

ECONOMIC GROWTH CENTER

YALE UNIVERSITY

Box 1987, Yale Station
New Haven, Connecticut

CENTER DISCUSSION PAPER NO. 352

BIRTHWEIGHT, THE PRODUCTION OF CHILD HEALTH, AND INPUT DEMAND

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June 1980

Notes: Prepared for a conference: "Economic Aspects of Health," National Bureau of Economic Research at Palo Alto, July 30 - August 1, 1980. The research in this paper was supported in part by a grant from NIH, Center for Population Research, HD 12172. Research assistance was provided by Cynthia Arfken. Useful comments on an earlier version were received from members of the Economics Workshop at SUNY Stonybrook.

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Introduction

The characteristics of infants at birth appear to play important roles in their subsequent growth, morbidity, and survival.¹ Such characteristics--birthweight, the length of the gestation period, and the rate of intrauterine growth--are affected by parental behavior, which is modified in order to affect (favorably) birth characteristics and which may also unknowingly condition the health of the new-born.² In recognition of the importance of these birth characteristics, many studies of "health production" focus on the relationship between the behavior of the mother while pregnant and the subsequent characteristics of her new-born. Other studies examine the relationships between parental socioeconomic characteristics and/or the availability of 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 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, and 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 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 socioeconomic 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 important consequences for child health and development.

In Section 1, 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 (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 such 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 we know that a particular behavioral

activity decreases child health or well-being, a tax on such an activity may decrease child health even though it results in a decline in the activity. The implications of the production-utility demand system for a simple parameterization of the model are derived for the Cobb-Douglas case.

In Section 2, we apply the framework to a national probability sample of approximately 10,000 live legitimate births from the National Natality Followback Surveys in the years 1967-1969. Based on the sample socioeconomic information and merged geographic information on such variables as per-capita medical doctors, health expenditures, hospital beds, family planning services and other variables, we present two sets of estimates of the relationships between birthweight and gestation and the mother's cigarette consumption while pregnant, use of prenatal medical services, age at birth, and number of births. The first estimates are obtained from the Cobb-Douglas demand system. The second set of estimates are based on more general functional forms for the production function and on linear approximations to the demand equations. The sensitivity of these estimates to changes in functional form and estimation technique is shown, and estimates are obtained of the effects of male earnings, the educational attainment of the mother, and medical service variables on the birth outcome measures and on the health production inputs. Findings regarding infant health are discussed further in Section 3.

1. The Model

a. 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 the 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:

$$(1) \quad U = U(X, Y, H) .$$

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

$$(2) \quad H = F(Y, Z, \mu) \quad F_Y, F_Z, F_\mu \neq 0 ,$$

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 below, 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)

$$(3) \quad I = Xp_x + Yp_y + Zp_z$$

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 (i) health cannot be purchased directly, other goods must be bought or utilized which influence health in a way described by (2), and (ii) the family does not maximize child health, rather it 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:

$$(4) \quad U_x = \lambda P_x,$$

$$(5) \quad U_y + U_H F_y = \lambda P_y,$$

$$(6) \quad U_H F_z = \lambda P_z,$$

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 ($F_y < 0$) or subsidy ($F_y > 0$) of the Y good.

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

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

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

$$(9) \quad Z = D_z(P_x, P_y, P_z, I, \mu);$$

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

$$(10) \quad dH = F_y dY + F_z dZ + F_\mu d\mu.$$

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

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

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

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

since $d\mu/dp_i = 0$, $i = x, y, z$. Expressions (11), (12) and (13) indicate that the price effects on child health depend on the effects of changes in prices on the demand for the 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 new-born child (we test for this in Section 2). A rise in the price of cigarettes, through 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 though it does lead to a change in the consumption of the taxed good in the predicted direction, and even if we have information on the technical or biological relationships between child health and the consumption of the good or health input. We now consider what the model implies regarding 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 .

b. Population Heterogeneity and the Estimation of the Health Production Functions

We have shown that 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. Such information is also useful for guiding potential parents in efficiently attaining 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 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 unobserved factors known to the parental decision-makers but not to the researcher, 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:

$$(14) \quad \frac{dH}{dZ} = F_z + F_\mu \frac{d\mu}{dZ}$$

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

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

so that, from (14),

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

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 are blessed biologically, i.e., expect to have relatively healthy children based perhaps on observations on past births or from the birth outcomes of close kin, and/or inhabit a relatively healthy environment, will be observed to use less of the variable input Z , but to have healthier children, than parents

less well-endowed or residing 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), (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 equations; the health production function (2) is the "structural" equation.

c. Education, Information, and the Production of Child Health

In the literature utilizing the household production framework,⁵ educational attainment is usually treated as an "environmental" variable which affects the marginal products of production inputs. 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:

$$(2a) \quad H = F' (Y, Z, \mu; e)$$

$$F'_{ye}, F'_{ze} > 0$$

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, without any missing inputs in (2), education can actually alter marginal products of inputs or biological processes embedded in (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 the Y , Z and H in a way 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 taken into account.

Indeed, if households vary in their perceptions of the true parameters of the 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 requires, however, that some structure be imposed on either the relationships between perceptions and observable characteristics or on the distributional characteristics of perceptions of health technology or of "perception errors".

d. Parameterizing the Model: The Cobb-Douglas Production-Utility Demand (CDPUD) System as a Special Case.

As an illustration of the relationships between the input and health demand equations and the production of health within the household framework and as one strategy for applying the model to data, we parameterize the model by assuming that both the household utility and production functions, corresponding to (1) and (2), take on the Cobb-Douglas form:

$$(17) \quad U = AX^{\alpha_1} Y^{\alpha_2} H^{\alpha_3},$$

$$(18) \quad H = BY^{\beta_1} Z^{\beta_2} u^{\beta_3},$$

where all variables are defined as before. While the Cobb-Douglas parameterization embodies highly implausible restrictions, as will be seen, the demand equations and production relations can be explicitly solved and are linear in parameters so that the system can be readily estimated. Moreover, estimates of the complete system, if not rejected by the data, yield solutions for all the parameters which describe both the household's preference orderings and the health production technology, the α_i 's and β_i 's.

The demand equations for the three goods derived from the maximization of (17) subject to (18) and (3), and corresponding to (7), (8) and (9), are:

$$(19) \quad X = \frac{I}{P_x} \left[\frac{\delta}{\alpha_1} + \left(1 + \frac{Y}{\alpha_1}\right) \right] = \frac{I}{P_x} a_o,$$

$$(20) \quad Y = \frac{I}{P_y} \frac{Y}{\alpha_1} \left[\frac{\delta}{\alpha_1} + \left(1 + \frac{Y}{\alpha_1}\right) \right] = \frac{I}{P_y} b_o,$$

$$(21) \quad Z = \frac{I}{P_z} \frac{\delta}{\alpha_1} \left[\frac{\delta}{\alpha_1} + (1 + \frac{\gamma}{\alpha_1}) \right] = \frac{I}{P_z} c_o ,$$

where $\gamma = \alpha_2 + \alpha_3 \beta_1$, $\delta = \alpha_3 \beta_2$;

or taking logarithms:

$$(22) \quad \ln X = \ln I - \ln P_x + \ln a_o ,$$

$$(23) \quad \ln Y = \ln I - \ln P_y + \ln b_o ,$$

$$(24) \quad \ln Z = \ln I - \ln P_z + \ln c_o ,$$

from which it can be seen that:

1. The input and goods (X,Y,Z) demand equations are separable--the demand for each good depends only on its own price and on income; there are no cross-price effects as would exist in a more general model.

2. The income and (uncompensated) own price elasticities are one and minus one, respectively, for all of the choice variables: X, Y, and Z.

3. The demand for each of the goods does not depend on the heterogeneity parameter, μ , if parameterized as a multiplicative factor in the production function, as in (18). OLS estimation of the production function yields consistent estimates of β_1 and β_2 , which would not be the case for most models or treatments of heterogeneity.

4. From (18), (20), (21) the demand equation for health in logs, is:

$$(25) \quad \ln H = - \beta_1 \ln P_y - \beta_2 \ln P_z + (\beta_1 + \beta_2) \ln I + \beta_1 \ln b_o + \beta_2 \ln c_o + \beta_3 \mu .$$

Thus, in this system, estimation of the reduced-form demand equation for health yields the same information as does direct estimation of the production function--the coefficient on the ($\ln$) price of each input in (25) is identical to the negative of its output elasticity parameter in (18).

5. Estimation of the full set of demand equations and either (18) or (25) yields estimates of all (five) parameters of the system--those describing the utility function-- α_1 's--and those characterizing the technical or biological relationships between health inputs and health outcomes--the β_1 's. In this case, however, it can be easily seen that if z entered the utility as well as the production functions, identification of the utility parameters would not be possible--the prior restriction that one input enters production but does not yield utility "identifies" the utility function.

6. We can think of schooling attainment in this system as possibly affecting preference orderings through its effects on the utility function parameters, the α_1 's, and/or as affecting perceptions about the true technology, the β_1 's; namely, $\alpha_1 = f(e)$ and $\beta_1 = g(e)$, where the β_1 's are the assumed parameters. If the f and g functions are such that e appears as a linear term in each of the demand equations (22), (23), and (24), (and such functions are assumed to exist), then none of the predictions of the model nor the functional forms are affected. Regardless, estimates of the health "demand" function (25) or direct estimation of the production function (18) would yield the true β_1 's, not the perceived β_1 's. While the education variables would not enter into the estimation of the ("true") production function, they would appear in linear form in (25), if they affected the demand for (the log of) inputs linearly.

If the parameter estimates of the Cobb-Douglas Production-Utility Demand (CDPUD) System, do not conform to the predictions of this system, i.e., if output elasticities, and the negative of price elasticities for the health inputs are not equal and/or if the own price and income elasticities are not one and minus one, respectively, any one or all of the assumptions embedded in the parameterized household model described by (17) and (18) would have to be rejected. Indeed, if

the unobserved endowment parameter, μ , is additive and/or if the unobservable utility function is not Cobb-Douglas, the demand equations are non-linear in parameters and are not readily solved in closed-form. Moreover, estimation of the production function, even if Cobb-Douglas, then requires an instrumental variable or two-stage estimation procedure for consistency. Of course, the structure of the production function can be estimated directly or approximated using some generalized functional form which is linear in parameters; similarly, approximations to the demand equations for H, X, Y, and Z can be estimated. In the next section, we implement both types of estimation strategies, first estimating and testing the complete CDPUD system and then estimating linear or first-order approximations to the demand equations and testing various specifications of the health production function under different assumptions concerning heterogeneity and functional form.

2. Empirical Application

a. 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 factors or behavior influencing 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 useful for obtaining consistent estimates of the former in most cases. To apply the model thus requires information on birth outcomes reflecting infant well-being, a complete set of parental behavior and characteristics related to child health production, and the price and/or availability variables which affect such behavior. The 1967, 1968 and 1969 National Natality Followback Surveys appear to meet most of these requirements. These national probability samples of approximately 10,000 births, for the three years combined, contain information on birthweight and gestation period for each birth as well as subsequent child mortality, on the school attainment of both parents, the earnings of the husband, and three retrospectively obtained aspects of the mother's behavior which are potentially linked to infant health at birth--smoking, working and 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 the merging of local-area price and health program variables with the micro data.

We selected for analysis all non-multiple births, resulting in a sample size of 9621 births. Based on the geographical information from the data tapes, 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) with family planning, medical doctors per-capita (MD), the unemployment rate for women 15-59 (UNEMPR-W), the percent of persons employed in service industries (SERVICE), the cost (including excise taxes) of cigarettes (CPRCE), the sales tax on cigarettes (TAXSALES), and the size of the SMSA (SIZE) for inhabitants of SMSAs. The sample characteristics and definitions of all variables are listed in Table 1.

The weight of a child at birth has much to do with its prospects for survival. In 1964-65 among U.S. births weighing less than 2500 grams, 18.6 percent did not reach their first birthday, whereas the proportion dying among those weighing more than 2500 grams was .97 percent, a ratio of 19 to 1. Grouping births by parent economic characteristics yields much narrower differentials, such as a 1.86 to 1 ratio for infant mortality rates to mothers with eight years or less to those with 16 years or more of education, or a 1.67 to 1 ratio for infant mortality rate of births in families with annual incomes of under \$3000. to those in families with incomes of \$10,000 and over (MacMahon, et al., Tables 18, 21, and 22).

If low birthweight was a genetically determined predisposing factor for 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 birthweight of less than 2500 grams, is almost twice as great among mothers with 8 years or less education com-

Table 1

Variable Dictionary, Means and Standard Deviations

Variable	Definition	Mean	Standard Deviation
<u>Endogenous</u>			
Birthweight	Weight of baby at birth, in grams	3288	568
Gestation Period	Length of pregnancy, in weeks	39.1	2.45
Standardized Birth-weight	Birthweight/Predicted birthweight 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 packs of cigarettes mother smoked while pregnant	4.71	8.64
AGE	Age of mother at birth	24.9	5.61
BIRTHS	Number of previous live births born to mother	2.54	1.90
<u>Exogenous-Individual</u>			
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
<u>Exogenous-Area</u>			
BEDS	Number of hospital beds per capita	.00466	.00109
HEXP	Governmental health and hospital expenditures per capita (\$000)	.0203	.0226
HOSPFP($\times 10^{-8}$)	Number of hospitals with family planning program per capita	299.	158.
HDFF($\times 10^{-8}$)	Number of health departments with family planning per capita	95.0	199
MD($\times 10^{-3}$)	Number of persons per medical doctor	1.42	.695
UNEMPR-W	Unemployment rate of women	.0526	.0104
SERVICE	Percent of persons employed in service industries	77.9	15.3
CPRCE	Price of cigarettes including state and local excise taxes per pack (¢)	34.61	3.38
TAXSALES	Tax on retail sales (cents/package)	.583	.490
SIZE($\times 10^{-3}$)	Population of SMSA	1349.6	2087
n			9621

pared with mothers with 16 years or more, 10.6 versus 5.6 percent (Ibid., Table 18). Moreover, the proportion of all U.S. births thus classified as premature increased for no obvious reason from about ten to more than 13 percent from 1950 to 1966, and has only in the last decade begun to decline. The proportion of underweight births in the U.S. remains substantially in excess of that recorded in other industrially advanced countries and would seem to be related to the relatively high level of child mortality in the U.S. Moreover, most of the increase in the proportion of premature births in the U.S. can be attributed to the increase in the fraction of nonwhite births that are underweight (Taffel, 1980; Chase and Byrnes, 1972).

Public health investigations of low birthweight often distinguish only births below 2500 grams as "premature", which included eight percent of the live births but accounted for 62 percent of the infant deaths in the U.S. in 1964-65. (MacMahon, et al., 1972). Though deaths are concentrated among very low birthweight infants, the inverse relationship between infant mortality rates and birthweight is roughly linear from under 1000 grams to about 3000 grams. Slightly elevated mortality levels are also recorded for infants weighing more than 4500 grams, who constituted less than two percent of U.S. live births in 1960 (Chase, 1969). The analysis of birthweight as a continuous linear indicator of child health has obvious statistical advantages over a dichotomous and relatively infrequent event, such as infant mortality.⁶

A second indicator of the health of the newborn is gestational age. Births of short gestation die much more frequently during the first month of life: 79 percent of the U.S. births in early 1950 whose period of gestation was under 28 weeks died, whereas only .88 percent of those whose period of gestation was 37 weeks or more died (Shapiro, 1965, Table H). Gestational age, however, is not reported in a few states on birth certificates, and some epidemiologists suspect information on gestation reported on birth certificates is subject to greater error than that associated with registered birthweight (Eisner, et al., 1979).

Recently, two health effects of prematurity have been distinguished: a relatively transitory trauma associated with leaving the womb and establishing 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 low gestational age. The latter more permanent effect is represented by the individual's average rate of weight gain to birth, normalized by the usual weight associated with infants of the same gestational age. Since the birthweight distribution by gestational age differs notably for whites and blacks, normalization functions for gestation are fit separately for white and black births in the sample as well as combined (Chase, 1962).⁷

Three measures of birth outcomes are used in the analysis: birthweight, gestation period and a standardized measure of birthweight which controls for gestation and thus reflects uterine growth -- actual birthweight divided by predicted birthweight based on that child's gestation period. Predicted birthweight was obtained in two ways: from a third-order polynomial regression applied to the whole sample and from race-specific equations.⁸ This variable thus reflects the size of the family's infant relative to other infants born after the same gestation period; it is a relative measure of the rate of intrauterine development.

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 packs of cigarettes smoked by the mother while pregnant (SMOKING), the order of the current live birth (BIRTHS) and the age of the mother at birth (AGE). We note that 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 significant determinant of birth outcomes. In the reported specifications we consequently drop 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, 1969) and whether or not the mother is black (BLACK). The birth characteristics production function in its general form is thus:

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

Of the non-environmental or genetic variables, all but DELAY would be a "health-related good" (Y), providing direct utility to the mother irrespective of its impact on child health.

To estimate the demand for the goods potentially affecting the birth characteristics, the endogenous "inputs" in (17), corresponding to equations (7), (8), (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 areal 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 SERVICE, a female-intensive industry, we expect 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 HOSFPF, 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 HOSFPF should all be positively associated with lower costs of medical care, inducing less delay by mothers in seeking prenatal medical care and thus should be 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 (26), but allow an assessment of the relative influence of individual characteristics and the local availability of medical services on activities which affect the conditions of the child at birth.

b. Estimating the CDPUD System

To estimate and test the complete CDPUD system, we first assume that the log of the unobserved prices of AGE, DELAY, and BIRTHS, P_A , P_D , and P_B are linear functions of the areal program or price variables, such that

$$(27) \quad \ln P_A = d_{10} + d_{11} \text{HOSPFP} + d_{12} \text{HDFP} + d_{13} \text{UNEMPRW} + d_{14} \text{SERVICE} + u_1,$$

$$(28) \quad \ln P_D = d_{20} + d_{21} \text{MD} + d_{22} \text{HEXP} + d_{23} \text{BEDS} + d_{24} \text{UNEMPRW} + d_{25} \text{SERVICE} + u_2,$$

$$(29) \quad \ln P_B = d_{30} + d_{31} \text{HOSPFP} + d_{32} \text{HDFP} + d_{33} \text{UNEMPRW} + d_{34} \text{SERVICE} + u_3,$$

where the u_i are random error terms.

Then, from (22) - (24), the CDPUD demand equations for AGE, DELAY, SMOKING and BIRTHS are ($\ln = L$):

$$(30) \quad L \text{ AGE} = e_{10} + e_{11} \text{HOSPFP} + e_{12} \text{HDFP} + e_{13} \text{UNEMPRW} + e_{14} \text{SERVICE} + e_{15} \text{LHINC} + e_{16} \phi + u_1,$$

$$(31) \quad L \text{ DELAY} = e_{20} + e_{21} \text{MD} + e_{22} \text{HEXP} + e_{23} \text{BEDS} + e_{24} \text{UNEMPRW} + e_{25} \text{SERVICE} + e_{26} \text{LHINC} + e_{27} \phi + u_2,$$

$$(32) \quad L \text{ BIRTHS} = e_{30} + e_{31} \text{HOSPFP} + e_{32} \text{HDFP} + e_{33} \text{UNEMPRW} + e_{34} \text{SERVICE} + e_{35} \text{LHINC} + e_{36} \phi + u_3,$$

$$(33) \quad L \text{ SMOKING} = e_{40} + e_{41} \text{LCPRCE} + e_{42} \text{LHINC} + e_{43} \phi + u_4$$

where ϕ is a vector containing the variables, MHSI, MHSG, MCOLI, MCOLC, SMSA, SIZE, 1967, 1968 and BLACK; i.e., education is assumed to affect the log of the inputs in a non-linear way but independent of income and prices. We note that if we can accept the CDPUD system, in which the coefficients on the $\ln P_i$'s equal -1 (i.e., $e_{41} = -1$) then it is possible

to compute the d_{ij} 's, which give the proportional effects of each service or program availability variable on the unobserved prices of the health inputs, since $d_{ij} = -e_{ij}$. However, we cannot retrieve the utility function parameters because we are not estimating the demand for non-health related goods.

Finally, we specify the production function and reduced form demand equation for birthweight; corresponding to (18) and (25):

$$(34) \quad L \text{ BIRTHWEIGHT} = f_0 + f_1 \text{ LAGE} + f_2 \text{ LDELAY} + f_3 \text{ LBIRTHS} + f_4 \text{ LSMOKING} + u_5,$$

$$(35) \quad L \text{ BIRTHWEIGHT} = g_0 + g_1 \text{ LCPRCE} + g_2 \ln P_A + g_3 \ln P_D + g_4 \ln P_p + g_5 \text{ LHINC} + u_6,$$

to which are added year, SMSA and race variables. The implications of the CDPUD model are that: (1) $e_{41} = -1$; (2) $e_{15} = -e_{26} = e_{35} = e_{42} = 1$; (3) $f_4 = -g_1$; and (4) $g_5 = \sum_{i=1}^9 f_i$.⁹ These cross-equation restrictions can be tested if the set of equations (30) through (35) are estimated as a system.

An additional advantage of system estimation in the CDPUD model is that if the error terms, u_i 's, are correlated across equations, there are efficiency gains, for unlike in the general household demand model not all the regressors appear on the right hand side of each demand equation due to the separability property of the Cobb-Douglas utility function.

Table 2 reports the CDPUD system parameter estimates using three-stage least squares.¹⁰ The production function estimates in column (1) suggest that of all the input variables subject to choice by the parents, only age at birth is not statistically significant. A doubling in the time after conception before the mother seeks medical care and a doubling in the consumption of cigarettes (at the sample means) is associated with a reduction in birthweight by 7 and 3 percent, respectively. An increase in

Table 2

Three-stage Least Squares Estimates: Cobb-Douglas Production-Utility Demand System

Variable	L Birthweight (Production Function) (1)	L AGE (2)	L DELAY (3)	L SMOKING (4)	L BIRTHS (5)
LAGE	-.0283 (0.47)	-	-	-	-
LDELAY	-.0660 (2.05)	-	-	-	-
LSMOKING	-.0297 (3.21)	-	-	-	-
LBIRTHS	.0544 (2.36)	-	-	-	-
LHINC	-	.0856 (32.84)	-.0896 (11.60)	.1526 (3.79)	.1731 (34.77)
MHSI	-	-.1383 (17.72)	-.1314 (6.68)	.4537 (4.46)	-.3438 (14.67)
MHSC	-	-.0894 (12.09)	-.2861 (15.38)	-.1375 (1.43)	-.5495 (24.66)
MCOLI	-	-.0671 (7.74)	-.3631 (16.67)	-.3098 (2.75)	-.6328 (24.23)
MCOLC	-	.0094 (0.96)	-.3795 (15.44)	-.4624 (3.64)	-.6938 (23.56)
LCIGPR	-	-	-	.2583 (0.98)	-
HOSPFP	-	1445.2 (1.12)	-	-	-.12132 (3.174)
HDFP	-	-877.8 (0.77)	-	-	797.81 (0.24)
MD($\times 10^{-5}$)	-	-	.5755 (0.78)	-	-
HEXP	-	-	-.0313 (0.10)	-	-
BEDS	-	-	5.897 (1.18)	-	-
UNEMPRW	-	-.9806 (4.93)	.5942 (1.17)	-	-1.952 (3.35)
SERVICE	-	-.0005 (3.67)	-.0005 (1.47)	-	-.0020 (5.20)
BLACK	-.0927 (9.37)	.0361 (6.62)	.1431 (10.32)	-.5008 (7.03)	.3002 (18.55)
SMSA	-.0046 (0.81)	.0017 (0.36)	-.0206 (1.46)	.1475 (2.29)	-.0496 (3.05)
SIZE($\times 10^{-8}$)	.2697 (2.08)	.0526 (0.46)	.0591 (0.18)	4.562 (3.25)	-.9005 (2.62)
YR1	.0088 (1.41)	.0163 (3.32)	-.0846 (6.81)	.3197 (4.97)	.0322 (2.19)
YR2	.0064 (1.10)	-.0007 (0.14)	-.0740 (5.97)	.1720 (2.68)	-.0144 (0.98)
CONSTANT	8.164 (40.93)	2.620 (109.78)	1.903 (23.09)	-3.597 (3.69)	.0431 (2.06)

birth order from 2 to 3 increases birthweight, however, by approximately 2.5 percent. The production function estimates also suggest that for given behavior by mothers, as measured by the four inputs, the birthweight of white babies appears to exceed that of black babies by 9 percent.

The demand equation estimates suggest that early pre-natal medical care, smoking, fertility and age at birth have positive income elasticities, as implied by the CDPUD model. However, none of the point estimates, all of which are statistically significant, exceed .17, far below the predicted value of one, and the point estimate for the cigarette price elasticity is positive.¹ An F-test performed on the implied coefficient vector equalities (1) and (2) of the CDPUD system, indeed, suggests rejection of this restrictive model ($F(5,47352) = 22387$). A further test of restrictions (3) and (4) relating to the correspondence between the birthweight production and demand functions, reported in Table 3, also leads to a rejection of the model ($F(2,18939) = 5.04$). Discussion of demand equation estimates are thus postponed until the next section, where more general specifications are used.

As was indicated, rejection of the highly restricted Cobb-Douglas Production-Utility Demand Model could be due to the misspecification of the utility function, the production function and/or the specification of population heterogeneity. Conditional on the appropriateness of the Cobb-Douglas form (only) for the production of birthweight, the results in Table 3 indicate that unobserved differences in infant health do influence the mother's health-related behavior, as the OLS and TSLS estimates differ importantly, with the TSLS output elasticities exceeding greatly those estimated with OLS. Because of strong correlations across the residuals from the demand equations the use of 3SLS lowers appreciably coefficient standard errors; however, the misspecification of those equations, documented above, suggests that such system-wide estimates may not be preferable to those derived from the two-stage single-equation procedure.

Table 3

Determinants of Log Birthweight: Production and Demand Functions,
Cobb-Douglas Demand System

Estimation Technique	Production Function			Demand Function ^a	
	OLS	TOLS	3SLS ^b	OLS	SUR ^b
LAGE	.0118 (0.91)	.0066 (0.11)	-.0283 (0.47)	-	-
LDELAY	-.0014 (0.32)	-.0511 (1.58)	-.0660 (2.05)	-	-
LSMOKING	-.0115 (13.74)	-.0230 (2.46)	-.0297 (3.21)	-	-
L BIRTHS	.0214 (4.79)	.0385 (1.66)	.0544 (2.36)	-	-
LHINC	-	-	-	.0110 (3.26)	.0108 (3.21)
LCIGPR	-	-	-	.0495 (2.10)	.0461 (1.97)
BLACK	-.0890 (15.52)	-.0880 (8.87)	-.0927 (9.37)	-.0669 (11.12)	-.0669 (11.12)
SMSA	.0060 (1.17)	-.0062 (1.08)	-.0046 (0.81)	-.0163 (2.42)	-.0163 (2.42)
SIZE(x10 ⁻⁸)	.1822 (1.60)	.2262 (1.74)	.2697 (2.08)	-.0229 (0.15)	-.0265 (0.18)
YR1	.0069 (1.31)	.0078 (1.26)	.0088 (1.41)	.0068 (1.26)	.0067 (1.25)
YR2	.0065 (1.23)	.0062 (1.05)	.0064 (1.10)	.0060 (1.12)	.0060 (1.12)
CONSTANT	8.027 (198.21)	8.060 (40.22)	8.164 (40.93)	7.818 (89.79)	7.840 (90.31)
R ²	.044			.026	

^a Coefficients of other exogenous variables (see Table 2) not reported.

^b 3SLS and SUR estimates from systems with demand equations for LAGE, LDELAY, SMOKING and LPARITY. See Table 2.

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

Given the rejection of the simple CDPUD model by the data and the intractability of alternative complete parameterizations of the household production model, we pursue the alternative estimation strategy of estimating approximations to the demand equations corresponding to (7), (8) and (9) and to the health production function (2) or (26). For the demand equations, we do not impose separability, allowing 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:

$$\begin{aligned} (36) \quad & \text{AGE, DELAY, SMOKING, BIRTHS} = D(\text{MHSI, MHSC, MCOLI, MCOLC, HINC,} \\ & \text{through} \\ (39) \quad & \text{BEDS, HEXP, HOSPFP, HDFP, MD, UNEMPR-W, SERVICE, CPRCE, CPRCE2} \\ & \text{TAXSALES, SMSA, SIZE, BLACK, 1967, 1968).} \end{aligned}$$

where CPRCE2 is the square of CPRCE.

The generalized 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, as in the Cobb-Douglas specification, 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:

$$(40) \quad H = \sum_{ij} \sum \beta_{ij} y_i^{\frac{1}{2}} y_j^{\frac{1}{2}} + \sum_i \beta_i y_i^2 + \gamma_1 \text{SMSA} + \gamma_2 \text{SIZE} + \gamma_3 \text{BLACK} + \gamma_4 1967 + \gamma_5 1968 + \gamma_0 + \mu$$

where the y_i are DELAY, SMOKING, BIRTHS, AGE, $\beta_{ij} = \beta_{ji}$, $\beta_{ij} = 0$, $i \neq j$ 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.¹¹ 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 estimates of the β_{ij} parameters in (40) may be inconsistent. Two-stage least squares, utilizing estimates of the demand equations for the four behavioral variables y_i in the first stage, as well as OLS are thus used to estimate (40) in order to determine the importance of heterogeneity.¹² 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 4. 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, consistent with the CDPUD estimates, that more educated women and women in higher income families seek prenatal care earlier, whereas black mothers postpone such care by just over a half of a month more than do white mothers with similar personal and regional characteristics. Among the variables representing the availability of medical services, only residence in an SMSA and in a county with greater numbers of health department with family planning programs appears to encourage the receipt of earlier prenatal medical care. Over the sample three year period, delay in seeking prenatal care was decreasing, 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 packs of cigarettes she smokes while pregnant. Pregnant black women smoke more than two packs of cigarettes less, on average, while pregnant than do white mothers, however. Where female unemployment rates are high, mothers appear to smoke less, although, the number of doctors per capita and the availability of family planning services in the local area are positively associated with female

Table 4

Linear Input Demand Equation Estimates

Independent Variable	DELAY	SMOKING	AGE	BIRTHS
MHSI	-.561 (9.48)	1.49 (4.46)	-3.44 (17.06)	-1.06 (15.33)
MHSC	-1.08 (19.30)	-.426 (1.35)	-2.56 (13.45)	-1.68 (25.70)
MCOLI	-1.25 (18.96)	-1.01 (2.71)	-2.37 (10.57)	-1.95 (25.29)
MCOLC	-1.27 (16.87)	-1.66 (3.91)	-.873 (3.42)	-2.21 (25.19)
HINC($\times 10^{-3}$)	-.041 (9.03)	.127 (4.91)	.548 (35.28)	.122 (22.85)
BEDS	19.16 (1.14)	83.35 (0.88)	258.2 (4.53)	60.07 (3.06)
HEXP	-.296 (0.30)	2.65 (0.48)	-7.25 (2.18)	-1.92 (1.68)
HOSPFP	-3679 (0.35)	17838 (3.04)	29697 (0.84)	-39247 (3.23)
HDFP	-6781 (2.66)	28316 (1.96)	-1002 (0.12)	-5646 (1.89)
MD($\times 10^{-5}$)	-.518 (0.22)	.298 (2.27)	3.71 (0.47)	-4.00 (1.47)
UNEMPR-W	3.06 (1.73)	-22.68 (2.27)	-11.20 (1.86)	.948 (0.46)
SERVICE	.0023 (1.87)	-.012 (1.64)	-.0094 (2.21)	-.00010 (0.04)
CPRCE	.054 (0.65)	-.031 (0.07)	-.226 (0.80)	.295 (3.02)
CPRCE2	-.00083 (0.65)	.0011 (0.15)	.0040 (0.92)	-.0045 (3.00)
TAXSALES	-.042 (1.10)	-.273 (1.25)	.095 (0.72)	-.028 (0.61)
BLACK	.661 (15.77)	-2.35 (9.90)	1.16 (8.13)	1.03 (20.96)
SMIA	-.141 (2.82)	.719 (2.54)	.090 (0.53)	-.187 (3.36)
SIZE($\times 10^{-8}$)	-.205 (.205)	.150 (2.65)	2.78 (0.82)	-1.40 (1.20)
1967	-.172 (4.56)	.857 (4.01)	.686 (5.34)	.171 (3.87)
1968	-.162 (4.33)	.396 (1.86)	.222 (1.75)	.021 (0.50)
CONSTANT	2.74 (2.04)	4.20 (0.62)	26.47 (5.80)	-2.82 (1.80)
R ²	.130	.031	.170	.146
F	71.70	15.58	98.17	82.13

smoking, as is city size. The year dummy coefficients suggest a decline in cigarette consumption by pregnant women, by 18 percent from 1967 to 1969.

The AGE equation coefficients suggest a U-shaped relationship between school attainment of mothers and age at birth. While mothers with less than nine years of schooling appear to be older on average, of women with at least some high school education those with the most schooling have their children at older ages. The earnings of the husband appear to be positively associated with delay in child-bearing, while where the unemployment rates of women are high, 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 child-bearing age of mothers and the number of hospital beds per capita are positively correlated with this variable. The year dummy coefficients suggest a temporal decline in the average age of child bearing.

The BIRTHS equation results are consistent with findings obtained in many prior studies of fertility behavior--more educated women tend to have fewer births, while husband's earnings and cumulative fertility are positively correlated, and black women have on the average almost one more birth than white women prior their current pregnancy. Mothers in urban environments have lower fertility. Most interestingly, while local family planning programs do not appear to influence the timing of births i.e. affect AGE, the BIRTHS equation indicates that they are effective in reducing cumulative fertility--the coefficients of both HOSFPF and HDFF are negative and statistically significant. Health expenditures also appear to reduce total fertility, although 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 birthweight and gestation are reported in Table 5, and to birthweight standardized for gestation in the total population and within race groups in Table 6. 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 a delay in seeking prenatal care by the mother is an unimportant determinant of birthweight, while use of the more defensible TSLS procedure indicates that such a delay would lower birthweight. The results also suggest that while the more generalized functional specifications do not yield precise TSLS estimates because of the collinearity among the transformations of the input variables, the linear (Leontief) specification of the production relationship appears to mask important interactions among the designated inputs and our measures of child health.

The marginal products, F_{ij} , of the four input activities evaluated at the sample means are summarized in Table 7 for the various functional specifications of the production relationships and for the OLS and TSLS estimates. We will focus the discussion of the empirical results primarily on the TSLS estimates of the marginal productivities of inputs obtained from the generalized Leontief-Diewert functional form of the health production function. Such estimates

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TABLE 5

BIRTH CHARACTERISTICS PRODUCTION FUNCTION ESTIMATES: LINEAR AND GENERALIZED LEONTIEF-DIEWERT

Independent Variable	Birthweight						Gestation Period (x10 ²)					
	(1)		(2)		(3)		(1)		(2)		(3)	
Technique	OLS	TSLS	OLS	TSLS	OLS	TSLS	OLS	TSLS	OLS	TSLS	OLS	TSLS
AGE	3.58 (2.79)	1.83 (0.38)	-1.48 (0.64)	-32.8 (1.54)	-59.5 (2.81)	-206 (1.32)	-.677 (1.20)	1.46 (0.70)	-1.34 (1.32)	14.6 (1.54)	-28.4 (3.03)	-166 (2.28)
DELAY	-1.56 (0.42)	-39.6 (1.71)	-44.3 (3.05)	-177 (1.16)	-37.6 (1.68)	-129 (0.62)	-2.25 (1.38)	-8.23 (0.82)	-19.4 (3.03)	91.1 (1.33)	-16.9 (1.72)	233 (2.44)
SMOKING	-10.1 (15.4)	-16.2 (3.49)	5.48 (2.47)	-1.80 (0.06)	5.74 (2.54)	-9.76 (0.32)	-.784 (2.72)	1.45 (0.72)	.398 (0.41)	-14.7 (1.13)	.236 (0.24)	-19.6 (1.37)
BIRTHS	20.9 (5.34)	43.3 (2.31)	-14.7 (1.64)	128 (0.79)	-74.2 (3.18)	43.9 (0.24)	.174 (0.10)	-5.26 (0.65)	1.01 (0.25)	102 (1.42)	6.15 (0.60)	34.6 (0.41)
1/2 (AGE • DOCTOR)	-	-	27.7 (2.56)	193 (1.79)	-8.65 (0.27)	155. (0.58)	-	-	8.97 (1.88)	-53.9 (1.12)	10.49 (0.74)	31.5 (0.25)
1/2 (AGE • SMOKE)	-	-	-13.5 (4.65)	12.1 (0.34)	-9.19 (1.26)	-44.1 (0.61)	-	-	-1.30 (1.01)	19.9 (1.25)	-3.51 (1.09)	-5.17 (0.15)
1/2 (AGE • PARITY)	-	-	10.8 (1.79)	43.8 (0.41)	83.09 (2.29)	236 (0.93)	-	-	-6.03 (2.28)	-45.3 (0.96)	-5.32 (0.33)	94.6 (0.80)
1/2 (DOCTOR • SMOKE)	-	-	-3.74 (0.58)	21.2 (0.27)	-1.30 (0.17)	20.7 (0.24)	-	-	.086 (0.03)	-6.39 (0.18)	.385 (0.12)	18.2 (0.45)
1/2 (DOCTOR • PARITY)	-	-	22.5 (1.52)	-276 (1.20)	58.7 (1.79)	-213 (0.76)	-	-	16.8 (2.57)	-75.9 (0.74)	11.5 (0.80)	-125 (0.96)
(SMOKE • PARITY)	-	-	-3.66 (0.87)	-100 (1.28)	-6.99 (0.87)	-80.8 (0.98)	-	-	.458 (0.25)	-3.01 (0.09)	2.45 (0.69)	9.62 (0.25)
AGE ^{1/2}	-	-	-	-	533 (2.66)	1568 (1.00)	-	-	-	-	282 (3.19)	1497 (2.05)
DELAY ^{1/2}	-	-	-	-	86.5 (0.53)	-77.5 (0.05)	-	-	-	-	-14.1 (0.20)	-855 (1.28)
SMOKING ^{1/2}	-	-	-	-	-20.0 (0.58)	296 (0.80)	-	-	-	-	7.99 (0.52)	99.5 (0.58)
PARITY ^{1/2}	-	-	-	-	-178 (1.11)	-809 (0.72)	-	-	-	-	-17.6 (0.25)	-401 (0.77)
BLACK	-252 (16.8)	-257 (11.1)	-245 (16.3)	-234 (8.16)	-244 (16.1)	-229 (6.54)	-71.5 (10.8)	-53.6 (5.31)	-70.7 (10.7)	-62.3 (4.87)	-69.7 (10.4)	-64.1 (3.94)
SMSA	-20.8 (1.55)	-18.9 (1.31)	-21.0 (1.57)	-17.3 (1.16)	-21.8 (1.63)	-16.4 (1.08)	.996 (0.17)	-4.56 (0.72)	1.13 (0.19)	-4.93 (0.74)	.677 (0.11)	-5.08 (0.72)
SIZE(x10 ⁻⁸)	465 (1.56)	525 (1.67)	475 (1.60)	388 (1.15)	482 (1.62)	300 (0.86)	-282 (2.15)	-354 (2.59)	-283 (2.16)	-376 (2.50)	-290 (2.21)	-418 (2.56)
1967	18.1 (1.32)	19.8 (1.36)	22.4 (1.64)	31.9 (1.89)	23.7 (1.73)	30.8 (1.56)	54.5 (9.05)	52.7 (8.28)	55.0 (9.12)	44.5 (5.90)	56.2 (9.31)	42.8 (4.65)
1968	14.8 (1.08)	14.2 (0.99)	19.6 (1.43)	25.2 (1.53)	19.9 (1.45)	25.7 (1.48)	11.1 (1.85)	11.1 (1.78)	12.0 (1.99)	3.15 (0.43)	12.8 (2.12)	3.69 (0.46)
CONSTANT	3263 (95.0)	3360 (24.0)	3267 (91.9)	3190 (18.1)	1943 (3.84)	-205 (0.05)	3927 (261)	3896 (63.9)	3922 (250)	3969 (50.3)	3222 (14.4)	1153 (0.58)
R ²	.053	-	.061	-	.062	-	.024	-	.025	-	.027	-
F	-	29.46 (10,9611)	-	17.53 (16,9605)	-	13.82 (20,960)	-	24.94 (10,9611)	-	14.21 (16,9605)	-	10.76 (20,9601)

STANDARDIZED BIRTHWEIGHT PRODUCTION FUNCTION ESTIMATES: LINEAR AND GENERALIZED LEONTIEF-DIEWERT

Independent Variable	Total Population Standardization ($\times 10^2$)						Race-Specific Standardization ($\times 10^2$)					
	(1)		(2)		(3)		(1)		(2)		(3)	
Technique	OLS	TSLs	OLS	TSLs	OLS	TSLs	OLS	TSLs	OLS	TSLs	OLS	TSLs
AGE	.112 (3.04)	-.0571 (0.39)	-.017 (0.25)	-1.68 (2.57)	-.802 (1.26)	.315 (0.07)	.115 (3.00)	-.078 (0.40)	-.148 (1.33)	-1.62 (2.50)	-.982 (1.54)	.275 (0.06)
DELAY	-.0623 (0.56)	-1.03 (1.48)	-.795 (1.83)	-7.99 (1.70)	-.803 (1.20)	-12.1 (1.89)	.101 (0.88)	-1.04 (1.50)	-.592 (1.13)	-7.96 (1.70)	-.705 (1.06)	-11.4 (1.81)
SMOKING	-.275 (14.1)	-.563 (4.03)	.177 (2.67)	.766 (0.85)	.194 (2.87)	.793 (0.84)	-.275 (14.1)	-.562 (4.04)	.188 (2.83)	.709 (0.80)	.202 (2.98)	.705 (0.75)
BIRTHS	.600 (5.12)	1.57 (2.80)	-.398 (1.48)	.518 (0.10)	-2.33 (3.34)	1.45 (0.25)	.592 (5.03)	1.57 (2.80)	-1.94 (3.16)	.700 (0.14)	-2.20 (3.14)	1.44 (0.26)
AGE ^{1/2} (AGE · DOCTOR)	-	-	.637 (1.97)	7.80 (2.40)	-.611 (0.64)	3.54 (0.43)	-	-	.233 (0.54)	7.62 (2.31)	-.424 (0.44)	4.33 (0.53)
AGE ^{1/2} (AGE · SMOKE)	-	-	-.409 (4.70)	-.395 (0.36)	-.144 (0.66)	-.529 (0.24)	-	-	-.384 (3.71)	-.343 (0.31)	-.196 (0.90)	-.647 (0.29)
AGE ^{1/2} (AGE · PARITY)	-	-	.467 (2.61)	3.81 (1.17)	2.62 (2.42)	2.12 (0.27)	-	-	1.53 (3.39)	3.55 (1.10)	2.45 (2.26)	2.12 (0.27)
(DOCTOR · SMOKE) ^{1/2}	-	-	-.0773 (0.40)	1.73 (0.72)	.0200 (0.09)	.970 (0.36)	-	-	-.0853 (0.42)	1.70 (0.72)	.0226 (0.10)	.933 (0.35)
(DOCTOR · PARITY)	-	-	.102 (0.23)	-8.05 (1.14)	1.47 (1.50)	-5.68 (0.66)	-	-	.905 (1.17)	-7.62 (1.09)	1.33 (1.35)	-5.44 (0.64)
(SMOKE · PARITY) ^{1/2}	-	-	-.0818 (0.65)	-4.57 (1.90)	-.249 (1.04)	-4.62 (1.81)	-	-	-.162 (0.74)	-4.53 (1.89)	-.252 (1.05)	-4.50 (1.78)
AGE ^{1/2}	-	-	-	-	6.16 (1.03)	-10.7 (0.22)	-	-	-	-	8.00 (1.33)	-11.5 (0.24)
DELAY ^{1/2}	-	-	-	-	3.86 (0.80)	32.3 (0.73)	-	-	-	-	2.91 (0.60)	25.6 (0.58)
SMOKING ^{1/2}	-	-	-	-	-1.26 (1.22)	1.56 (0.14)	-	-	-	-	-1.04 (1.00)	2.51 (0.22)
PARITY ^{1/2}	-	-	-	-	-5.16 (1.07)	1.69 (0.05)	-	-	-	-	-4.57 (0.94)	1.17 (0.03)
BLACK	-5.16 (11.5)	-5.97 (8.55)	-4.95 (11.0)	-4.68 (5.32)	-4.95 (10.9)	-4.69 (4.32)	1.81 (2.89)	1.94 (2.80)	-1.12 (2.47)	.686 (0.78)	-1.08 (2.38)	-.633 (0.59)
SMSA	-.815 (2.02)	-.522 (1.20)	-.821 (2.05)	-.458 (1.00)	-.828 (2.06)	-.416 (0.88)	-.718 (1.77)	-.505 (1.17)	-.723 (1.79)	-.441 (0.97)	-.733 (1.81)	-.409 (0.88)
SIZE ($\times 10^{-8}$)	30.9 (3.48)	35.8 (3.79)	31.3 (3.53)	30.7 (2.97)	31.8 (3.59)	30.8 (2.83)	30.4 (3.42)	35.5 (3.77)	31.1 (3.51)	30.4 (2.96)	31.2 (3.52)	30.1 (2.79)
1967	-1.64 (4.01)	-1.52 (3.46)	-1.52 (3.71)	-.820 (1.58)	-1.51 (3.70)	-.699 (1.14)	-1.53 (3.73)	-1.48 (3.38)	-1.43 (3.50)	-.806 (1.57)	-1.40 (3.40)	-.731 (1.20)
1968	-.073 (0.18)	-.094 (0.22)	.052 (0.13)	.529 (1.05)	.036 (0.09)	.569 (1.06)	-.034 (0.08)	-.058 (0.14)	.062 (0.15)	.544 (1.08)	.081 (0.20)	.565 (1.06)
CONSTANT	99.1 (97.0)	.105 (24.8)	99.2 (93.3)	95.8 (17.7)	838 (5.54)	94.9 (0.72)	97.5 (99.7)	104 (24.7)	97.0 (97.2)	95.4 (17.7)	78.4 (5.17)	102 (0.78)
R ²	.0398	-	.0468	-	.047	-	.0313	-	.0386	-	.0389	-
F	-	16.88	-	10.39	-	8.04	-	7.06	-	4.91	-	3.83

Table 7

Estimates of Marginal Products of Birth Characteristics Inputs at
Sample Means for Alternative Specifications and Estimation Techniques

Dependent Variable Model Specification and Estimation techniques	Marginal Product (F_1)			
	AGE (years)	DELAY (months)	SMOKING (packs/ pregnancy)	BIRTHS (number)
<u>BIRTHWEIGHT (grams)</u>				
Cobb-Douglas System				
OLS	1.56	-1.68	-8.03	27.7
TSLs	0.87	-61.3	-16.1	49.8
3SLS	3.73	-79.2	-20.7	70.4
Leontief				
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
Generalized Leontief-Diewert				
OLS	3.58	8.32	-12.50	25.8
TSLs	4.48	-7.54	-13.95	-6.35
<u>GESTATION (weeks $\times 10^2$)</u>				
Leontief				
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
Generalized Leontief-Diewert				
OLS	-.0662	0.424	-0.914	-0.484
TSLs	2.85	-25.8	7.88	-1.63
<u>RACE-SPECIFIC STANDARDIZED BIRTHWEIGHT ($\times 10^2$)</u>				
Leontief				
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
Generalized Leontief-Diewert				
OLS	.0946	.188	-.347	.721
TSLs	.0405	.856	-.753	-.769

Source: Derived from Tables 1, 5 and 6.

indicate that delay in seeking prenatal care appears to reduce both birthweight and gestation, having evidently little residual effect on birthweight standardized for gestation--our proxy for the more permanent health consequences of prematurity. (Table 7). A delay of six months in obtaining prenatal care is estimated to reduce birthweight by 45 grams or about one percent, and to reduce gestation by 1.6 weeks or four percent, with a negligible relative effect on standardized birthweight. Smoking while pregnant, on the other hand, reduces notably birthweight but is linked to longer gestational age. Smoking is related, therefore, to markedly lower birthweight standardized for gestation. Consuming an additional five packages of cigarettes during a pregnancy (approximately one-tenth of a pack more per week of pregnancy) is associated with a birthweight loss of 140 grams, or four percent, and a larger five percent reduction in standardized birthweight.

The effects of age and fertility of the mother appear to be nonlinearly related to birthweight in other studies using quite different analytical techniques (Eisner, et al., 1969), and thus the average effect of these variables may obscure their true effects. At the means of the sample, the effect of age is to increase slightly birthweight and to increase more strongly the gestation age of the newborn. No substantial average effect of age is noted on standardized birthweight. The number of births the mother has had decreases slightly birthweight, but may add to the period of gestation, with a consequent negative effect on standardized birthweight. Births to very young mothers, however, 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 birthweight is standardized for gestation age. The deleterious effects of smoking on

birthweight and birthweight standardized for gestational age are, however, increased for older mothers as well as for mothers having more births. The positive birthweight 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 birthweight 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 is a two-thirds of a week difference in average gestation period between black and white mothers. This is also evident for differences in birthweight by race--even after taking into account differences in fertility, age, smoking, and the timing of prenatal care. There is a statistically significant 229 gram differential in birthweight between the babies of black and white mothers, and about a five percent difference in the total population rate of uterine growth (standardized birthweight). The difference in birthweight 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 birthweight 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 birthweight standards are employed, the black-white difference disappears in the TSLS estimates of the Leontief-Diewert Production function.

d. Birth Characteristics, Socioeconomic Variables and Health Programs:

Linear Reduced Forms

The estimates of the reduced-form equations relating the socioeconomic variables and variables representing the availability of health services and programs to the three birth characteristics are presented in Table 8. These estimates

Table 8

Birth Characteristics Reduced Form Demand Equations

Independent Variable	Birthweight	Total Standardized Birthweight ($\times 10^2$)	Gestation Period
MHSI	-60.28 (2.71)	-2.15 (3.27)	.104 (1.09)
MHSC	-16.62 (0.80)	-1.01 (1.63)	.175 (1.94)
MCOLI	-11.58 (0.47)	-1.11 (1.52)	.231 (2.18)
MCOLC	-12.85 (0.46)	-1.26 (1.51)	.253 (2.09)
HINC($\times 10^{-2}$)	5.45 (3.20)	.1410 (2.78)	.0010 (0.14)
BEDS	10590 (1.78)	358.3 (1.93)	-18.08 (0.67)
HEXP	427.86 (1.17)	8.60 (0.79)	1.72 (1.09)
HOSPFP	-125758 (3.25)	-243276 (2.11)	-24882 (1.49)
HDFP	-27338 (0.03)	-16314 (0.58)	663.1 (0.16)
MD($\times 10^{-5}$)	-1063.7 (1.23)	31.76 (1.23)	1.83 (0.49)
UNEMPR-W	2105.1 (3.20)	96.77 (4.94)	-2.93 (1.03)
SERVICE	-1.62 (3.48)	-.048 (3.46)	.001 (0.60)
CPRCE	38.16 (1.23)	1.51 (1.64)	-.069 (0.52)
CPRCE2	-.518 (1.09)	-.022 (1.60)	.0014 (0.66)
TAXSALES	-5.51 (0.38)	.685 (1.61)	-.247 (4.00)
BLACK	-184.98 (11.83)	-3.35 (7.20)	-.642 (9.52)
SMSA	-50.62 (2.71)	-1.49 (2.68)	-.093 (1.16)
SIZE($\times 10^{-8}$)	-76.70 (0.21)	16.53 (1.49)	-4.05 (2.52)
1967	20.79 (1.48)	-1.59 (3.79)	.547 (9.02)
1968	14.98 (1.07)	-.124 (0.30)	.063 (2.09)
Constant	2627.6 (5.25)	74.00 (4.97)	40.21 (18.64)
R ²	.030	.021	.027
F	14.84	10.38	13.09

contain several puzzles. For example, the relationship between the schooling level of the mother and birthweight is U-shaped; mothers with only some high school education have babies with the lowest birthweight, 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 a high school education. Family planning programs associated with hospitals (HOSPFP) appear to reduce birthweight significantly, as does residing in an SMSA. In contrast, husband's income (HINC) and the unemployment of women are positively associated with birthweight. The estimates of the behavioral and technical relationships of Tables 4, 5, and 6 and the computed sample-mean marginal products, however, should help account for these findings, as the reduced-form birth characteristics equations reflect, as shown by equations (11) through (13), the marginal products of the health-related activities as well as the effects of the socioeconomic and program variables on their levels.

The estimates of the production functions presented in Tables 5 and 6 indicated that of the activities considered, delay in seeking prenatal medical care, smoking during the 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, birthweight appeared to be most sensitive to levels of smoking--Table 7 indicates that while a delay in seeking medical care of 6 months would lower birthweight by 45 grams, an increase of only one pack of cigarettes per week of pregnancy lowers birthweight by 546 grams. The estimates of Table 4 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 thus suggest that the non-linear effects of education on birthweight are the result of the differential effects of schooling on the several input activities. For example, women with only some high school education (MHSI) appeared to have the second highest fertility of all the educational groups and also appeared to smoke more than all other women when pregnant 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 8 that this educational group has the lowest birthweight children. The birthweight of women with higher schooling levels, however, appears to differ only trivially from that of women with less than a high school education, mainly because of only small differences in smoking habits between these groups.

Because of the important impact on birthweight 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 birthweight. These effects are evidently offset only negligibly by the tendency of mothers with higher-income husbands to consume somewhat more cigarettes while pregnant and to have higher fertility.

The negative effect (marginal product) of birth order on birthweight does not appear to be consistent with the significant negative effect of family planning programs on birthweight--such programs, which appear to be successful in reducing family size, should increase, through the BIRTHS effect, average birthweight levels. However, while BIRTHS appears to be lower in urban setting, such an environment appears to be associated with significantly greater cigarette consumption by expectant mothers so that

because of the evident importance of this activity, birthweight 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 birthweight between the black and white children, while such behavioral differences do account for more of the difference in gestation by race. Differences in input activities affecting newborn health between black and white women appear to have an ambiguous net impact on birthweight 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, the black-white birthweight differential, controlling for socioeconomic characteristics and variables representing the availability of health services and programs at the county level remains 185 grams, and that differential controlling for the health-related input behavior of the mother appears of a similar magnitude, namely 220 grams, suggesting that the net impact of black-white differences in measured behavior does not account for the noted birthweight differential between these racial groups. Standardizing birthweight for period of gestation, moreover, reduces the black-white differential only modestly. Black births weigh six percent less than those of whites, given gestation age and the measured health input behavior of the mother. But if the relationship between birthweight and gestation age is separately fit for white and black births in the sample, the estimates of a race-specific standardized birthweight production function exhibits no statistically significant black-white differences.

3. 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 size of children at birth and 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 heterogeneous) with respect to factors affecting health which are known to them 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 progress, considered four endogenous health-related inputs -- smoking while pregnant, timing of prenatal medical care, age at birth and birth order -- and, initially, two dimensions of prematurity at birth -- birthweight and gestation.

The data rejected a full parameterization of the model based on combined Cobb-Douglas forms for the household utility function and production technology. The Cobb-Douglas Production-Utility Demand system provides readily estimable input demand and production functions and permits identification of both the underlying utility and production parameters. The rejection of this system, therefore, necessitated, in the absence of alternative household production parameterizations with such nice properties, estimating approximations to the unknown component equations of the system describing input demand and the infant health production

technology.

Experimentation with functional forms for the birth outcome production function indicated that estimates of the impact of household activities on the characteristics of children at birth are more sensitive to whether or not heterogeneity is taken into account in estimation than to the choice of functional form. In particular, heterogeneity appeared to almost completely mask a negative impact on child health of delay by mothers in seeking medical care. Estimates of the production functions also indicated that smoking by the mother while pregnant had the largest negative impact of all the inputs considered on birthweight and on the rate of foetal growth, 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 birthweight than of gestation, suggesting that variation in gestation may be less affected than birthweight 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 gestational age 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. Widely noted, but infrequently analyzed, differences in the distribution of birthweight by gestation between white and black births led us to also perform this standardization procedure within our sample for black

and white births separately, and then combine these standardized birth-weight values in estimating the total sample child health production function. In some instances we found that the effects of an input on birthweight and gestation cancel in terms of their permanent effects on standardized birthweight, as is 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.

Our treatment of the heterogeneity problem made use of information on local market prices and health programs to both estimate input demand equations and to identify the child health production functions. We think it unlikely that the demand for inputs would be independent of latent population characteristics known to households and affecting child health production, as is consistent with our estimates, and, therefore, conclude that the treatment of heterogeneity is crucial for deriving sound causal conclusions that might be useful for policy analysis. However, 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 values of μ . Alternatively, individuals may themselves migrate to regions according to which region has lower prices for preferred inputs and/or available programs; i.e., they are drawn according to their μ 's. In this case, the source of identifying information required to estimate the child health production function

may prove difficult to obtain and consequently estimates of input productivities and price and program effects using such information should be interpreted with caution.

FOOTNOTES

1. See, for examples, 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 (Hebel 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 examples, Michael (1973) and Grossman (1972).

6. Subsequent research by the authors is planned to explore nonlinear transformations of birthweight and mortality outcomes as well.

7. Infant mortality rates in the U.S. in 1960 were 41.4 per thousand nonwhite births and 22.2 per thousand white births. Also, 12.9 percent of nonwhite births weighed less than 2500 grams, whereas only 6.8 percent 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 birthweights than do whites, given similar health inputs. Differences between races in pelvic structures might rationalize such a difference in birthweight. 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 standardizations of birthweight 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.

8. The estimates are:

$$\begin{aligned} \text{total sample: birthweight} &= 10107 - 1042 \text{ weeks} + 37.8 \text{ weeks}^2 - .398 \text{ weeks}^2 \\ &\quad (7.72) \quad (9.95) \quad (10.44) \\ R^2 &= .227, \bar{n} = 9763 \end{aligned}$$

$$\begin{aligned} \text{whites: birthweight} &= 13416 - 1354 \text{ weeks} + 47.3 \text{ weeks}^2 - .492 \text{ weeks}^3 \\ &\quad (8.48) \quad (9.13) \quad (10.4) \quad (10.9) \\ R^2 &= .201, n = 7896 \end{aligned}$$

$$\begin{aligned} \text{blacks: birthweight} &= 9188 - 937 \text{ weeks} + 34.4 \text{ weeks}^2 - .366 \text{ weeks}^3 \\ &\quad (4.39) \quad (4.57) \quad (5.26) \quad (5.39) \\ R^2 &= .273, n = 1867 \end{aligned}$$

9. LDELAY is assumed to affect health negatively and thus, through its health effect, is a "bad" in the system, with a negative income elasticity. The production function estimates reported below confirm this assumption when heterogeneity is taken into account.

10. Because the SMOKING variable had a large number of observations with 0's, we arbitrarily assigned a value of .05 to non-smokers, perhaps reflecting environmental smoking levels. Households in which the husband reported no earnings during the year prior to the survey were also excluded, slightly reducing the sample size from 9621 to 9484. For all samples we set DELAY equal to the sample mean gestation period (39 weeks) if no pre-natal medical care was sought and to 4 weeks if "immediate" care was received upon learning of the pregnancy. Preliminary investigations based on the linear specifications reported in the next section suggested that, for those specifications, results were not sensitive to these sample alterations.

11. 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:

$$\sigma_{ij} = [-F_{ii}/F_i^2 + 2(F_{ij}/F_i F_j) - F_{jj}/F_j^2][(y_i F_i)^{-1} + (y_j F_j)^{-1}]^{-1}$$

12. An alternative estimation 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. Such a technique would require longitudinal data or good retrospective information on prior births to implement and requires 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 characteristic of most "fixed effects" models.

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