Proceedings of the American Statistical Association, Social Statistics Section. Washington, D.C.: American Statistical Association, 1981.

Rosenzweig, Mark R., and Schultz, T. Paul. "The Behavior of Mothers as Inputs to Child Health: The Determinants of Birth Weight, Gestation, and Rate of Fetal Growth." In Economic Aspects of Health, edited by Victor R. Fuchs. Chicago: University of Chicago Press for the National Bureau of Economic Research, 1982.

Rosenzweig, Mark R., and Schultz, T. Paul. "Consumer Demand and Household Production: The Relationship Between Fertility and Child Mortality." American Economic Review, 73, No. 2 (May 1983a).

Rosenzweig, Mark R., and Schultz, T. Paul. "Estimating a Household Production Function: Heterogeneity, the Demand for Health Inputs, and their Effects on Birth Weight." Journal of Political Economy, 91 No. 5 (October 1983b).

Taffel, Selma. Factors Associated with Low Birth Weight, United States, 1976. U.S. Department of Health, Education, and Welfare, Public Health Service, National Center for Health Statistics, Vital and Health Statistics, Series 21, No. 37 (April 1980).

Taffel, Selma. Prenatal Care in the United States, 1969-1975. U.S. Department of Health, Education, and Welfare, Public Health Service, National Center for Health Statistics, Vital and Health Statistics, Series 21, No. 33 (September 1978).

U.S. Department of Health and Human Services. The Health Consequences of Smoking for Women: A Report of the Surgeon General. Washington, D.C.: Office of the Assistant Secretary for Health, Office of Smoking and Health, 1980.

Williams, Ronald L. "Measuring the Effectiveness of Perinatal Medical Care." Medical Care, 17 (1979).

Williams, Ronald L. "Outcome-Based Measurements of Medical Care Output: The Case of Maternal and Infant Health." Ph.D. dissertation, University of California at Santa Barbara, 1974.

Wu, D. "Alternative Tests of Independence Between Stochastic Regressors and Disturbances." Econometrica, Vol. 41, No. 4 (July 1973).

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