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Chapter Author: Mark R. Rosenzweig, T. Paul Schultz

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2 The Behavior of Mothers as Inputs to Child Health: The Determinants of Birth Weight, Gestation, and Rate of Fetal Growth

Mark R. Rosenzweig and T. Paul Schultz

The characteristics infants show at birth appear to play important roles in their subsequent growth, morbidity, and survival.[1] Such characteristics—birth weight, length of the gestation period, and rate of fetal growth—are affected by parental behavior, which usually is modified to favorably influence birth characteristics and which may also unknowingly condition the health of the newborn.[2] In recognition of the importance of these birth characteristics, many studies of health production focus on the relationship between the behavior of the pregnant mother and the subsequent characteristics of her newborn. Other studies examine the relationships between parental socioeconomic characteristics and/or access to health services and infant mortality (one indicator of infant health), as well as the relationship between parental socioeconomic characteristics and the mother's utilization of those prenatal medical services that are presumed to affect child health. Most studies suffer, however, from one or a combination of problems—the use of a choice-based sample, such as mothers visiting a subsidized clinic; the lack of control for other health-related behavior or inputs beyond the one studied; the use of implausible econometric specifications of health production relations; or the inattention to the possible importance of population heterogeneity in unobserved characteristics which may affect child health and condition parental health production behavior.

In this paper, we attempt to deal with many of these problems by specifying and estimating a simple model of the parental production of

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child health. The model illustrates the need for examining jointly (1) the determinants of the demand for health production inputs, or parental behavior, including both scioeconomic and health program variables, and (2) the parameters of the technical-biological health production function—the relationship between behavioral inputs and the newborn's health characteristics, the output. Estimates of the production function and the input demand equations are needed to understand and interpret the reduced-form demand equations for birth characteristics, while knowledge of the factors conditioning parental behavior is generally required to obtain consistent estimates of the health production function. These estimates also provide information on the importance of socioeconomic factors compared to the availability of medical services in determining the initial conditions of an infant's life, as well as which type of parental behavior has serious consequences for child health and development.

In the first section of the paper, a model is formulated which embeds a health production function in a utility maximizing framework, distinguishing among goods which have no effect on child health but are desired for their own sake, goods which affect child health but are not desired for the direct utility they provide, and goods or behavior which both augment parental satisfaction directly and affect birth outcomes that indirectly affect parent utility. Implications are derived from the model regarding the demand for these three types of goods and the estimation of the health production function when families differ in either their genetic health endowments or their "caring" for child health. The model also indicates that even when a particular behavior decreases child health or well-being, a tax on such an activity may decrease child health even while resulting in a reduction of the activity.

In the second part we apply the framework to a national probability sample of approximately 10,000 legitimate live births from the National Natality Followback Surveys in the years 1967–1969. Based on the sample socioeconomic information and merged geographic information on such per capita variables as medical doctors, health expenditures, hospital beds, and family planning services, we present estimates of the relationships between birth weight, gestation, and the rate of fetal growth and four inputs: the mother's cigarette consumption while pregnant, her use of prenatal medical services, her age at the infant's birth, and her number of births. Our estimates are based on general functional forms for the health production function and on linear approximations to the input demand equations. Estimates are reported of the effects of father's income, mother's education, and regional medical service and price variables on the three measures of birth outcomes and on the derived demands for the four health inputs. The sensitivity of these estimates to changes in functional form and estimation is discussed. The final section

compares our findings with those of other studies and considers alternative interpretations of the differences in birthweight among black and white women.

The Model

Child Health Production and the Demand for Child Health and Inputs

Assume that a family derives satisfaction from three types of goods—the health of each of its children, H; consumer goods, Y, which affect H (health-related goods, such as smoking or number of children); and consumer goods, X, which are health-neutral (have no effect on H, such as books). The health of children is affected by the level of Y goods, as well as other purchased or family inputs, Z, which are bought or allocated only because they contribute to child health (medical services, for example). Thus, the utility function of the family is

$$U = U(X, Y, H). \tag{1}$$

The relationship between child health and the levels of Y and Z is described by a production function,

$$H = F(Y, Z, \mu),\ F_y,\ F_z,\ F_\mu \neq 0, \tag{2}$$

where μ is "endowment" health, that component of child health due either to genetic or environmental conditions uninfluenced by parental behavior, but known to them.[3] Distinctions between the perceived production function and the true production function are discussed later, as is the role of schooling.

The family maximizes (1), given (2), which is assumed to be known, and subject to the budget constraint, given by (3)

$$I = XP_x + YP_y + ZP_z \tag{3}$$

where P_x, P_y, P_z are the prices of the health-neutral and health-related consumption goods and child health investment goods, respectively, and I is income.

The important features of this model are that (*a*) health cannot be purchased directly; rather, other goods must be bought or utilized to influence health in a way described by (2), and (*b*) the family does not maximize child health, but looks at child health as one utility-augmenting "good" for which it must sacrifice other goods. Since the X or Y goods can include the number of children, the model also accommodates family choices regarding family size and child health and any trade-offs between them, as in the Becker-Lewis-Tomes interactive model.[4]

The first-order maximization conditions are

$$U_x = \lambda P_x \quad , \tag{4}$$

$$U_y + U_H F_y = \lambda P_y \quad , \tag{5}$$

$$U_H F_z = \lambda P_z \quad , \tag{6}$$

where λ is the Lagrangian multiplier.

While condition (4), applying to the health-neutral good, is conventional, expression (5) indicates the dual role of the health-related consumption good Y in augmenting utility directly and indirectly by its effect on H, through (2). The health investment good Z is demanded, as shown in (6), only because child health contributes to utility. Note, however, that even if Y had no effect, or an adverse effect, on H ($F_y < 0$), Y might still be consumed. The marginal product of Y in health production is an implicit tax ($F_y < 0$) on or subsidy ($F_y > 0$) of the Y good.

The model yields three demand equations for the three goods in terms of prices and income:

$$X = D_x(P_x, P_y, P_z, I, \mu) \quad , \tag{7}$$

$$Y = D_y(P_x, P_y, P_z, I, \mu) \quad , \tag{8}$$

$$Z = D_z(P_x, P_y, P_z, I, \mu) \quad . \tag{9}$$

The effects of changes in the prices of the three types of goods on the level of child health can be derived from these equations, noting that

$$dH = F_y dY + F_z dZ + F_\mu d\mu. \tag{10}$$

From (2), these effects can be written as:

$$\frac{dH}{dP_x} = F_y \frac{dY}{dP_x} + F_z \frac{dZ}{dP_x} \quad , \tag{11}$$

$$\frac{dH}{dP_y} = F_y \frac{dY}{dP_y} + F_z \frac{dZ}{dP_y} \quad , \tag{12}$$

$$\frac{dH}{dP_z} = F_y \frac{dY}{dP_z} + F_z \frac{dZ}{dP_z} \quad , \tag{13}$$

since $d\mu / dp_i = 0, i = x, y, z$. Expressions (11), (12), and (13) indicate that price effects on child health depend on the effects of changes in prices on the demand for health production inputs as well as on the marginal products of these inputs in the production of health. The equations also suggest that changes in the prices of health-neutral goods will also affect the level of child health. It is essential, however, to appreciate what the expressions cannot predict without additional restrictions. For example, assume that it is known that the higher the consumption of the Y good the lower is child health ($F_y < 0$) and that $F_z > 0$. While the model predicts that a rise in P_y will reduce the consumption of Y, ignoring income effects

($dY/dP_y < 0$), the sign of (12) cannot be predicted since dZ/dP_y is not signed. For example, assume that smoking by the mother while pregnant is known to adversely affect the newborn child (we test for this later). A rise in the price of cigarettes because of taxation, while decreasing cigarette consumption, might also lower H if smoking and H were complements in the utility function, or if smoking and labor force participation were complements and the latter augmented health.

The model thus indicates that we must know the parameters of the health production function as well as the price effects of goods in order to predict how changes in prices will affect child health. We cannot know a priori whether a tax on or subsidy of a health-related or health-investment good will actually improve child health, even if it does lead to a predicted change in the consumption of the taxed good and even if we have information on the technical or biological relationships between child health and the consumption of the good or health input. The estimation of such technical relations, i.e., the characteristics of the production function (2) which enable the measurement of F_y and F_z, is considered next.

Population Heterogeneity and the Estimation of the Health Production Functions

Information on the technological or biological relationships between behavioral variables and child health outcomes, i.e., knowledge of (2), is useful for predicting and assessing the effects of health-related policies, but such information is also useful for helping potential parents efficiently attain their desired child health goals. Unfortunately, the opportunity to perform controlled experiments to ascertain the partial, causative effects of any one behavioral variable on birth outcomes, while controlling for all other factors, is minimal. We now show that the observed population associations between behavioral and child health variables, even when all commonly observed factors are held constant, are unlikely to provide the correct estimates of the $F_i, i = y, z$ as long as there are observed factors known to the parents but not to the researcher (μ), and even if such family-specific factors are randomly distributed in the population and unaffected by behavior. Knowledge of the determinants of the health production inputs, however, can enable us to obtain consistent estimates of the relevant parameters of the health production function.

To simplify the discussion, assume that function (2) has only one factor in addition to the unobserved health endowment or environmental variable, i.e., $F_y = 0$, and that Y and X are treated as a single variable, X. Then, controlling for all prices and income, the relationship between H and the health factor Z in the heterogeneous (in μ) population is

$$\frac{dH}{dZ} = F_z + F_\mu \frac{d\mu}{dZ} \quad . \tag{14}$$

The observed population association between child health and the behavioral variable Z thus does not in this case correspond to the technical relationship or marginal product F_z, but is contaminated by the unobserved, random μ factor as long as Z and μ are not uncorrelated. To see that $d\mu/dZ$ or $dZ/d\mu$ is not likely to be equal to zero, assume for simplicity that $F_{z\mu} = 0$. Then it can be demonstrated that

$$\frac{dZ}{d\mu} = F_\mu \left[U_{HH} F_z \frac{dZ}{dP_z} + U_{XH} \frac{dZ}{dP_x} \right] \tag{15}$$

so that, from (14),

$$\frac{dH}{dZ} = F_z + \left[U_{HH} F_z \frac{dZ}{dP_z} + U_{XH} \frac{dZ}{dP_x} \right]^{-1} \tag{16}$$

Expression (16) indicates that in the simple model the population association between H and the input Z, given by an ordinary least squares regression coefficient, for example, is an upwardly biased estimate of the true, technical parameter F_z, because second-order conditions imply that, controlling for income, $dZ/dP_z < 0$ and $dZ/dP_x > 0$ while $U_{HH} < 0$ and $U_{XH} > 0$. In other words, the model suggests that parents who expect to have relatively healthy children, based perhaps on observations on past births or from the birth outcomes of close kin, and/or who reside in relatively healthy environment, will be observed to use less of the variable input Z but to have healthier children than parents who are less well-educated or reside in less healthy family environments. The positive association between Z and H is in part spurious, the result of choices by the parents conditioned by factors, in this case μ, unknown to the researcher. In the more general case in which there is more than one factor in (2), the bias cannot be signed a priori.

While μ affects parental behavior and thus influences the level of child health and input use, it is not presumably correlated with those factors, affecting behavior, the P's. It is thus possible to estimate without bias the effects on H of the inputs, i.e., to purge the variation in μ from the variation in the Z and Y. In the simple model here, it is possible to obtain an unbiased estimate of dZ/dP_z in the presence of μ, since $dP_z/d\mu = 0$. The association between that part of the variation in Z due only to the variation in P_z and the variation in H provides an unbiased estimate of F_z. In econometric terms, to estimate the parameters characterizing the child health production function (2) requires a two-stage procedure in which the first-stage equations, providing unbiased estimates of the dZ/dP_z, correspond to the demand equations for the behavioral variables (7), (8),

and (9). The predicted values of these variables based on the first-stage estimates, orthogonal to the μ, are used to estimate the production function parameters. The demand equations (7), (8), (9) for the Z and Y in terms of the P_z, P_y and P_x and I are the reduced-form input demand equations; the health production function (2) is the "structural" equation.

Education, Information, and the Production of Child Health

In the literature utilizing the household production framework, educational attainment is usually treated as an "environmental" variable which affects the marginal products of production inputs.[5] It is assumed that more educated parents or consumers are more efficient producers of commodities providing utility, where efficiency is defined to mean more output for given inputs. Hence, rewriting (2) with e defined as the level of educational attainment:

$$H = F'(Y, Z, \mu; e) \tag{2a}$$
$$F'_{ye}, F'_{ze} > 0$$

Given the first-order conditions (4), (5), and (6), it is easy to see that the demand for all health inputs, as well as the pure utility good X, will be functions of schooling attainment in addition to prices and income. It is not clear, however, how education can actually alter marginal products of inputs or biological processes embedded in (2) unless inputs are omitted from (2). That is, it is doubtful that schooling can affect the production of H without it being associated with some alteration in an input. Instead, education, by augmenting information, may be thought to affect parental *perceptions* of the relationships between inputs and outputs. Parents maximize utility subject to production relations which they think exist; equation (2a) can be thought of, therefore, as the perceived production function. If parents differ in their understanding of the true technical or biological relationships between Y, Z, and H in ways related to educational attainment, as given in (2a), then input demand in any population will be a function of schooling. Education would not, however, appear empirically to affect actual marginal products of the production inputs as long as all of the inputs which varied across families were suitably taken into account.

Indeed, if households vary in their perceptions of the true parameters of health production relations, then it is possible to estimate the "true" production function (2) even if prices or income do not vary across the population, as long as a variable can be found which is related to such perceptions but which itself plays no direct role in production—such as schooling attainment. To obtain predictions from the model when perceptions concerning (2) differ, one must impose some structure on

either the relationships between perceptions and observable characteristics or on the distribution of perceptions of health technology.

Empirical Application

The Data and Econometric Framework

The preceding analysis suggests that to understand and predict the effects of changes in medical or health programs which alter the costs of behavior that influences child health, it is necessary to estimate both the technical or biological relationships between behavior and child health (the health production function) and the determinants of the behavioral variables (the input demand equations). Moreover, knowledge of the latter is often useful for obtaining consistent estimates of the former. To apply the model one needs information on birth outcomes that reflects infant well-being, knowledge of parental behavior, or inputs, related to child health production, and the price and/or availability variables which affect such behavior. The 1967, 1968, and 1969 National Natality Follow-back Surveys appear to meet most of these requirements. These national probability samples of approximately 10,000 legitimate births for the three years combined contain information on birth weight and gestation period for each birth, as well as subsequent child mortality, the educational attainment of both parents, the earnings of the husband, three aspects of the mother's behavior during pregnancy which are potentially linked to infant health at birth—smoking, working, and receiving prenatal medical care—in addition to data on age at birth and parity. The survey also provides information on the county of residence of the mother at the time of the birth, enabling us to merge local price and health program variables with the microdata.

We selected for analysis all nonmultiple births, resulting in a sample of 9,621 births. Based on the geographical information we collected and merged county or state level data on hospital beds per capita (BEDS), per capita governmental health expenditures (HEXP), the per capita number of hospitals (HOSPFP) and health departments (HDFP) offering family planning services, medical doctors per capita (MD), the proportion of women aged 15–59 in the labor force who are unemployed (UNEMPR-W), the percentage of persons in service industries which employ a disproportionately large share of women (SERVICE), the cost (including excise taxes but excluding retail sales taxes) of cigarettes (CPRCE), the retail sales tax (TAXSALES) on cigarettes, and the size of the SMSA (SIZE) for inhabitants of SMSAs. The sample characteristics and definitions of all variables are listed in Table 2.1.

The weight of a child at birth has much to do with its prospects for survival (e.g., Susser et al. 1972). During 1964–65 in the United States,

18.6% of infants weighing less than 2,500 grams did not reach their first birthday; among those weighing more than 2,500 grams the proportion dying in the first year was .97%, for a ratio of 19 to 1. Grouping births by parents' economic characteristics yields much narrower differentials: the ratio of infant mortality rates for mothers with eight years education or less compared to those with sixteen years or more is 1.86 to 1, and for families with annual incomes of under $3,000 compared to those with incomes of $10,000 and over the ratio is 1.67 to 1 (MacMahon et al. 1972, Tables 18, 21, and 22).

If low birth weight is a genetically determined factor predisposing to early death, economic analysis of this indicator of child health in a production function framework would not be useful. But the frequency of prematurity, measured by a birth weight of less than 2,500 grams, varies substantially across social and economic groups in the society. It is almost twice as great among mothers with less than nine years of education as among those with sixteen years or more—10.6% versus 5.6%, respectively (Ibid., Table 19). Moreover, the proportion of nonwhite U.S. births thus classified as premature is much higher than for whites, and increasing from about 10% to more than 13% from 1950 to 1967.[6] The overall proportion of underweight births in the United States remains in excess of that recorded in other industrially advanced countries and is frequently linked to the relatively high level and distribution of infant mortality in the United States (Wiener and Milton 1970; Chase and Byrnes 1972; Hemminki and Starfield 1978; Taffel 1980).

For simplicity, birth weight is treated in this study as a linear indicator of good child health. Though deaths are highly concentrated among very low birth weight infants, the inverse relationship between infant mortality rates and birth weight is approximately linear from under 1,000 grams to about 3,000 grams. Slightly elevated mortality levels are also recorded for infants weighing more than 4,500 grams, but these "overweight" births constituted less than 2% of U.S. live births in 1960 (Chase 1969). The analysis of birth weight as a continuous linear indicator of child health has obvious statistical advantages over a dichotomous and relatively infrequent event such as infant mortality.[7]

A second indicator of the newborn's health is its gestational age. Infants of short gestation die much more frequently during the first month of life: in early 1950, 79% of the U.S. births whose periods of gestation were under 28 weeks died, whereas only .88% of those whose gestation was 37 weeks or more died (Shapiro 1965, Table H). However, gestation is not reported on birth certificates in a few states, and some epidemiologists suspect that reported information on gestation is subject to greater error than that on birth weight (Eisner et al. 1979).

Recently, two health effects of prematurity have been distinguished: a relatively transitory trauma associated with leaving the womb and estab-

Table 2.1 **Variable Definition, Means, and Standard Deviations**

Variable	Definition	Mean	Standard Deviation
Endogenous			
Birth weight	Weight of baby at birth, in grams	3288	568
Gestation Period	Length of pregnancy, in weeks	39.1	2.45
Standardized birth weight	Birth weight/Predicted birth weight based on gestation period (see text)	1.00	.170
DELAY	Number of months of elapsed pregnancy before mother saw a doctor	2.74	1.55
SMOKING	Number of cigarettes mother smoked per day while pregnant	4.71	8.64
AGE	Age of mother at birth in years	24.9	5.61
BIRTHS	Number of live births born to mother including current one	2.54	1.90
Exogenous–Individual			
MGRM	= 1 if mother did not enter high school	.095	.301
MHSI	= 1 if mother attended high school for less than 4 years	.230	.421
MHSC	= 1 if mother completed high school	.445	.497
MCOLI	= 1 if mother attended college for less than 4 years	.142	.350
MCOLC	= 1 if mother completed college	.087	.282

HINC	Annual income of husband	6132	3785
SMSA	= 1 if family is located in an SMSA	.700	.458
BLACK	= 1 if mother is black	.190	.392
1967	= 1 if birth occurred in 1967	.332	.470
1968	= 1 if birth occurred in 1968	.330	.470
Exogenous–Area			
BEDS	Number of hospital beds per capita	.00466	.00109
HEXP	Governmental health and hospital expenditures in thousands of dollars per capita	.0203	.0226
HOSPFP($x10^{-8}$)	Number of hospitals with family planning program per capita	299.	158.
HDFP($x10^{-8}$)	Number of health departments with family planning per capita	95.0	199
MD($x10^{-3}$)	Number of persons per medical doctor	1.42	.695
UNEMPR-W	Unemployed proportion of women in labor force aged 15–59	.0526	.0104
SERVICE ($x10$)	Percent of persons employed in service industries	77.9	15.3
CPRCE	Price of cigarettes including state and local excise taxes (cents/package)	34.61	3.38
TAXSALES	Retail sales tax on cigarettes (cents/package)	.583	.490
SIZE($x10^{-3}$)	Population of SMSA	1349.6	2087
n	Number of Observations	9621	

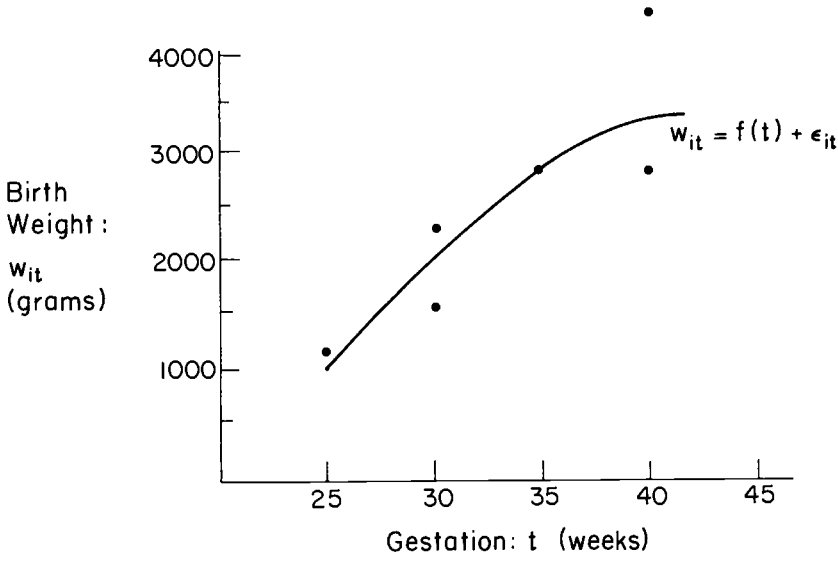

Fig. 2.1 Hypothetical pattern of birthweight by gestation for live births.

lishing viable body functions, primarily respiratory, and more permanent debilities that are more frequently associated with congenital defects and excessive risks of morbidity and mortality continuing beyond the second year of life (Beck and van den Berg 1975). The former transitory health effect is approximated here by short gestation. The latter, more permanent effect is represented by the fetal rate of weight gain to birth, normalized by the average weight gain associated with infants of that gestation.

It is assumed that a biological-technical relationship exists between birth weight and gestation. This is illustrated in Figure 2.1, where the birth weight of individual i at gestation t, w_{it} is represented by a nonlinear function of gestation, $f(t)$, and an individual disturbance, ε_{it}, that could embody random and genetic differences across individuals, variation in behavioral inputs of parents, and errors of measurement. To compare individuals of different gestations, the systematic effect of gestation on birth weight, $f(t)$, must be removed; a measure of individual deviation from the normal fetal growth curve is thereby obtained. The nonlinear fetal growth function is first estimated from our sample as a cubic function of gestation in weeks. The individual's birth weight is then divided by the expected birth weight for the individual's gestation, predicted by the estimated fetal growth function.[8] This measure of normalized birth weight, illustrated in Figure 2.2, represents the more permanent child

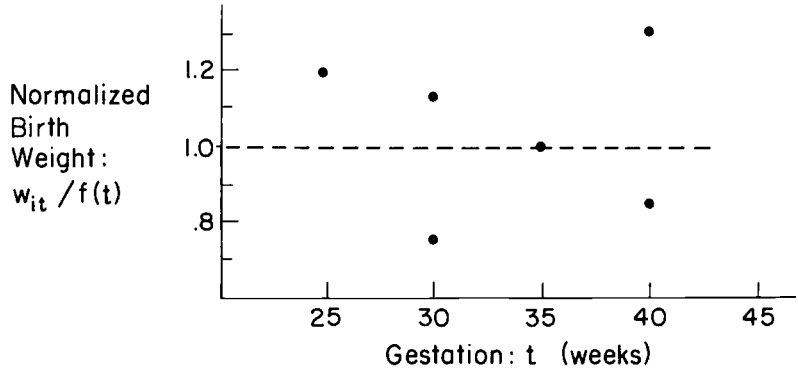

Fig. 2.2 Hypothetical pattern of birthweight normalized for gestation.

health effects of prematurity. The variance of the error associated with this normalized measure of birth weight should also be approximately constant, improving the efficiency of our estimates of a child health production function.[9]

Differences in the distribution of birth weight between distinct ethnic or racial groups might arise for at least two reasons. On the one hand, the demand for health inputs might differ between groups because their income, education, and local prices differ. On the other hand, the biological-technical relationships, such as $f(t)$, may differ between genetic groups, posing a problem of comparability of birth characteristics used as indicators of development or health across such groups.

In this study, and many others (e.g., Chase 1962; Baumgartner 1962), birth weights for blacks and whites differ. Estimates of the production function for birth characteristics and input demands provide a methodology for appraising whether demand for health inputs differs between blacks and whites, and whether birth characteristics differ for blacks and whites given their input demands (Cf. Wiener and Milton 1970). A third approach is to estimate distinct fetal growth functions for black and white births separately, and define the normalized birth weight specific to these race groups.[10] Given the relatively small number of black births in our sample, the entire production function is not estimated for blacks alone, although estimates for only white births are reported in the Appendix for the purposes of comparison.

Thus, three measures of birth outcomes are used in the analysis: birth weight, gestation, and birth weight normalized for gestation, which represents the rate of fetal growth. Birth weight is first normalized for the fetal growth function fitted to the entire sample, and then normalized according to black and white specific growth functions.

The endogenous or behavioral variables considered to be potential determinants of the birth outcomes are the number of months the mother

worked while pregnant, the number of months of elapsed pregnancy before the mother visited a medical doctor (DELAY), the number of cigarettes smoked per day by the mother while pregnant (SMOKING), the order of the current live birth (BIRTHS), and the age of the mother at birth (AGE). The mother's age in this context is a choice variable, as it refers to the point in her life cycle at which she is choosing to have a child. In all specifications and tests of the health production function, working by the mother while pregnant never appeared to be a signicant determinant of birth outcomes, and in what follows we consequently do not include this variable. Variables reflecting a part of the health environment as well as possible biological differences are SMSA residence, SMSA size, dummy variables for the year of the child's birth (1967, 1968, or 1969), and whether or not the mother is black (BLACK). The birth characteristics production function in its general form is thus:

$$H = F(\text{AGE, DELAY, SMOKING, BIRTHS, SMSA, SIZE, 1967, 1968, BLACK; } \mu). \tag{17}$$

AGE, SMOKING, and BIRTHS are all health-related goods (Y), providing direct utility to the mother in addition to their impact on child health, whereas DELAY is perceived as only affecting child health (Z).

To estimate the demand for goods potentially affecting the birth characteristics—the endogenous inputs in (17), corresponding to equations (7), (8), and (9)—we utilize both the socioeconomic information from the survey data and the areal program and price variables. Included among the former are school attainment variables of the wife and the annual earnings of the husband as well as the race variable. The area variables correspond to or are determinants of the prices in the model; CPRCE and TAXSALES are components of the price of SMOKING, and should be negatively associated with that activity; UNEMPR-W represents the lack of demand for female work and should be negatively correlated with the value of the mother's time, while we expect SERVICE, a female-intensive industry, to be positively associated with the value of time, one component of the price of both visiting a doctor and the fertility variables. HDFP and HOSPFP, the family planning variables, should be negatively correlated with BIRTHS, as they should be inversely associated with the cost of averting births, and may affect AGE as well; HEXP, BEDS, MD, and HDFP and HOSPFP should all be positively associated with lower costs of medical care, inducing less delay by mothers in seeking prenatal medical care and thus negatively correlated with DELAY. The demand equations will not only enable us to obtain consistent estimates of the effects of the health-related activities on initial infant well-being (17), but allow an assessment of the relative influence of individual characteristics and the local availability of medical services on the mother's input activities which affect the conditions of the child at birth.

Estimating the Infant Health Technology and the Demand for Infant Health Inputs using Approximations.

Because of the difficulty of obtaining exact solutions for the set of input demand equations from a complete parameterization of the household production model (1), (2), and (3) which does not impose implausible behavioral restrictions, we pursue an alternative estimation strategy involving the use of approximations to the demand equations corresponding to (7), (8), and (9) and to the health production function (2). This strategy was also motivated by the rejection by the data of the set of restrictions implied by one completely parameterized demand system involving Cobb-Douglas forms, as reported in Rosenzweig and Schultz (1980). For the approximate demand equations, we do not impose separability (as in the Cobb-Douglas demand system), but allow for the effects of changes in the price or availability of each input to affect other inputs, as implied by the general model. All prices or program variables thus appear on the right-hand side of each child health input demand equation. The equations we estimate, in linear form, are thus:[11]

(18) through (21) AGE, DELAY, SMOKING, BIRTHS = D(MHSI, MHSC, MCOLI, MCOLC, HINC, BEDS HEXP, HOSPFP, HDFP, MD, UNEMPR-W, SERVICE, CPRCE, CPRCE2 TAXSALES, SMSA, SIZE, BLACK, 1967, 1968),

where CPRCE2 is the square of CPRCE.

The functional form used to estimate the infant health production function is the generalized Leontief-Diewert (Diewert 1971). Three specifications are estimated. The first assumes that the relationships between the health inputs and the birth outcome measures are described by a simple linear or Leontief fixed-coefficient model. The second assumes a more general form for these relationships, allowing for substitutions between inputs, but imposes local linear homogeneity. The third, most general parameterization, does not impose linear homogeneity. All specifications assume that birth outcomes are affected linearly by the biological-environmental variables represented by SMSA, SIZE, 1967, 1968, and BLACK. The health production functions we estimate is thus given by:

$$H = \sum_i \sum_j \beta_{ij} y_i^{\frac{1}{2}} y_j^{\frac{1}{2}} + \Sigma_i \beta_i y_i^{\frac{1}{2}} + \gamma_1 \text{SMSA} + \gamma_2 \text{SIZE} \tag{22}$$

$$+ \gamma_3 \text{BLACK} + \gamma_4 1967 + \gamma_5 1968 + \gamma_o + \mu$$

where the y_i are AGE, DELAY, SMOKING, BIRTHS, $\beta_{ij} = \beta_{ji}, \beta_{ij} = 0$, $i \neq j$ for the linear model, $\beta_{ij} \neq 0$, $i \neq j, \beta_i = 0$, for the more general linear

model, and $\beta_i \neq 0$ for the general case in which local linear homogeneity is not imposed.

This flexible functional form, which can be considered a second-order approximation to any production function, can be used to test the three models against each other and to compute elasticities of substitution between the inputs, measures of the degree to which each input can substitute for another in the production of infant well-being.[12] Such elasticities are assumed to equal one in the Cobb-Douglas case.

Because, as we have shown, the error term μ is likely to be correlated with the y_i, ordinary least squares (OLS) estimates of the β's (22) may be inconsistent. Two-stage least squares estimates are required, utilizing exogenous determinants of the input demand equations for the four behavioral variables y_i in the first stage. OLS estimates of (22) are also reported in order to evaluate the importance of heterogeneity.[13] The first-stage equations contain, in addition to the variables specified, interactions of the education and race dummies and husband's income with all of the price and program availability variables.

Child Health Input Demand Equation Estimates: Linear Specifications

The four linear health production input demand equations are reported in Table 2.2 In all equations, both the sets of socioeconomic variables and the regional health input availability and price variables contribute significantly to explanatory power. While in most cases parameter estimates are precise and conform to expectations and/or findings from prior household-level studies, the R^2's are relatively low, ranging from .03 for the SMOKING equation to .15 for BIRTHS.

The demand estimates for DELAY indicate that women with more education and women in high income families seek prenatal care earlier, and that black mothers postpone such care by just over a half-month more than do white mothers with similar personal and regional characteristics. Among the variables representing the availability of medical services, two variables are associated with mothers obtaining early prenatal care: residence in an SMSA and in counties where more public health facilities include family planning services. Over the sample three-year period, prenatal care was sought earlier during the later years, particularly between 1968 and 1969.

While mothers with husbands who have high levels of earnings appear to smoke more while pregnant, there is a clear negative relationship between the mother's school attainment and the number of cigarettes per day she smokes while pregnant. On average, black women smoke half as much while pregnant as do white mothers, other things equal.[14] Where female unemployment rates are high, mothers appear to smoke less,

Table 2.2 **Linear Input Demand Equation Estimates**

Independent Variable	AGE	DELAY	SMOKING	BIRTHS
MHSI	−3.44	−.561	1.49	−1.06
	(17.06)	(9.48)	(4.46)	(15.33)
MHSC	−2.56	−1.08	−.426	−1.68
	(13.45)	(19.30)	(1.35)	(25.70)
MCOLI	−2.37	−1.25	−1.01	−1.95
	(10.57)	(18.96)	(2.71)	(25.29)
MCOLC	−.873	−1.27	−1.66	−2.21
	(3.42)	(16.87)	(3.91)	(25.19)
HINC($x10^{-3}$)	.548	−.041	.127	.122
	(35.28)	(9.03)	(4.91)	(22.85)
BEDS	258.2	19.16	83.35	60.07
	(4.53)	(1.14)	(0.88)	(3.06)
HEXP	−7.25	−.296	2.65	−1.92
	(2.18)	(0.30)	(0.48)	(1.68)
HOSPFP	29697	−3679	17838	−39247
	(0.84)	(0.35)	(3.04)	(3.23)
HDFP	−1002	−6781	28316	−5646
	(0.12)	(2.66)	(1.96)	(1.89)
MD($x10^{-5}$)	3.71	−.518	.298	−4.00
	(0.47)	(0.22)	(2.27)	(1.47)
UNEMPR-W	−11.20	3.06	−22.68	.948
	(1.86)	(1.73)	(2.27)	(0.46)
SERVICE	−.0094	.0023	−.012	−.00010
	(2.21)	(1.87)	(1.64)	(0.04)
CPRCE	−.226	.054	−.031	.295
	(0.80)	(0.65)	(0.07)	(3.02)
CPRCE2	.0040	−.00083	.0011	−.0045
	(0.92)	(0.65)	(0.15)	(3.00)
TAXSALES	.095	−.042	−.273	−.028
	(0.72)	(1.10)	(1.25)	(0.61)
BLACK	1.16	.661	−2.35	1.03
	(8.13)	(15.77)	(9.90)	(20.96)
SMSA	.090	−.141	.719	−.187
	(0.53)	(2.82)	(2.54)	(3.36)
SIZE($x10^{-8}$)	2.78	−.205	.150	−1.40
	(0.82)	(.205)	(2.65)	(1.20)
1967	.686	−.172	.857	.171
	(5.34)	(4.56)	(4.01)	(3.87)
1968	.222	−.162	.396	.021
	(1.75)	(4.33)	(1.86)	(0.50)
CONSTANT	26.47	2.74	4.20	−2.82
	(5.80)	(2.04)	(0.62)	(1.80)
R^2	.170	.130	.031	.146
F Statistic	98.17	71.70	15.58	82.13

Absolute value of *t* statistics in parentheses.

although the number of doctors per capita and the availability of family planning services in the local area are positively associated with female smoking, as are SMSA residence and city size. The year dummy coefficients suggest a decline in cigarette consumption by pregnant women by 18% from 1967 to 1969.

The AGE equation coefficients suggest a U-shaped relationship between school attainment of mothers and age at infant's birth. While mothers with less than nine years of schooling (the omitted category) appear to be older on average, among women with at least some high school education those with more schooling have their children at older ages. The earnings of the husband appear to be positively associated with delay in childbearing, while in regions of high female unemployment, fertility appears to occur at younger ages. Family planning programs do not appear to affect the timing of births, although local health expenditures per capita are negatively associated with childbearing age of mothers, and the number of hospital beds per capita is positively correlated with this variable. The year dummy coefficients suggest a temporal decline in the average age of childbearing.

The estimates of the BIRTHS equation are consistent with findings of many prior studies of fertility behavior—more educated women tend to have fewer births, husband's earnings and cumulative fertility are positively correlated, and black women have on the average almost one more birth than white women. Mothers in urban environments have lower fertility. Most interesting, while local family planning programs do not appear to influence the timing of births, i.e., affect AGE, the BIRTHS equation indicates that family planning programs are effective in reducing cumulative fertility—the coefficients of both HOSPFP and HDFP are negative and statistically significant. Public health expenditures per capita also appear to reduce total fertility, although public and private hospital BEDS and BIRTHS are positively correlated. Finally, as would be expected in the sample years, fertility displays a decline, by almost one-fifth of a child in the 1967–1969 period.

Birth Characteristics Production Function Estimates: Linear and Generalized Leontief-Diewert Specifications.

Estimates for the three specifications of the production function relating the behavioral variables to birth weight and gestation are reported in Table 2.3 and to birth weight normalized for gestation in the total population and within race groups in Table 2.4. The results suggest that the neglect of population heterogeneity in unobserved health characteristics affects (biases) the estimates of the effects of health input activities on the health characteristics of the newborn: the two-stage least squares (TSLS) and ordinary least squares (OLS) estimates of the production parameters differ substantially. For example, the OLS estimates of the

Leontief model suggest that the mother's delay in seeking prenatal care is an unimportant determinant of birth weight, while use of the more defensible TSLS procedure indicates that such a delay would lower birth weight. This pattern of differences is anticipated, if women who have had problems with their prior pregnancies are more likely to seek early prenatal care and deliver low birth weight infants. These biological factors that are unobserved by the researcher but known to some degree by the woman are responsible for the heterogeneity bias. The direct association between DELAY and birth weight captured in the OLS estimates includes this heterogeneity effect, whereas the TSLS estimates are purged of these effects and confirm that across otherwise comparable women, those who seek medical care earlier in their pregnancy have heavier babies.

The production function estimates also suggest that while the more generalized functional specifications do not yield precise TSLS estimates because of the collinearity among the many transformations of the input variables, the linear (Leontief) specification of the production relationship appears to mask relatively important and anticipated interactions among the designated inputs and our measures of child health.

The marginal products, F_i, of the four input activities evaluated at the sample means are summarized in Table 2.5 for the various functional specifications of the production relationships and for the OLS and TSLS estimates. Discussion focuses here on the TSLS estimates of the marginal productivities of inputs obtained from the generalized Leontief-Diewert functional form. These estimates indicate that delay in seeking prenatal care appears to reduce both birth weight and gestation, and has little residual effect on birth weight normalized for gestation—our proxy for the more permanent health consequences of prematurity (Table 2.5). A delay of six months in obtaining prenatal care is estimated to reduce birth weight by 45 grams or about 1%, and to reduce gestation by 1.6 weeks, or 4%, with a net effect of increasing birth weight normalized for gestation. Direct epidemiological correlational studies have not always found an effect on birth weight of the timing of prenatal care (Eisner 1979).

Smoking while pregnant, on the other hand, notably reduces birth weight but is linked to longer gestation. Smoking is related, therefore, to lower birth weight normalized for gestation. Although other estimates of the effect of smoking on birth characteristics are not precisely comparable to those reported here, the direct correlational evidence of many epidemiological studies as summarized by the recent Surgeon General's report on smoking is that "babies born to women who smoke during pregnancy are, on the average, 200 grams lighter," U.S. Department of Health and Human Services 1980, p. 225). By comparison our direct (OLS) estimates of the fixed coefficient (Leontief) linear model suggest smokers (a third of our sample of mothers) would have babies weighing

Table 2.3 **Birth Characteristics Production Function Estimates: Linear and Generalized Leontief-Diewert**

Independent Variable	A. Birth weight					
	(1)		(2)		(3)	
	OLS	TSLS	OLS	TSLS	OLS	TSLS
AGE	3.58	1.83	−1.48	−32.8	−59.5	−206
	(2.79)	(0.38)	(0.64)	(1.54)	(2.81)	(1.32)
DELAY	−1.56	−39.6	−44.3	−177	−37.6	−129
	(0.42)	(1.71)	(3.05)	(1.16)	(1.68)	(0.62)
SMOKING	−10.1	−16.2	5.48	−1.80	5.74	−9.76
	(15.4)	(3.49)	(2.47)	(0.06)	(2.54)	(0.32)
BIRTHS	20.9	43.3	−14.7	128	−74.2	43.9
	(5.34)	(2.31)	(1.64)	(0.79)	(3.18)	(0.24)
$(AGE \cdot DELAY)^{1/2}$			27.7	193	−8.65	155.
			(2.56)	(1.79)	(0.27)	(0.58)
$(AGE \cdot SMOKE)^{1/2}$			−13.5	12.1	−9.19	−44.1
			(4.65)	(0.34)	(1.26)	(0.61)
$(AGE \cdot BIRTHS)^{1/2}$			10.8	43.8	83.09	236
			(1.79)	(0.41)	(2.29)	(0.93)
$(DELAY \cdot SMOKE)^{1/2}$			−3.74	21.2	−1.30	20.7
			(0.58)	(0.27)	(0.17)	(0.24)
$(DELAY \cdot BIRTHS)^{1/2}$			22.5	−276	58.7	−213
			(1.52)	(1.20)	(1.79)	(0.76)
(SMOKE · BIRTHS)			−3.66	−100	−6.99	−80.8
			(0.87)	(1.28)	(0.87)	(0.98)
$AGE^{1/2}$					533	1568
					(2.66)	(1.00)
$DELAY^{1/2}$					86.5	−77.5
					(0.53)	(0.05)
$SMOKING^{1/2}$					−20.0	296
					(0.58)	(0.80)
$BIRTHS^{1/2}$					−178	−809
					(1.11)	(0.72)
BLACK	−252	−257	−245	−234	−244	−229
	(16.8)	(11.1)	(16.3)	(8.16)	(16.1)	(6.54)
SMSA	−20.8	−18.9	−21.0	−17.3	−21.8	−16.4
	(1.55)	(1.31)	(1.57)	(1.16)	(1.63)	(1.08)
$SIZE(x10^{-8})$	465	525	475	388	482	300
	(1.54)	(1.67)	(1.60)	(1.15)	(1.62)	(0.86)
1967	18.1	19.8	22.4	31.9	23.7	30.8
	(1.32)	(1.36)	(1.64)	(1.89)	(1.73)	(1.56)
1968	14.8	14.2	19.6	25.2	19.9	25.7
	(1.08)	(0.99)	(1.43)	(1.53)	(1.45)	(1.48)
CONSTANT	3263	3360	3267	3190	1943	−205
	(95.0)	(24.0)	(91.9)	(18.1)	3.84	0.05
R^2	.053	—	.061	—	.062	—
F	—	29.46 (10,9611)	—	17.53 (16,9605)	—	13.82 (20,060)

In parentheses are the absolute values of the t and asymptotic t statistics for the OLS and TSLS coefficients, respectively.

B. Gestation Period ($x10^2$)					
(1)		(2)		(3)	
OLS	TSLS	OLS	TSLS	OLS	TSLS
−6677	1.46	−1.34	14.6	−28.4	−166
(1.20)	(0.70)	(1.32)	(1.54)	(3.03)	(2.28)
−2.25	−8.23	−19.4	91.1	−16.9	233
(1.38)	(0.82)	(3.03)	(1.33)	(1.72)	(2.44)
−.784	1.45	.398	−14.7	.236	−19.6
(2.72)	(0.72)	(0.41)	(1.13)	(0.24)	(1.37)
.174	−5.26	1.01	102	6.15	34.6
(0.10)	(0.65)	(0.25)	(1.42)	(0.60)	(0.41)
		8.97	−53.9	10.49	31.5
		(1.88)	(1.12)	(0.74)	(0.25)
		−1.30	19.9	−3.51	−5.17
		(1.01)	(1.25)	(1.09)	(0.15)
		−6.03	−45.3	−5.32	94.6
		(2.28)	(0.96)	(0.33)	(0.80)
		.086	−6.39	.385	18.2
		(0.03)	(0.18)	(0.12)	(0.45)
		16.8	−75.9	11.5	−125
		(2.57)	(0.74)	(0.80)	(0.96)
		.458	−3.01	2.45	9.62
		(0.25)	(0.09)	(0.69)	(0.25)
				282	1497
				(3.19)	(2.05)
				−14.1	−855
				(0.20)	(1.28)
				7.99	99.5
				(0.52)	(0.58)
				−17.6	−401
				(0.25)	(0.77)
−71.5	−53.6	−70.7	−62.3	−69.7	−64.1
(10.8)	(5.31)	(10.7)	(4.87)	(10.4)	(3.94)
.996	−4.56	1.13	−4.93	.677	−5.08
(0.17)	(0.72)	(0.19)	(0.74)	(0.11)	(0.72)
−282	−354	−283	−376	−290	−418
(2.15)	(2.59)	(2.16)	(2.50)	(2.21)	(2.56)
54.5	52.7	55.0	44.5	56.2	42.8
(9.05)	(8.28)	(9.12)	(5.90)	(9.31)	(4.65)
11.1	11.1	12.0	3.15	12.8	3.69
(1.85)	(1.78)	(1.99)	(0.43)	(2.12)	(0.46)
3927	3896	3922	3969	3222	1153
(261)	(63.9)	(250)	(50.3)	(14.4)	(0.58)
.024	—	.025	—	.027	—
—	24.94	—	14.21	—	10.76
	(10,9611)		(16,9605)		(20,9601)

Table 2.4 **Production Function Estimates of Birth Weight Normalized for Gestation: Linear and Generalized Leontief-Diewert**

Independent Variable	A. Total Population Normalization ($x10^2$)					
	(1)		(2)		(3)	
	OLS	TSLS	OLS	TSLS	OLS	TSLS
AGE	.112	−.057	−.017	−1.68	−.802	.315
	(3.04)	(0.39)	(0.25)	(2.57)	(1.26)	(0.07)
DELAY	−.623	−1.03	−.795	−7.99	−.803	−12.1
	(0.56)	(1.48)	(1.83)	(1.70)	(1.20)	(1.89)
SMOKING	−.275	−.563	.177	.766	.194	.793
	(14.1)	(4.03)	(2.67)	(0.85)	(2.87)	(0.84)
BIRTHS	.600	1.57	−.398	.518	−2.33	1.45
	(5.12)	(2.80)	(1.48)	(0.10)	(3.34)	(0.25)
$(AGE \cdot DELAY)^{1/2}$			.637	7.80	−.611	3.54
			(1.97)	(2.40)	(0.64)	(0.43)
$(AGE \cdot SMOKE)^{1/2}$			−.409	−.395	−.144	−.529
			(4.70)	(0.36)	(0.66)	(0.24)
$(AGE \cdot BIRTHS)^{1/2}$			.467	3.81	2.62	2.12
			(2.61)	(1.17)	(2.42)	(0.27)
$(DELAY \cdot SMOKE)^{1/2}$			−.0773	1.73	.0200	.970
			(0.40)	(0.72)	(0.09)	(0.36)
$(DELAY \cdot BIRTHS)$			.102	−8.05	1.47	−5.68
			(0.23)	(1.14)	(1.50)	(0.66)
$(SMOKE \cdot BIRTHS)^{1/2}$			−.0818	−4.57	−.249	−4.62
			(0.65)	(1.90)	(1.04)	(1.81)
$AGE^{1/2}$					6.16	−10.7
					(1.03)	(0.22)
$DELAY^{1/2}$					3.86	32.3
					(0.80)	(0.73)
$SMOKING^{1/2}$					−1.26	1.56
					(1.22)	(0.14)
BIRTHS					−5.16	1.69
					(1.07)	(0.05)
BLACK	−5.16	−5.97	−4.95	−4.68	−4.95	−4.69
	(11.5)	(8.55)	(11.0)	(5.32)	(10.9)	(4.32)
SMSA	−.815	−.522	−.821	−.458	−.828	−.416
	(2.02)	(1.20)	(2.05)	(1.00)	(2.06)	(0.88)
SIZE ($x10^{-8}$)	30.9	35.8	31.3	30.7	31.8	30.8
	(3.48)	(3.79)	(3.53)	(2.97)	(3.59)	(2.83)
1967	−1.64	−1.52	−1.52	−.820	−1.51	−.699
	(4.01)	(3.46)	(3.71)	(1.58)	(3.70)	(1.14)
1968	−.073	−.094	.052	.529	.036	.569
	(0.18)	(0.22)	(0.13)	(1.05)	(0.09)	(1.06)
CONSTANT	99.1	105	99.2	95.8	838	94.9
	(97.0)	(24.8)	(93.3)	(17.7)	(5.54)	(0.72)
R^2	.0398		.0468		.047	
F		16.88		10.39		8.04

In parentheses are the absolute values of the t and asymptotic t statistics for the OLS and TSLS coefficients, respectively.

B. Race-Specific Normalization ($x10^2$)					
(1)		(2)		(3)	
OLS	TSLS	OLS	TSLS	OLS	TSLS
.115	−.078	−.148	−1.62	−.982	.275
(3.00)	(0.40)	(1.33)	(2.50)	(1.54)	(0.06)
.101	−1.04	−.592	−7.96	−.705	−11.4
(0.88)	(1.50)	(1.13)	(1.70)	(1.06)	(1.81)
−.275	−.562	.188	.709	.202	.705
(14.1)	(4.04)	(2.83)	(0.80)	(2.98)	(0.75)
.592	1.57	−1.94	.700	−2.20	1.44
(5.03)	(2.80)	(3.16)	(0.14)	(3.14)	(0.26)
		.233	7.62	−.424	4.33
		(0.54)	(2.31)	(0.44)	(0.53)
		−.384	−.343	−.196	−.647
		(3.71)	(0.31)	(0.90)	(0.29)
		1.53	3.55	2.45	2.12
		(3.39)	(1.10)	(2.26)	(0.27)
		−.0853	1.70	.0226	.933
		(0.42)	(0.72)	(0.10)	(0.35)
		.905	−7.62	1.33	−5.44
		(1.17)	(1.09)	(1.35)	(0.64)
		−.162	−4.53	−.252	−4.50
		(0.74)	(1.89)	(1.05)	(1.78)
				8.00	−11.5
				(1.33)	(0.24)
				2.91	25.6
				(0.60)	(0.58)
				−1.04	2.51
				(1.00)	(0.22)
				−4.57	1.17
				(0.94)	(0.03)
1.81	1.94	−1.12	.686	−1.08	−.633
(2.89)	(2.80)	(2.47)	(0.78)	(2.38)	(0.59)
−.718	−.505	−.723	−.441	−.733	−.409
(1.77)	(1.17)	(1.79)	(0.97)	(1.81)	(0.88)
30.4	35.5	31.1	30.4	31.2	30.1
(3.42)	(3.77)	(3.51)	(2.96)	(3.52)	(2.79)
−1.53	−1.48	−1.43	−.806	−1.40	−.731
(3.73)	(3.38)	(3.50)	(1.57)	(3.40)	(1.20)
−.034	−.058	.062	.544	.081	.565
(0.08)	(0.14)	(0.15)	(1.08)	(0.20)	(1.06)
97.5	104	97.0	95.4	78.4	102
(99.7)	(24.7)	(97.2)	(17.7)	(5.17)	(0.78)
.0318		.0386		.0389	
	7.06		4.91		3.83

Table 2.5 **Estimates of Marginal Products of Inputs at Sample Means on Birth Characteristics**

Model specification and estimation techniques	Marginal Products (F_i) AGE (years)	DELAY (months)	SMOKING (cigarettes per day)	BIRTHS (number)
Leontief (input-output coefficient)				
OLS	3.58	−1.56	−10.1	20.9
TSLS	1.83	−39.6	−16.2	43.3
Leontief-Diewert Locally linear Homogeneous				
OLS	1.91	5.80	−12.82	11.41
TSLS	8.83	−4.79	−16.42	−15.14
Leontief-Diewert Generalized				
OLS	3.58	8.32	−12.50	25.8
TSLS	4.48	−7.54	−13.95	−6.35
Gestation (weeks x 10^2)				
Leontief (input-output coefficient)				
OLS	−.677	−2.25	−.784	.174
TSLS	1.46	−8.23	1.45	−5.26
Leontief-Diewert Locally Linear Homogeneous				
OLS	−1.09	2.24	−0.896	0.631
TSLS	2.77	−30.76	4.64	−10.41
Leontief-Diewert Generalized				
OLS	−.0662	0.424	−0.914	−0.484
TSLS	2.85	−25.8	7.88	−1.63
Race-Specific Standardized Birth Weight ($x10^2$)				
Leontief (input-output coefficient)				
OLS	.115	.101	−.275	.592
TSLS	−.078	−1.04	−.562	1.57
Leontief-Diewert Locally Linear Homogeneous				
OLS	.0506	.138	−.346	.814
TSLS	.135	.978	−.695	−.794
Leontief-Diewert Generalized				
OLS	.0946	.188	−.347	.721
TSLS	.0405	.856	−.753	−.769

Source: Derived from tables 1, 3, and 4.

144 grams less than nonsmokers (i.e., $-10.1 * 4.71 * 1/.33$). The generalized Leontief-Diewert approximation to the production function implies that this average level of smoking of fourteen cigarettes a day lowers birth weight by 179 grams, based on direct (OLS) partial correlations. When population heterogeneity is taken into account, the impact of smoking is increased further to 195 grams ($-13.95 * 14$).[15]

The epidemiological evidence based on direct correlations is that smoking only minimally reduces gestation by about two days (Ibid., p. 229), which is not inconsistent with our Leontief OLS estimate of a reduction of about one day ($-.784$ weeks$* 14 \div 100$). However, when population heterogeneity is taken into account in the TSLS estimates, babies of smokers are found to have 1.1 weeks longer gestation, leading to our new finding that for birth weight normalized for gestation the impact of smoking is substantially increased. The generalized TSLS estimates imply that the average consumption of cigarettes by smokers is related to an 11% reduction in birth weight normalized for gestation ($-.753 * 14$). The retardation in the fetal growth rate attributable to average smoking levels is, therefore, larger than has heretofore been estimated.

The effects of age and fertility of the mother appear to be nonlinearly related to birth weight in other studies using quite different analytical techniques (Eisner et al. 1979), and thus considering only the average effect of these variables may obscure their effects in particular segments of the population. At the means of the sample, the effect of age is to increase birth weight slightly and to increase more strongly the gestation of the newborn. No substantial average effect of age is noted, therefore, on normalized birth weight. The number of births the mother has had slightly decreases birth weight, but may decrease the period of gestation, with a consequent negative effect on normalized birth weight.

Interactions between inputs, particularly with AGE (Tables 2.3 and 2.4), appear quantitatively important in several cases, confirming the cluster of characteristics that describe subpopulations which are at high risk of having premature babies. Births to very young mothers and to women who have already had many births tend to be particularly low in weight. Moreover, the AGE and BIRTHS interactions with DELAY suggest that delay in seeking prenatal care is more critical for younger mothers and for high-fertility mothers, whether or not birth weight is normalized for gestation. Moreover, the deleterious effects of smoking on birth weight and birth weight normalized for gestation are increased for older mothers as well as for mothers having many births. The positive birth weight effect of the AGE-BIRTHS interaction suggests that delaying childbearing (or childspacing) enhances, on balance, the health prospects for the newborn. The one interaction that is hard to interpret is the positive birth weight effect of smoking and delaying prenatal medical care.

The estimates of the health production function also indicate that once fertility, age at birth of the mother, health-related activities, and the presence of heterogeneity are taken into account, there remains a two-thirds of a week difference in average gestation period between black and white mothers. There is also a difference in birth weight by race—even after taking into account differences in input behavior, represented here by fertility, age, smoking, and the timing of prenatal care. There is a statistically significant 229 gram differential in birth weight between the babies of black and white mothers, and about a 5% difference in the total population rate of uterine growth (normalized birth weight, Table 2.4). Further investigation shows that this racial difference in birth weight does not appear to be a function of racial differences in the sex ratio at birth—while female infants appeared to have a slightly lower birth weight than did male infants, there is not a statistically significant higher proportion of female infants among blacks than among whites in the sample. However, when race-specific birth weight-gestation functions are estimated and employed to normalize birth weight in Table 2.4-B, the black-white difference disappears in the TSLS estimates of the Leontief-Diewert production function. These findings suggest that there may be distinct black-white differences in the biological-technical relationship between birth weight and gestation. It is possible, moreover, that distinct scaling of these birth characteristics by genetic group would improve their predictive value as indicators of future child health across a mixed population. In the Appendix Tables 2.A.1 and 2.A.2 we report the estimates for all three birth characteristic production functions based only on births of white mothers.

Birth Characteristics, Socioeconomic Variables and Health Programs: Linear Reduced Forms

Table 2.6 presents estimates of the reduced-form equations relating the socioeconomic variables and those variables representing the availability of health services and programs to the three birth characteristics. These estimates contain several puzzles. For example, the relationship between the schooling level of the mother and birth weight is U-shaped; mothers with only some high school education have babies with the lowest birth weight, whether or not standardized for gestation period, while mothers with less than nine years of schooling bear children of about the same weight as mothers with at least high school educations. Family planning programs associated with hospitals (HOSPFP) appear to reduce birth weight significantly, as does residing in an SMSA. In contrast, husband's income (HINC) and the unemployment of women are positively associated with birth weight. The estimates of the behavioral and technical relationships of Tables 2.2, 2.3, and 2.4 and the computed sample mean marginal products should help account for these findings, however, since

Table 2.6 **Birth Characteristics Reduced Form Demand Equations**

Independent Variable	Birth weight	Birth weight ($x10^2$)*	Gestation Period
MHSI	−60.28	−2.15	.104
	(2.71)	(3.27)	(1.09)
MHSC	−16.62	−1.01	.175
	(0.80)	(1.63)	(1.94)
MCOLI	−11.58	−1.11	.231
	(0.47)	(1.52)	(2.18)
MCOLC	−12.85	−1.26	.253
	(0.46)	(1.51)	(2.09)
HINC($x10^{-2}$)	5.45	.1410	.0010
	(3.20)	(2.78)	(0.14)
BEDS	10590	358.3	−18.08
	(1.78)	(1.93)	(0.67)
HEXP	427.86	8.60	1.72
	(1.17)	(0.79)	(1.09)
HOSPFP	−125758	−243276	−24882
	(3.25)	(2.11)	(1.49)
HDFP	−227338	−16314	663.1
	(0.03)	(0.58)	(0.16)
MD($x10^{-5}$)	−1063.7	31.76	1.83
	(1.23)	(1.23)	(0.49)
UNEMPR-W	2105.1	96.77	−2.93
	(3.20)	(4.94)	(1.03)
SERVICE	−1.62	−.048	.001
	(3.48)	(3.46)	(0.60)
CPRCE	38.16	1.51	−.069
	(1.23)	(1.64)	(0.52)
CPRCE2	−.518	−.022	−.0014
	(1.09)	(1.60)	(0.66)
TAXSALES	−5.51	.685	−.247
	(0.38)	(1.61)	(4.00)
BLACK	−184.98	−3.35	−.642
	(11.83)	(7.20)	(9.52)
SMSA	−50.62	−1.49	−.093
	(2.71)	(2.68)	(1.16)
SIZE($x10^{-8}$)	−76.70	16.53	−4.05
	(0.21)	(1.49)	(2.52)
1967	20.79	−1.59	.547
	(1.48)	(3.79)	(9.02)
1968	14.98	−.124	.063
	(1.07)	(0.30)	(2.09)
Constant	2627.6	74.00	40.21
	(5.25)	(4.97)	(18.64)
R^2	.030	.021	.027
F	14.84	10.38	13.09

*Normalized for gestation within total population.

the reduced-form birth characteristics equations reflect the marginal products of the health-related activities as well as the effects of the socioeconomic and program variables on the levels of input activities demanded.

The estimates of the production functions presented in Tables 2.3 and 2.4 indicated that of the activities considered, delay in seeking prenatal medical care, smoking during pregnancy, and birth order had significant effects on child health, with such effects somewhat dependent on the age of the mother. At the sample means, however, birth weight appeared to be most sensitive to levels of smoking—Table 2.5 indicates that while a delay in seeking medical care of six months would lower birth weight by 45 grams, an increase of one pack of cigarettes per day during pregnancy lowers birth weight by 279 grams or by 8.5%. The estimates of Table 2.2 suggested that the health-related activities were importantly but differently related to both the income and educational level of the father and mother, respectively, as well as to the program and health services variables. These findings together imply that the nonlinear effects of education on birth weight are the result of the differential effects of schooling on the several input activities. For example, women with incompleted high school educations (MHSI) appeared to have the second highest fertility of all the educational groups, to smoke more when pregnant than all other women, and to delay more in seeking prenatal medical care than did women with higher levels of schooling. This combination of behavior is consistent with the finding reported in Table 2.6 that this educational group has the lowest birth weight children. The birth weight of women with higher schooling levels, however, appears to differ only modestly from that of women with less than a high school educations, mainly because of only small differences in smoking habits between these groups.

Because of the impact on birth weight of the timing of prenatal medical care, the relatively strong effects of HINC in hastening the utilization of prenatal medical services appears to account for the net positive association between husband's earnings and birth weight. These effects are evidently only partly offset by the tendency of mothers with higher-income husbands to consume more cigarettes while pregnant and to have higher fertility.

The negative effect (marginal product) of birth order on birth weight stands in contrast to the significant negative effect of hospital family planning programs on birth weight—such programs, which appear to be successful in reducing family size, should increase, through the BIRTHS effect, average birth weight levels. However, while BIRTHS appears to be lower in urban settings, such environment appears to be associated with significantly greater cigarette consumption by expectant mothers; because of the large effect of smoking, birth weight tends to be lower in

SMSAs, even though such areas appear to provide better access to and thus encourage earlier use of prenatal medical services.

Finally, we have seen that differences in health-related behavior of mothers do not account for all of the difference in average birth weight between the black and white children, although such behavioral differences account for more of the difference in gestation by race. Differences [between black and white women] in input activities affecting newborn health appear to have an ambiguous net impact on birth weight and gestation: while black mothers postpone seeking prenatal medical care, they smoke significantly less than do white mothers while pregnant, and have larger families. The net effect of this behavioral combination on gestation length is minimal. However, controlling for socioeconomic characteristics and variables representing the availability of health services and programs at the county level, the black-white birth weight differential remains 185 grams, and controlling for the health-related input behavior of the mother that differential is of similar magnitude, namely 229 grams. This suggests that the net impact of black-white differences in measured behavior does not account for the noted birth weight differential between these racial groups. Normalizing birth weight for period of gestation, moreover, reduces the black-white differential only modestly (Table 2.4). Black infants weigh 5–6% less than whites, given gestation and the measured health input behavior of the mother. But if the relationship between birth weight and gestation is fit separately for white and black births in the sample, the TSLS estimates of a race-specific normalized birth weight production function exhibit no statistically significant black-white differences.

Conclusion

In this paper we have formulated an economic model of the household in order to estimate (1) the determinants of activities (inputs) affecting the production of the weight of children at birth and length of gestation, and (2) the biological-technical relationships between parental behavior and birth outcomes in the presence of population heterogeneity. The theoretical model was used to illustrate the advantages of estimating jointly the health production technology and the determinants of the activities potentially affecting infant health, particularly when households differ (are heterogenous) with respect to factors affecting health which are known to the households but not to the researcher. The empirical analysis, based on a probability sample of over 9,000 legitimate births in the United States between 1967 and 1969 combined with geographical information on prices and health programs, considered four endogenous health-related inputs—smoking while pregnant, timing of prenatal medical care, mother's age at birth and birth order—and, ini-

tially, two dimensions of prematurity at birth—birth weight and gestational age.

Experimentation with functional forms for the birth outcome production function indicated that estimates of the impact of household input activities on birth characteristics of children are more sensitive to whether or not the estimates account for heterogeneity than to the choice of the functional form. In particular, heterogeneity appeared to almost completely mask a negative impact on child health of the mothers' delay in seeking medical care. Estimates of the production functions also indicated that smoking by the mother while pregnant had the largest (negative) impact on birth weight and on the rate of fetal growth of all the inputs considered while work by the mother during pregnancy did not appear to affect the birth outcomes. Significant interactions were also found among birth order, the timing of births, prenatal care, and smoking; however, the estimated production functions indicated that the four behavioral inputs are more important in the determination of birth weight than of gestation, suggesting that variation in gestation may be less affected by economic and social conditions and more a reflection of biologically exogenous variability.

Since it has been suggested that babies who are underweight relative to other infants of the same gestation exhibit weight deficiencies that persist into later childhood and, after the first month of life, are sick and die more frequently, a normalization procedure was also developed to isolate these more permanent impairments of prematurity related to the rate of fetal growth. Widely noted, but infrequently analyzed, differences in the distribution of birth weight by gestation between white and black births led us to also perform this standardization procedure within each sample for black and white births separately, and then combine these normalized birth weight values in estimating the total sample child health production function. In some instances we found that the effects of an input on birth weight and gestation cancel in terms of their permanent effects on normalized birth weight, as in the case of the mother's age, or are magnified, as in the case of smoking, indicating that the effects of smoking by the mother while pregnant may have a more lasting effect on the child's health. The impact of smoking on the rate of fetal growth (birth weight normalized for gestation) was doubled by taking account of population heterogeneity.

Our treatment of the heterogeneity problem used information on local market prices and health programs both to estimate input demand equations and to identify the child health production functions. We think it unlikely that the demand for health input activities would be independent of latent population characteristics that affect child health production and are known to households. The appropriate treatment of this form of population heterogeneity is crucial for obtaining consistent estimates of

underlying health production functions and deriving sound causal conclusions that might be useful for policy.

The next step in such an analysis is to ask whether the identifying program and price variables are themselves independent of our heterogeneous population characteristics, i.e., μ. Government health programs may be established to serve groups in the population that are known by the government to have distinctly different health endowments or environments, or in our notation different values of μ. Alternatively, individuals may themselves migrate to regions with lower prices for preferred inputs and/or available programs; in other words, individuals are drawn selectively to certain regions according to their own perceived health endowments or preferences. In either instance, the source of identifying information required to obtain consistent estimates of the child health production function may prove more difficult to observe. Estimates of input productivities and price and program effects based on regional price and program information could in this case be inconsistent, for the regional variables would no longer be independently distributed with regard to heterogeneous population characteristics.

Notes

1. See, for example, Baumgartner (1962), Beck and van den Berg (1975), Chase (1969), Chernichovsky and Coate (1979), Eisner et al. (1979), and Shapiro (1965).

2. Examples of behavioral correlates of early child health indicators, based on univariate associations, are medical care (Shah and Abbey 1971, Rosenwaike 1971, and Iba et al. 1973), cigarette smoking by mothers (Hobel et al. 1971) and wife's work (Coombs et al. 1969).

3. We abstract from uncertainty, or alternatively, assume that parents are risk-neutral. Under the latter assumption random effects on health outcomes unknown to the family decision-makers at the time when decisions are made will not enter the process of optimization. Variations in μ, however, do effect decisions and, as shown below, have important econometric implications. See also Mundlak and Hoch (1965).

4. Becker and Lewis (1973) and Becker and Tomes (1974). For a discussion of the predictive content of models which assume interactions between family size and investments in children, see Rosenzweig and Wolpin (1980).

5. See, for example, Michael (1973) and Grossman (1972). The conventional assumption that education is exogenous to adult demand behavior might also require reconsideration here. Some young women become pregnant unexpectedly and therefore terminate their schooling at an earlier stage than they would otherwise. If these women also tend to have low birth weight babies, part of the association between mother's low education and low birth weight would be generated by unplanned pregnancies and the endogeneity of the mother's educational attainment. This effect may be reduced by the exclusion of illegitimate births from the natality followback survey files.

6. Victor Fuchs noted that this development might be explained by the increasing proportion of nonwhite births occurring in hospitals in this period, where (low) birth weight was more accurately reported. (See Querac and Spratley 1978, Fig. 6.) In 1950 the nonwhite

"prematurity" rate was 10.2% and by 1967 had increased to 13.6%, while the rate for whites was 7.1% in both years (Chase and Byrnes 1972). In the mid–1960s, 8% of all U.S. births were under 2,500 grams, but these births accounted for 62% of the deaths occurring in the first year of life (MacMahon et al. 1972). It is not surprising, therefore, that some investigations have found birth weight alone explains an overwhelming share (90%) of the variance in perinatal mortality (Susser et al. 1972).

7. The authors plan subsequent research to explore nonlinear transformations of birth weight and mortality outcomes as well.

8. The estimates are

$$\begin{aligned}
&\text{total sample: birth weight} = 10107 - 1042\ \text{weeks} + 37.8\ \text{weeks}^2 - .398\ \text{weeks}^3 \\
&\qquad\qquad\qquad\qquad (7.72)\quad (9.95)\qquad\quad (10.44)\qquad\quad (10.90) \\
&\qquad R^2 = .227,\ n = 9763 \\
&\text{whites: birth weight} = 13416 - 1354\ \text{weeks} + 47.3\ \text{weeks}^2 - .492\ \text{weeks}^3 \\
&\qquad\qquad\qquad\qquad (8.48)\quad (9.13)\qquad\quad (10.4)\qquad\quad (10.9) \\
&\qquad R^2 = .201,\ n = 7896 \\
&\text{blacks: birth weight} = 9188 - 937\ \text{weeks} + 34.4\ \text{weeks}^2 - .366\ \text{weeks}^3 \\
&\qquad\qquad\qquad\qquad (4.39)\ (4.57)\quad (5.26)\qquad\quad (5.39) \\
&\qquad R^2 = .273,\ n = 1867
\end{aligned}$$

where the absolute values of t statistics are reported in parentheses beneath the regression coefficients, and n is the sample size. For additional evidence of this relationship, see Querec and Spratley (1978, Fig. 2).

9. The unexplained variation of individual birth weights around the estimated fetal growth function is undoubtedly heteroscedastic; that is, the absolute magnitude of this variation increases systematically with the level of birth weight expected on the basis of gestation, or $f(t)$. If the variance of these individual deviations increased in proportion to $f(t)^2$, our normalization measure of birth weight controlling for gestation would exhibit a constant variance error, and standard estimation procedures would then be efficient. Heteroscedasticity would, on the other hand, reduce the efficiency of our estimates, but not affect their consistency or probability limits in a large sample.

10. Infant mortality rates in the United States in 1960 were 41.4 per thousand nonwhite births and 22.2 per thousand white births. Also, 12.9% of nonwhite births weighed less than 2,500 grams, whereas only 6.8% of the white births were so classified. Yet when periods of infancy are distinguished, mortality rates for whites exceed those for nonwhites in each of these periods within the high mortality weight categories below 3000 grams (Chase, 1962). This might suggest that nonwhite births have a lower distribution of birth weights than do whites, given similar health inputs. Differences between races in pelvic structures might rationalize such a difference in birth weight. Also, more rapid postnatal skeletal growth has been noted in nonwhite than in white U.S. populations. Regardless of the origin or function of possible racial differences, separate normalizations of birth weight for gestational age are explored since the size of the non white sample population did not permit us to estimate with any confidence the parameters of the production function for each racial group separately.

11. We set $DELAY$ equal to the sample mean gestation period (39 weeks) if no prenatal medical care was sought, and to 4 weeks if "immediate" care was received upon learning of the pregnancy.

12. The formula for computing elasticities of substitution between any two inputs y_i and y_j (σ_{ij}) based on production functions which are linear in parameters (Fuss and McFadden 1978, Chapter II.1) is

$$\sigma_{ij} = \left[-F_{ii}/F_i^2 + 2(F_{ij}/F_iF_j) - F_{jj}/F_j^2 \right] \left[(y_iF_i)^{-1} + (y_jF_j)^{-1} \right]^{-1}$$

where it is assumed that the quantities of other inputs and output are held constant.

13. An alternative strategy which could provide consistent estimates of the health production function in the presence of heterogeneity would make use of differences in birth outcomes and parental behavior between births within the same family. To implement such a technique would require longitudinal data or good retrospective information on prior births and would necessitate the assumption that (perceived) μ is constant across all births in the same household, ruling out modifications in expectations through experience. This technique can only be applied, of course, to families with at least two live births and would suffer from the imprecision of estimates obtained from most individual fixed effects models.

14. In 1978 black women over the age of 17 were less likely to be smokers than white women, 39.8% versus 45.6% (U.S. Department of Health and Human Services, 1979, Table 3). Moreover, white women over the age of 17 reported smoking more cigarettes per day: twice the percentage of black as white female smokers reported smoking less than fifteen cigarettes daily, whereas five times as many white as black female smokers reported smoking twenty-five cigarettes or more daily (Table 5). These estimates based on the National Health Interview Survey for 1978 are similar to those published in earlier years, but rarely are the black-white differences reported in greater detail, by age and sex and in particular by pregnancy status.

15. Analysis of current population survey data collected in June 1966 (Ahmed and Gleeson 1970) confirms roughly similar levels of cigarette consumption as found in the 1967–69 NNFS, assuming that about one-third of the smokers stop smoking during pregnancy. For example, among women aged 25–44, 42.9% were currently smokers in June 1966. This percentage fell to 38.8% by 1970 (U.S. Department of Health and Human Services, 1979, Table 1) and to 35.9% in 1978. From the 1966 survey (Table 6), one can estimate that among smokers 16.4 cigarettes were smoked per day, implying an average for the total female population aged 25–44 of 7.04 cigarettes daily. If one-third of the smokers stopped smoking while pregnant, but those continuing to smoke continued to smoke 16.4 cigarettes daily, the average consumption for pregnant women would have been 4.7 cigarettes. This compares with our estimate for legitimate births in the three subsequent years of 1967–69 of 4.71 cigarettes per day.

References

Ahmed, P.I., and Gleeson, G.A. 1970. Changes in cigarette smoking habits between 1955 and 1966. *Vital and Health Statistics,* series 10, no. 59. Rockville, Maryland: U.S. Department of Health and Human Services.

Baumgartner, L. 1962. The public health significance of low birth weight in U.S.A. *Bulletin of the World Health Organization* 26: 175–82.

Beck, G.J., and van den Berg, B.J. 1975. The relationship of the rate of intrauterine growth of low birth weight infants to later growth. *Journal of Pediatrics* 86: 504–11.

Becker, G., and Lewis, H.G. 1974. On the interaction between quantity and quality of children. *Journal of Political Economy* 82: S279–S288.

———, and Tomes, N. 1976. Child endowments and the quantity and quality of children. *Journal of Political Economy* 84: S143–S162.

Chase, H.C. 1969. Infant mortality and weight at birth: 1960 United States birth cohort. *American Journal of Public Health* 59: 1618–28.

———, and Byrnes, M.E. 1972. Trends in prematurity: United States, 1950–1967. *Vital and Health Statistics,* series 3, no. 15. Rockville, Maryland: U.S. Dept. of Health, Education, and Welfare.

Chernickovsky, D., and Coate, D. 1979. An economic analysis of diet, growth, and health of young children in the United States. Working Paper No. 416. Cambridge: National Bureau of Economic Research.

Coombs, L.; Freedman, R.; and Namboothiri. 1969. Inferences about abortion from foetal mortality data. *Population Studies* 23: 247–65.

Diewert, W.E. 1971. An application of the Shepherd duality approach: a generalized Leontief production function. *Journal of Political Economy* 79, no. 3: 481–507.

Eisner, V.; Brazie, J.V.; Pratt, M.W.; and Hexter, A.C. 1979. The risk of low birth weight. *American Journal of Public Health* 69: 887–93.

Fuss, M., and McFadden, D. 1978. *Production economics: a dual approach to theory and applications,* vols. 1 and 2. Amsterdam: North-Holland.

Grossman, M. 1972. On the concept of health capital and the demand for health. *Journal of Political Economy* 80:223–55.

Hemminki, E., and Starfield, B. 1978. Prevention of low birth weight and preterm birth: literature review and suggestions for research policy. *Milbank Memorial Fund Quarterly–Health and Society* 56: 339–61.

Hobel, R.; Entwisle, G.; and Tayback, M. 1971. A risk adjustment technique for comparing prematurity rates among clinic populations. *HMSHA Health Reports* 86:946–52.

Iba, B.Y.; Niswander, J.D.; and Woodville, L. 1973. Relation of prenatal care to birth weights, major malformations, and newborn deaths of American Indians. *Health Services Reports* 88: 697–701.

MacMahon, B.; Kovar, M.G.; and Feldman, J.J. 1972. Infant mortality rates: socioeconomic factors in the United States. *Vital and Health Statistics,* series 22, no. 14. Rockville, Maryland: U.S. Dept. of Health, Education, and Welfare.

Meyer, M.B.; Jonas, B.C.; and Tonascia, J.A. 1976. Perinatal events associated with maternal smoking during pregnancy. *American Journal of Epidemiology* 103: 464–76.

Michael, R.T. 1973. Education in nonmarket production. *Journal of Political Economy* 81: 306–27.

Mudlak, Y., and Hoch, I. 1965. Consequences of alternative specifications in estimation of the Cobb-Douglas functions. *Econometrica* 33: 814–28.

Querec, N.A., and Spratley, E. 1978. Characteristics of births: United States, 1973–1975. *Vital and Health Statistics,* series 21, no. 30. Hyattsville, Maryland: U.S. Department of Health, Education, and Welfare.

Rosenwaike, I. 1971. The influence of socioeconomic status on incidence of low birth weight. *HMSHA Health Reports* 86: 641–49.

Rosenzweig, M.R., and Schultz, T.P. 1980. Birth weight, the production of child health, and input demand. Discussion Paper 352, Yale Economic Growth Center.

———, and Wolpin, K.I. 1980. Testing the quantity-quality fertility model: the use of twins as a natural experiment. *Econometrica* 48: 227–40.

Shah, F.K., and Abbey, H. 1971. Effects of some factors on neonatal and postneonatal mortality. *Milbank Memorial Fund Quarterly* 49:33–57.

Shapiro, S. 1965. Weight of birth and survival of the newborn: United States, early 1950. *Vital and Health Statistics,* series 21, no. 3. Washington: U.S. Department of Health, Education, and Welfare.

Susser, M.W.; Marolla, F.A.; and Fleiss, J. 1972. Birth weight, fetal age, and perinatal mortality. *American Journal of Epidemiology* 96: 197–204.

Taffel, S. 1980. Factors associated with low birth weight: United States, 1976. *Vital and Health Statistics,* series 21, no. 37. Hyattsville, Maryland: U.S. Department of Health, Education, and Welfare.

U.S. Department of Health and Human Services. 1980. *The health consequences of smoking for women: a report of the Surgeon General.* Washington: Office of the Assistant Secretary for Health, Office of Smoking and Health.

U.S. National Center for Health Statistics. 1979. Changes in cigarette smoking and current smoking practices among adults: United States, 1978. *Advance data from Vital and Health Statistics,* no. 52. Hyattsville, Maryland: U.S. Department of Health, Education, and Welfare.

———. 1980. Selected demographic characteristics of teenage wives and mothers. *Advance data from Vital and Health Statistics,* no. 61. Hyattsville, Maryland: U.S. Department of Health and Human Services.

Valanis, B.M. 1979. Relative contribution of maternal social and biological characteristics to birth weight and gestation among mothers of different childhood socioeconomic status. *Social Biology* 26: 211–25.

van den Berg, B.J. 1968. Morbidity of low birth weight and/or preterm children compared to that of the 'mature'. *Pediatrics* 42: 590–97.

———, and Yerushalmy, J. 1966. The relationship of the rate of intrauterine growth of infants of low birth weight to mortality, morbidity, and congenital anomalities. *Journal of Pediatrics* 69: 531–97.

Werner, T. and Milton, T. 1969. Demographic correlates of low birth weight. *American Journal of Epidemiology* 91, no. 3.

Yerushalmy, J.; van den Berg, B.J.; Erhardt, C.L.; and Jacobziner, H. 1965. Birth weight and gestation as indicies of 'immaturity'. *American Journal Dis. Child* 109: 43–57.

Appendix

Table 2.A.1 **Birth Characteristics Production Function Estimates for White Population: Linear and Generalized Leontieff-Diewert.**

Independent Variable	A. Birthweight					
	(1)		(2)		(3)	
	OLS	TSLS	OLS	TSLS	OLS	TSLS
AGE	2.72	2.10	−8.01	−49.1	−56.1	−286
	(1.94)	(0.42)	(1.84)	(1.83)	(2.38)	(1.70)
DELAY	−2.52	−30.54	−31.4	−274	−24.2	−363
	(0.54)	(1.26)	(1.49)	(1.61)	(0.89)	(1.57)
SMOKING	−10.5	−17.6	5.74	13.9	6.14	6.82
	(15.6)	(4.11)	(2.42)	(0.49)	(2.52)	(0.22)
BIRTHS	24.7	44.9	−60.6	349.	−63.0	300
	(5.29)	(2.11)	(2.37)	(1.45)	(2.18)	1.06
$(AGE \cdot DELAY)^{1/2}$			17.9	367	7.18	12.4
			(1.04)	(2.82)	(0.19)	(0.03)
$(AGE \cdot SMOKE)^{1/2}$			−10.4	−2.44	−6.37	−64.1
			(2.80)	(0.06)	(0.82)	(0.88)
$(AGE \cdot BIRTHS)^{1/2}$			55.0	−2.28	78.2	176
			(3.02)	(0.02)	(1.83)	(0.54)
$(DELAY \cdot SMOKE)^{1/2}$			−6.85	14.7	−3.73	−4.95
			(0.90)	(0.16)	(0.44)	(0.05)
$(DELAY \cdot BIRTHS)^{1/2}$			16.4	−591	12.2	−441
			(0.50)	(2.10)	(0.30)	(1.23)
$(SMOKE \cdot BIRTHS)^{1/2}$			−12.3	−97.3	−14.7	−84.6
			(1.54)	(1.16)	(1.68)	(0.95)
$AGE^{1/2}$					474	2730
					(2.14)	(1.59)
$SMOKING^{1/2}$					−23.7	351
					(0.64)	(0.93)
$BIRTHS^{1/2}$					−104	−947
					(0.55)	(0.71)
$DELAY^{1/2}$					34.7	1894
					(0.18)	(1.04)
SMSA	−14.9	−13.2	−13.9	−13.3	−14.5	−8.48
	(1.02)	(0.86)	(0.96)	(0.81)	(0.99)	(0.50)
SIZE $(x10^{-8})$	172	258	191	105	178	−.038
	(0.54)	(0.77)	(0.60)	(0.29)	(0.55)	(0.00)
1967	25.3	26.7	29.3	48.6	31.4	60.3
	(1.70)	(1.71)	(1.97)	(2.52)	(2.11)	(2.67)
1968	9.39	7.57	13.8	23.4	14.8	27.3
	(0.63)	(0.49)	(0.92)	(1.18)	(0.99)	(1.33)
CONSTANT	3259	3334	3234	3067	2086	−4749
	(91.1)	(23.2)	(88.9)	(16.0)	(3.73)	(0.98)
R^2	.0386		.0468		.0475	
F		5.60		3.90		3.11

t-values in parentheses.

B. Gestation Period ($x10^2$)					
(1)		(2)		(3)	
OLS	TSLS	OLS	TSLS	OLS	TSLS
−.365	2.74	−1.86	11.4	−19.4	−88.5
(0.63)	(1.32)	(1.04)	(1.05)	(1.99)	(1.33)
−.649	.078	−28.0	24.6	−22.5	10.6
(0.34)	(0.01)	(3.22)	(0.35)	(2.00)	(0.12)
−1.03	.178	.020	−3.95	−.144	−4.73
(3.67)	(0.10)	(0.02)	(0.35)	(0.14)	(0.38)
−1.82	−9.76	2.62	121	3.56	74.6
(0.95)	(1.12)	(0.25)	(1.25)	(0.30)	(0.67)
		17.7	−.377	19.7	−138
		(2.48)	(0.01)	(1.27)	(0.96)
		−1.62	10.7	−3.81	−9.02
		(1.05)	(0.71)	(1.19)	(0.31)
		6.63	−68.9	−2.45	53.2
		(0.88)	(1.25)	(0.14)	(0.41)
		−1.77	−28.4	−2.56	−30.5
		(0.56)	(0.75)	(0.73)	(0.73)
		8.01	−51.6	1.41	17.6
		(0.59)	(0.46)	(0.08)	(0.12)
		3.71	12.4	4.64	14.2
		(1.13)	(0.37)	(1.28)	(0.40)
				175	1060
				(1.90)	(1.56)
				11.5	102
				(0.75)	(0.69)
				−15.9	−571
				(0.20)	(1.09)
				−16.7	640
				(0.21)	(0.88)
3.00	−6.05	−2.87	−8.00	−3.20	−6.01
(0.50)	(0.96)	(0.48)	(1.22)	(0.53)	(0.89)
−239	−284	−240	−304	−248	−335
(1.80)	(2.07)	(1.81)	(2.13)	(1.87)	(2.29)
51.3	50.9	52.5	46.9	53.1	50.6
(8.35)	(7.96)	(8.52)	(6.06)	(8.59)	(5.64)
8.50	8.81	9.95	5.08	10.2	6.32
(1.38)	(1.39)	(1.61)	(0.64)	(1.66)	(0.78)
3925	3861	3917	3899	3489	1099
(266)	(65.6)	(259)	(50.5)	(15.0)	(0.57)
.0127		.0144		0151	
	10.88		6.20		4.94

Table 2.A.2 **Birthweight Production Function**
Estimates for White Population: Linear and Generalized Leontief-Diewert

Independent Variable	Standardized for Gestation ($x10^2$)					
	(1)		(2)		(3)	
	OLS	TSLS	OLS	TSLS	OLS	TSLS
AGE	.0790	−.082	−.168	−2.12	−1.20	−5.62
	(1.94)	(0.55)	(1.34)	(2.62)	(1.75)	(1.11)
DELAY	.00592	−.891	.0829	−9.86	.0866	−12.7
	(0.04)	(1.26)	(0.14)	(1.91)	(0.11)	(1.83)
SMOKING	−.287	−.586	.189	.743	.202	.467
	(14.7)	(4.69)	(2.76)	(0.86)	(2.86)	(0.49)
BIRTHS	.756	1.65	−1.58	8.23	−1.68	8.52
	(5.59)	2.65	(2.14)	(1.13)	(2.00)	(1.00)
$(AGE \cdot DELAY)^{1/2}$			.0117	12.5	−.509	6.87
			(0.02)	(3.19)	(0.47)	(0.63)
$(AGE \cdot SMOKE)^{1/2}$			−.323	−.666	−.187	−2.28
			(2.99)	(0.59)	(−0.83)	(1.04)
$(AGE \cdot BIRTHS)^{1/2}$			1.71	2.51	2.31	3.26
			(3.23)	(0.61)	(1.86)	(0.33)
$(DELAY \cdot SMOKE)^{1/2}$			−.0734	2.64	.0154	1.80
			(0.33)	(0.92)	(0.06)	(0.57)
$(DELAY \cdot BIRTHS)^{1/2}$			−.107	−20.0	.0102	−17.5
			(0.11)	(2.35)	(0.01)	(1.62)

$(SMOKE \cdot BIRTHS)^{1/2}$			−.429 (1.86)	−4.62 (1.83)	−.503 (1.99)	−4.10 (1.53)
$AGE^{1/2}$					10.3 (1.61)	44.4 (0.86)
$SMOKING^{1/2}$					−.772 (0.72)	9.85 (0.87)
$BIRTHS^{1/2}$					−2.94 (0.54)	−8.18 (0.20)
$DELAY^{1/2}$					2.36 (0.43)	35.4 (0.64)
SMSA	−.361 (0.85)	−.161 (0.36)	−.334 (0.79)	−.077 (0.16)	−.344 (0.81)	−.0102 (0.02)
SIZE ($x10^{-8}$)	17.1 (1.83)	22.1 (2.26)	17.7 (1.90)	17.1 (1.59)	17.4 (1.87)	14.5 (1.31)
1967	−1.22 (2.83)	−1.13 (2.48)	−1.13 (2.62)	−.225 (0.39)	−1.08 (2.50)	−.023 (0.03)
1968	−.0702 (0.16)	−.133 (0.29)	.0153 (0.03)	.497 (0.83)	.0391 (0.09)	.567 (0.92)
CONSTANT	98.2 (94.8)	104 (24.7)	97.6 (92.6)	93.2 (16.1)	72.0 (4.44)	−44.4 (0.31)
R^2	.0375		.0455		.0460	
F		6.27		4.50		3.50

t-values are in parentheses.

Table 2.A.3 **Statistical Appendix: Level of Aggregation, Year and Source for Area Variables**

Variable Name	Aggregation Level and Year	Source
BEDS	State, 1965	*Hospital, Journal of the American Hospital Association: Guide Issue* American Hospital Association, 1965
HEXP	State or SMSA, 1965	*County and City Data Book*, Bureau of the Census 1967
HOSPFP	State, 1969	*Need for Subsidized Family Planning Services: United States, Each State and County*, Center for Family Program Development, 1969
HDFP	State or SMSA, 1969	*Need for Subsidized Family Planning Services: United States, Each State and County*, Center for Family Program Development, 1969
MD	State or SMSA, 1969	*Need for Subsidized Family Planning Services: United States, Each State and County*, Center for Family Program Development, 1969
UNEMPR-W	State, 1970	*Census of Population 1970*, U.S. Bureau of the Census, 1974
SERVICE	State, 1970	*County and City Data Book*, Bureau of the Census, 1972
CPRCE	State or Town, 1967–1969	*Tax Burden on Tobacco*, Tobacco Tax Council, Inc., Richmond, VA, 1975
TAX SALES	State or Town, 1967–1969	*Tax Burden on Tobacco*, Tobacco Tax Council, Inc., Richmond Va 1975
SIZE	SMSA, 1970	*Census of Population 1970*, U.S. Bureau of the Census, 1974