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BIRTH OUTCOME PRODUCTION FUNCTIONS IN THE U.S.: A STRUCTURAL MODEL

by

THEODORE JAY JOYCE

A dissertation submitted to the Graduate Faculty in Economics in partial fulfillment of the requirements for the degree of Doctor of Philosophy, The City Univeaity of New York

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Abstract

BIRTH OUTCOME PRODUCTION FUNCTIONS IN THE U.S.: A STRUCTURAL MODEL

by

Theodore Jay Joyce

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This dissertation examines the determinants of the race-specific neonatal mortality rate and the percentage of low-birth weight births across large counties in the U.S. in 1977. Applying the framework from household production theory, this study emphasizes the use of abortion, prenatal care, family planning clinics, and neonatal intensive care as endogenous inputs in the production of infant health.

Direct correlational estimates between a set of health inputs and a health outcome are potentially suspect due to the importance of an unobserved genetic component in the determination of health. The results from this study indicate that ordinary least squares (OLS) underestimates the impact of the aforementioned health inputs on neonatal mortality when no attempt is made to control for the effect of the parents' health endowment. However, when the percentage of low-birth weight births is held constant, a proxy for the health endowment, tests show that OLS is appropriate in some specifications.

This study also underscores the importance of abortion and neonatal intensive care in explaining variations in the neonatal mortality rate across counties in the U.S. The former input operates primarily by lowering the percentage of high-risk births, in particular births to teenage and unwed mothers. In the case of whites, however, abortion also has a negative, risk-specific effect on neonatal mortality.

Using the number of inpatient days per birth in a Level II or Level III neonatal intensive care unit as a measure of this technology, this study confirms the importance of neonatal intensive care in explaining variations in the neonatal mortality rate. These results should not be interpreted as support for a national health policy that would increase the availability of such technology at the expense of other programs designed to the lower the incidence of low-birth weight. To the contrary, these results suggest that a policy aimed at preventing the proportion of births to unwed teenagers could have a significant impact on the percentage of low-birth weight births resulting in a lower neonatal mortality rate.

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INTRODUCTION

The purpose of this study is to examine the determinants of neonatal mortality across large counties in the U.S. in 1977. This analysis combines recent theoretical advances in health economics with a unique data set that measures cross-sectional variations in the availability and utilization of medical technology as well as numerous government programs specifically designed to improve birth outcomes.

Household production theory, as outlined principally by Becker (1965), has been applied to a wide variety of behavior that traditionally has been beyond the purview of economists. Some applications have been received better than others. In the area of health, however, the notion that medical care is an input into the produciton of a utility-augmenting commodity called health (Grossman, 1972) has been widely accepted and fruitfully applied.

Recently however, Rosenzweig and Schultz (1982,1983a,1983b) have argued that the concept of a health production function, as developed by Grossman, has not been properly exploited to differentiate between parental decisions that have a biological effect (the consumption of medical care) and resource constraints that condition those decisions. For example, due to the paucity of data on health inputs, most researchers have regressed health outcomes on medical care utilization and resource measures. The resulting estimates yielded what has been labeled a "hybrid" production function because it combines health inputs with price and income variables which determine their use. Consequently, as demonstrated by Rosenzweig and Schultz (1983b), the coefficient of medical care is a biased estimate of its true impact on health.

This dissertation incoprates the conceptual framework advanced by Rosenzweig and Schultz (1982,1983a,1983b) to estimate race-specific infant health production functions. More specifically, the race-specific neonatal mortatity rate and the race-specific percentage of low-birth weight births are used as indicators of infant health. The use of prenatal care, family planning clinics, abortion, neonatal intensive care and per capita cigarette consumption are the basic set of inputs. Endogenous risk factors such as the race-specific percentage of births to teenagers, illegitimate births, fifth and higher order births and premature births are also integrated into the above framework.

By controlling for various risk factors, the channels through which the basic inputs operate can be traced out. For example, the increased use of abortion and family planning may lower the infant mortality rate within a county by reducing the fraction of births to adolescent mothers. Another possible channel is that relatively high utilization of abortion and family planning services may reduce the proportion of unplanned or unwanted prenancies. This in turn may increase the resources devoted to births which are not averted and thereby lower the probability of an unfavorable birth outcome.

A novel aspect of this research is the treatment of abortion and family planning use as choice variables along with the more traditional endogenous inputs in a structural model of birth outcomes. Although Grossman and Jacobowitz (1981), Corman and Grossman (1984), and Corman, Joyce, and Grossman (1984) have examined the relationship between abortion and family planning and neonatal mortality, only the latter employed these inputs in a structural equation for health. In that particular case, however, abortion and family planning were used as exogenous determinants of birth outcomes.

This assumption is certainly problematic. Abortion rates are highest among women whose observed genetic birth endowment is on average the weakest (adolescents and women over 40 years old). Furthermore, advances in prenatal diagnosis enhance the information individuals have concerning their genetic stock. Rosenzweig and Schultz (1982) argue forcefully that actions taken as a result of this information create a correlation between the inputs and the residuals causing biased and inconsistent estimates. One objective of this study is to test for, and if necessary, correct this potential source of bias.

Since 1964 the trend in the infant mortality rate in the U.S. has been dominated by the trend in neonatal mortality. Thus, efforts to understand the causes of infant mortality must pay particular attention to the determinants of neonatal mortality. As cited above, there is growing evidence that the increased use of abortion has had a major impact on the decline of neonatal mortality. Other researchers have pointed to the tremendous advances in the management of newborn care as the primary factor in the increased survival rates of high-risk births (McCormick, 1985). With data on both these factors and a well-developed theoretical framework, this study can offer policy-makers important insights into the most significant determinants of infant health in the United States.

CHAPTER I--Theoretical Review

The following chapter reviews the theoretical literature on economic models of birth outcomes. This survey is restricted to static models with no uncertainty. Parents are assumed to make all their decisions concerning the number and health of their offspring based on limited but accurate information on the probability of a successful birth. To be sure, sequential models where parents can alter their behavior after each birth have also been developed, but given the data set to be used in this study only static models can be empirically implemented.

The early models in this area were generally more concerned with fertility patterns than with infant health. For example, to account for the observed inverse relationship between income and fertility a framework was developed to explain the quality as well as the quantity of offspring. Quality represented the resources devoted to each child and was easily adapted by health economists to characterize birth outcomes. Parents who invested more of their energy and income during pregnancy to insure a favorable birth outcome had a higher probability of delivering a healthy infant. The most recent models of birth outcomes have integrated the quality/quantity interaction with household production theory while allowing for joint production.

This review attempts to show that although the most current models are more general than earlier ones, a number of issues are relevant to all. Therefore discussions of the models are organized around answers to the following questions: What is the effect of differences in the genetic component of a mother's health with respect to the consumption of medical care and its subsequent impact on infant health? If a woman has information concerning her genetic health endowment, how does this alter her behavior and hence the outcome of a birth? What is the relationship between the biological mechanisms which affect the outcome of a birth and parental choices? Can the model explain why wealthier families have fewer but healthier children than less wealthy families? Do parents with greater education use health inputs more efficiently than their less educated couterparts, or does schooling alter a parents' choice of inputs?

The simplest economic model of infant mortality was developed by Welch and Ben-Porath (1972) orginally to explain the sex compositon of a family's offspring. Later, Ben-Porath (1974) noted that the model could be generalized to incorporate infant mortality; however, it was Williams (1976) and Lewit (1977) who adapted the original framework by Welch and Ben-Porath to explicitly model mortality. According to Williams, parents maximize a utility function

$$U = U(c,x) = U(ns,x) \qquad (1)$$

where c=ns, and c is child services or the number of surviving children, n is the number of pregnancies, and s is the probability of survival. x is the numeraire and its price is set equal to one. The budget constraint is

$$Y = p_n n + x \qquad (2)$$

where Y is income and p_n is the price of a pregnancy. Parents choose the appropriate number of pregnancies so as to attain the demanded family size based on a survival rate beyond their control and known with certainty. Solving the budget constraint (2) in terms of x and substituting into the utility function (1), and then maximizing the resulting objective function with respect to n yields the following equilibrium condition:

$$U_c/U_x = p_n/s \tag{3}$$

According to (3) an increase in the survival rate (s) relative to the price of a pregnancy will increase the demand for child services; yet an increase in the demand for child services will not necessarily increase the demand for pregnancies. In fact, in a developed country the opposite is most likely to occur. Williams shows that using the relationships $c=ns$ and $p_n n=p_c c$ the demand for pregnancies as a function of the survival rate can be written as

$$e_{ns} = -(e_{cp} + 1) \tag{4}$$

where e_{ns} is the elasticity of pregnancies with respect to survival and e_{cp} is the price elasticity of demand for child services. If e_{cp} = 0 then any increase in the survival rate will be exactly offset by a decline in pregnancies. Moreover, as long as e_{cp} is inelastic, an increase in the survival rate will bring about a reduction in the number of pregnancies. Although the demand for survivors increases in this case, it is more than offset by the lower quantity of births needed to obtain the desired number of survivors.

By allowing parents only one response to changes in infant mortality (i.e. an increase or decrease in pregnancies) the model is unsatisfactory for the study at hand. Nevertheless, the model underscores the important demographic regularity that high birth rates are positively correlated with high rates of infant mortality across geographic units and at a moment in time. Hence, cross-sectional analysis of birth outcomes should incorporate this empirical relationship.

Model I can easily be altered and made more useful by endogenizing the survival rate. As before, Ben-Porath (1973) laid down the original framework, but Williams (1976) and especially Lewit (1977,1983) expanded the model. Let parents maximize a utility function identical to equation (1) in model I

$$U = U(c,x) = U(ns,x) \tag{5}$$

where $s = s(m,r)$ and dU/dn, dU/ds, dU/dx, ds/dm, ds/dr are all positive. m represents expenditures per pregnancy for such items as prenatal care, nutritional supplements (vitamins and special foods), extra clothing, hospital care, etc. r is an exogenously determined factor which varies across individuals but which is beyond parental control. Lewit terms r "reproductive efficiency" and states that it is not known by parents with certainty. According to Lewit r can be "loosely" regarded as a "set of biological factors."

Substituting (5) into (4) yields a utility function with three choice variables n, m, and x.

$$U = U[ns(m,r),x] \tag{6}$$

By allowing survival to be partially determined by parental decisions, a health technology has been introduced. The framework has moved closer to Grossman's consumption model for health, albeit in a simplified and static form. Still, an important difference remains.

Survival, the indicator of child health, does not enter the utility function separately. Rather it enters in a restrictive, multiplicative manner. Consequently, to obtain certain results, the budget constraint must be constructed in a plausible, but ad hoc form as will be demonstrated below.

Ben-Porath (1973) and Williams (1976) use equation (6) to focus on the effects of expenditures (m) and the genetic factor (r) on fertility (n). The budget contraint assumes the simplest possible form.

$$y = nm + x \qquad (7)$$

Solving for x and substituting into the utility function (6) and maximizing the objective function with respect to n and m yields the following equilibrium condition:

$$s/s_m = m \qquad (8)$$

where s_m is the marginal product of survival with respect to expenditures per pregnancy. From (8) it is apparent that expenditures per pregnancy are independent of the number of pregnancies. However, an exogenous improvement in reproductive efficiency, r, will increase the survival rate, s, and thereby lower the price of a surviving child since less must be expended per pregnancy to achieve the same probability of survival. If we assume that the price elasticity of child services (e_{cp}) is inelastic, then the ultimate effect on the elasticity of fertility with respect to reproductive efficiency (e_{nr}), depends on the change in pregnancy expenditures with respect to r (e_{mr}).

If parents respond to an exogenous increase in the survival probability by expending more per pregnancy (e_{mr}>0), then the cost per pregnancy will increase insuring that e_{nr} will be negative. However, if the parents' response to a favorable shift in the genetic birth endowment is to lower expenditures per pregnancy, then the sign of e_{nr} is indeterminate. Ultimately, the parents' response depends on the shape of the survival production function. Totally differentiating (8) and solving for dm/dr yields:

$$dm/dr = (s_r s_m - s s_{mr})/s_{mm} s \qquad (9)$$

where s_r is the marginal product of survival with respect to reproductive efficiency; s_{mr} is the change in the marginal product of pregnancy expenditures with respect to r; and s_{mm} is the change in the marginal product of pregnancy expenditures with respect to m. Given s_{mm} is negative by the second order conditions, then the sign of dm/dr rests critically on whether changes in the health endowment have an anti-complementary effect on pregnancy expenditures.

The reason this issue has been seemingly belabored is because the interaction between the endogenous inputs and the exogenous health component plays a major role in all the models to be discussed. If individuals have some knowledge or expectation concerning their endowed health, then they may alter their demand for medical care or other health inputs based on this information. This generates a correlation between the observed inputs and the unobserved genetic component. Consequently, direct associations between the inputs and the health outcome will be biased unless this correlation is purged.

A major limitation of the Ben-Porath model used by Williams is the assumption that expenditures per pregnancy are independent of income. Even if one assumes that survivors (ns) are a normal good, the only way wealthier families can increase their family size is by additional pregnancies. Lewit (1977,1983), however, employing the essential framework of Ben-Porath, is able to extract a positive income effect on pregnancy expenditures, and hence on survival as well. Lewit alters the budget constraint in (7) by appending a fixed time cost per pregnancy which varies across women but is fixed for each individual.

Lewit makes the distinction between variable time costs and fixed time costs. The former is subsumed under m, expenditures per pregnancy, and is therefore endogenous. He gives time spent in birthing classes or time expended obtaining prenatal care as examples. The fixed time cost consists of time lost giving birth in addition to the time lost late in a pregnancy from the difficulty or inability to perform tasks that can be routinely handled when not pregnant. Lewit defines this second component of fixed time costs as "a reduction in the effective productivity of maternal time."

The next step is to assume that the opportunity cost of a women's time is positively correlated to her income. Therefore, even if all women lose the same amount of time due to pregnancy, the fixed time costs will differ because of variations in the opportunity cost. In sum, women with more human capital have higher reservation or market wages, hence greater income, which creates a positive association between income and the fixed time costs of a pregnancy.

To incorporate this into model II presented above, let F represent the fixed time costs of a pregnancy. The budget constraint becomes:

$$y = nm + x + Fn \tag{10}$$

Solving (10) for x and substituting the result into the survival production function (6) and then maximizing this function with respect to m and n yields the following equilibrium condition

$$s/s_m = m + F \tag{11}$$

Differentiating (11) produces the following result:

$$dm/dF = -F_i[(s_m^2 s)/S_{mm}] > 0 \tag{12}$$

where F_i is the marginal change in fixed time costs with respect to a change in income.

This important result rests critically on the assumption that fixed costs are positively correlated to income. To support this Lewit cites the literature on household production theory relying on the relationship between human capital, or market efficiency, and income. Yet women with relatively high levels of human capital are very likely to be efficient in non-market activities as well. Hence they may be able to offset their large opportunity costs by greater efficiency in the non-market tasks relative to women with less human capital, lower opportunity costs and less efficiency in non-market activities.

This need not imply that women with more human capital can do household chores faster and more efficiently than women with less human capital. Instead, women with more human capital, and hence income, can hire help, pay them a wage which is less than their own opportunity costs, and free themselves to more effectively utilize their time and strength.

More formally, let the fixed time costs of a pregnancy equal the time lost due to birthing and recovery (L) plus the time lost from reduced productivity (t) all multiplied by the opportunity cost of time (w). Thus

$$F = wL + wt \tag{13}$$

L is constrained to be the same for all women and independent of human capital. W and t are both functions of human capital such that dw/dHC > 0, and dt/dHC < 0. It follows easily that the elasticity of fixed costs with respect to human capital is:

$$e_{fh} = e_{wh} + e_{th}k \quad \text{such that} \quad k=wt/F,\ e_{wh}>0, \text{ and } e_{th}<0 \tag{14}$$

where e_{wh} is the elasticity of the wage rate with respect to human capital and e_{th} is the elasticity of lost productivity with respect to human capital. If human capital is positively correlated to income, it is not necessarily true that increases in human capital across individuals (and hence income), lead to higher fixed costs.[1]

Intuitively, it is quite reasonable to assume that expenditures per pregnancy are a positive function of income. However, given the restrictive utility function used above, any attempt to extract a

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[1] In the case where a woman hires someone to help her, equation (13) can be amended as follows:

$$F = wL + w_0t_0 - w_1t_1$$

W_0 is the wage paid to hired help; t_0 is the time the help is paid for; w_1 is the opportunity cost of the pregnant woman and t_1 is the time she spends on other tasks while her hired help does the work that other pregnant women who don't have help must do.

positive effect of income on survival is bascially ad hoc. The only way an income effect can truly be derived from a model of birth outcomes like the ones presented above, is if the utility function is made more general allowing survival (s) to enter as a separate commodity. Yet the complications that are immediately introduced by this generalization generate indeterminate signs to most of the comparative statics. This model is dealt with in greater detail below.

The next major issue addressd by Lewit's model is the impact of changes in reproductive efficiency on expenditures. Lewit, following Grossman (1972), uses an asymptotically constrained production function to parameterize the probability of survival.

$$s = 1-A\exp(-bm) \qquad (15)$$

As Lewit explains, the functional form implies that zero expenditures per pregnancy does not preclude survival. As such, the intercept (1-A) can be interpreted as the parents' genetic birthing endowment. The greater the intercept (the smaller is A), the greater the likelihood of survival. Hence one can directly examine the result of a change in A on expenditures.

Substituting (15) into (11) and implicitly differentiating the result holding b and F constant yields:

$$dm/dA = (1+m+F)/(m+F)B^2A \quad > 0 \qquad (16)$$

By specifying the functional form of the production function as such, Lewit obtains the unambiguous result that parents will compensate for unfavorable changes in, or information about, their genetic birthing endowment by increasing their expenditures per pregnancy.

As mentioned above this may be an important behavioral response to information concerning the health of the fetus, or as a precautionary measure due to a family history of problematic births. Even though this issue is fundamentally an empirical one, it has important econometric implications which will be taken up in more detail in Chapter II.

The above model can be generalized further by entering survival as a separate commodity in the utility function.

$$U = (s,b,x) \qquad (17)$$

In equation (17), s is the probability of survival, b is the number of births and x is the composite commodity. Williams (1976), borrowing from the quantity/quality framework of Becker and Lewis (1973), uses s as the measure of child quality. What distinguishes the quality/quantity framework from the typical 3 commodity demand analysis is the budget constraint. In Williams' model y is money income, r_i (i=b,s,x) represents money prices and r is the price of survival and births together. The budget constraint can be written as

$$y = nsr + r_b b + r_s s + r_x x \qquad (18)$$

Maximizing (17) subject to (18) yields the following first order conditions:

$$U_b = l(sr + r_b) = lP_b \qquad (19)$$

$$U_s = l(br + r_s) = lP_s \qquad (20)$$

$$U_x = lr \qquad = lP_x \qquad (21)$$

P_b and P_s are the shadow prices of births and survival respectively and l is the lagrangian multiplier. The interaction occurs because changes in the market prices of the commodities alter the relative shadow prices. More specifically, let r_b be the cost of children which is independent of survival. Examples include the costs of discomfort and lost productivity during pregnacy, the costs of avoiding pregnancy and the cost of delivery. r_s is the cost of survival which is independent of parity. An example might be an exogenous decrease in air pollution. The effect, therefore, of an exogenous increase in the price of b relative to s is a substitution away from b towards s. An increase in s further raises the shadow price of b which in turn induces additional substitution from b to s. This interaction between births and survival continues until a new equilibrium is reached.

An important feature of the quantity/quality model is the full simultaneity between mortality and fertility. However, as Rosenzweig and Wolpin (1980) have pointed out, the unobservable shadow prices require that further restrictions be imposed on the utility function before one can distinguish between interactions in b and s arising from a true quality/quantity relationship rather than the simple complementarity of two goods.

The next advance on the 3 commodity model is to introduce a technology for survival. Willis (1973) was the first to integrate the household production framework with the quality/quantity interaction. The result was a very general model of fertility that included both the supply and demand of child services. However, since the focus of this study is on the demand for child quality, as represented by survival, I shall move to a more recent framework developed by

Rosenzweig and Schultz (1982,1983a,1983b) which integrates the innovations of Willis (1973) and Grossman (1972) in a model of the demand for child health.

Unlike Willis, Rosenzweig and Schultz are able to relax the restriction on joint production by permitting goods that are inputs into the production of child health to enter the utility function as a commodity. By doing so they have again resurrected the work of Grossman (1972), and used this very general framework for explaining the economic aspects of birth outcomes. Because the framework of Rosenzweig and Schultz will be used to implement this empirical study, it is worth discussing in some detail.

Rosenzweig and Schultz emphasize that household production theory is very amenable to the study of health because the concept of a health technology is biologically well accepted. Their model begins with a three commodity utility function.

$$U = U(X,H,I) \qquad (22)$$

X represents non-health related consumption; H is child health and I is the commodity that augments utility as well as affects health. The health production function can be written as follows:

$$H = H(I,Z,u) \qquad (23)$$

where Z is an input into the production of health and u is the unobserved health endowment of the mother. The budget constraint follows straight forwardly:

$$Y = P_x X + P_i I + P_z Z \qquad (24)$$

The dual role of I as an input into the production of health and as a commodity in the utility function is what distinguishes this model from Willis'. By incorporating joint production Rosenzweig and Schultz are able to explain the consumption of such items as cigarettes, alcohol, good food and exercise. Furthermore, X can also be the number of children which enables the model to account for the possible qualtiy/quantity interaction described by Becker and Lewis (1973).

Another feature emphasized by Rosenzweig and Schultz is the distinction between goods which directly affect health and variables which determine the demand for these inputs. To make this clearer equation (22) can be maximized subject to equations (23) and (24) and made to yield the following reduced-form demand functions:

$$X = X(P_x, P_i, P_z, Y, u) \tag{25}$$

$$I = I(P_i, P_x, P_z, Y, u) \tag{26}$$

$$Z = Z(P_z, P_x, P_i, Y, u) \tag{27}$$

Substitution of equations (25)-(27) into (23) generates what Grossman (1972) terms a reduced form demand for health.

$$H = D(P_x, P_i, P_z, Y, u) \tag{28}$$

Rosenzweig and Schultz point out that such reduced form equations, the focus of previous research, fail to exploit the insights offered by household production theory because of their inability to separate tastes from technology. This doesn't mean attempts have not been made to estimate a health technology; however, the estimates of equation (23) have been "hybrids" in that variables used in the reduced form equations (25)-(28), such as income, are employed in the production function as proxies for inputs lacking data, such as nutrition. The result, as demonstrated by the authors, is generally biased estimates of the true technology.

Their criticism is a valid one and it has the additional benefit of clarifying the distinction between exogenous and endogenous variables. Inputs (I) which are the result of parental choice are obviously endogenous. Inputs which do not directly enhance utility (Z), yet may be correlated to the endowment term, are also treated as endogenous. Environmental factors which impact on health but are essentailly beyond parental control or awareness, are assumed to be exogenous. Finally, there are the price and resource constraints (P,Y) which determine the utilization of the endogenous health inputs.

The use of proxies to estimate the health production function is not the only source of error. Even if the health production function were well-specified, the estimates would still be biased. The reason, the authors assert, is due to the correlation between the health inputs (Z,I), and the unobservable endowment term (u). Rosenzweig and Schultz term the variation in people's genetic stock, "health heterogeneity." Generally, this unobserved determinant of health is assumed to be randomly distributed and uncorrelated with other inputs. This is not to imply that the dangers of this assumption have gone unnoticed (Grossman, 1972,1975). However, Rosenzweig and Schultz are the first to systematically outline and estimate this potential source of bias.

According to the authors, individuals have some information concerning their endowed health. In the case of birth outcomes, parents often have knowledge obtained from past experience or family history about the possible complications expected at birth. The response of individuals to this expectation generates the correlation between the use of health inputs and the unobserved health endowment which is imbedded in the residuals. For example, women who have had a miscarriage in the past are more likely to seek out prenatal care earlier and to monitor their pregnancy more closely than women with no such prior experience. Yet, in spite of these precautionary measures, the birth may nevertheless be problematic (i.e. low-birth weight). Hence, the benefits of this enhanced care will have been masked. Stated differently, in an OLS regression of birth outcomes on prenatal care, the coefficient of prenatal care will underestimate its true impact.

There are good reasons to believe that such bias exist. Advances in prenatal diagnosis have been rapid. Procedures such as ultrasound and amniocentesis provide significant data on the health and viability of the fetus. This permits physicians and parents to adjust their programs of prenatal care and to more thoroughly prepare for a high risk birth. However, until such information in made a part of the available data, this interaction will continue to be a growing source of bias.

The notion that exogenous changes in the genetic birth endowment of a woman may alter her behavior has been modeled by Ben-Porath (1973), Williams (1976), and Lewit (1977,1983) as outlined above. By restricting their analysis to a two good utility function and a single health input, Rosenzweig and Schultz obtain the same result as Lewit (see equation 16)-- that parents will compensate for unfavorable information concerning their biological birthing endowment. In the more general case of three or more goods, the direction of the parents' response to such information cannot be determined a priori. Unlike others however, Rosenzweig and Schultz have made the issue of health heterogeneity of central importance in their modelling and empirical study of infant health.

The framework of Rosenzweig and Schultz is not without its problems. By not using proxies for variables that lack data they are essentially trading omitted variable bias for decreases in simultaneous equation bias. Second, being able to disentangle tastes from technology is a formidable econometric task which presupposes data on all the commodities and shadow prices relevant to the model (Pollack and Wachter 1975, 1977; Barnett 1977). Nevertheless, their model is able to account for a wider range of behavior than those before it. For these reasons the model developed by Rosenzweig and Schultz forms the basic analytical framework for this study.

A. Cost of Contraception

As mentioned in the introduction, a novel aspect of this research is the inclusion of abortion and family planning use as inputs (Z) into the production of child health (equation 23). Utilization of either service is obviously a method of achieving a desired family size. Yet the use of such contraception is not costless either materially or psychologically. Following Willis (1973), therefore, the costs of avoiding a birth can be included in the budget constraint. Let b* stand for the number of births a fecund couple would have if they made no attempt to prevent conception. The cost of fertilty control can be then written as C(b*-b).

To incorporate the cost of contraception into the framework above let X in equation (22) be the number of births (B) and let I be the only input into the production of health in equation (23). The decision facing the parents can be rewritten as follows:

$$\text{Maximize } U = U(B,I,H) \qquad (29)$$

subject to $H = H(I,u)$ (30) and $Y = P_b B + P_i IB + C(B^*-B)$ (31)

The first order conditons yield the following equilibrium condition:

$$U_b/U_i = (P_b + P_i I - C)/[P_i B-(U_h/1)H_i] \qquad (32)$$

An exogenous decrease in C due to Medicaid financing of abortions or federal subsidization of family planning clinics increases the shadow price of births relative to health inputs. This should initiate a subsitution away from B towards I; however, due to the interaction between B and I in the budget constraint, the shadow price of B should increase even further as the demand for I rises. The variation in demand for health inputs across individuals depends on the efficiency with parents can produce health and their taste for healthy children. If health production functions are asymptotic, as Grossman (1972) and Lewit (1977,1983) suggest, then the more successful parents are at producing healthy infants holding tastes constant, the smaller will be the change in H_i as more I is consumed and the greater will be the change in the relative shadow price of births to inputs.[2]

B. Abortion and Sample Selection Bias

The expanded use of abortion and family planning have enabled individuals to more effectively plan the number and timing of their offspring. The choice of whether to give birth or not has set up a potentially significant self-selectivity problem. The issue is analogous to the more commonly cited examples of self-selection found in the literature (see Maddala, 1977). For instance, the mean wage of working women is not a correct measure of the average wage of women if all women were to participate in the labor market; or, the earnings of individuals who choose to migrate is not a good estimate of what similar individuals would earn had they migrated. In the case of neonatal mortality the estimated survival probability based on a sample of women who choose to give birth may understate the neonatal mortality rate that would prevail if all pregnant women tried to carry to

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[2] In a more restricted model such as Lewit's (1977) discussed above, the cost of contraception can be shown to be inversely related to the expenditures per pregnancy. The budget constraint in Lewit's model (equation 10 above) can be amended by adding the costs of contraceptions as follows:

$$Y = nm + x + Fn + C(n^*-n) \qquad \text{(F2-1)}$$

Maximizing (4) subject to (5) and (F2-1) yields the equilibrium condition

$$s/s_m = m + F - C \qquad \text{(F2-2)}$$

where it is easily shown that

$$m_C = s_m^2/S_{mm}s < 0 \qquad \text{(F2-3)}$$

M_C is the partial derivative of a change in expenditures per pregnancy with respect to a change in the cost of fertility control.

full term. The fact that in 1977 the abortion rate for whites and non-whites was 20 and 59 and the abortion ratio was 333 and 679 respectively suggests that the magnitude of the selectivity bias may be substantial.[3]

Following Heckman, (1976,1979), selectivity bias may be viewed as a form of specification error. As such, the residuals in a structural equation of interest have a non-zero mean conditioned on the regressors in the sample selection equation. As Heckman writes, "fitted regressions functions confound the behavioral parameters of interest with parameters of the function determining the probability of entering the sample." (1979, p.154).

To adjust for selectivity bias using Heckman's two-stage procedure, a researcher would need a cohort of women observed from conception. A sample selection criteria could be established predicting the probability of not obtaining an abortion. Given all the necessary assumptions, the appropriate adjustment factor (lambda) could be estimated and entered into the structural equation as a right-hand-side variable.

Generally, this adjustment factor is not interpretable as a regressor. In this particular case, however, lambda has a very useful interpretation. For instance, the smaller the value of lambda, the greater the probability that a women is included in the sample. In other words, the more unlikely a women is to abort, the smaller the value of lambda. One interpretation of lambda, therefore, is that it

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[3] The abortion rate is the number of abortions per women 15-44 while the abortion ratio is the number of abortions per thousand live births (<u>Statistical Abstract of the United States, 1984</u>).

represents the "wantedness" of a birth. Women with a high probability of aborting, because of age, or marital status or from an inability to support an infant financially, may want a child less than women whose pregnancy was better planned and whose likelihood of aborting is very low.

Numerous researchers have alluded to wantedness as a potential explanation for the increased healthiness of recent newborns (Harris, 1982; David and Siegel, 1983). If a baby is more wanted, then all else being equal, a mother is likely to eat more nutritiously, seek prenatal care earlier, and in general have a more heightened concern for the impact of her behavior on the fetus. In short, the more unlikely a women is to abort, the more wanted the birth and the greater the probability of a favorable birth outcome.

Despite the rather formidable estimation problems, the above discussion offers insights into the interpretation of abortion at the aggregate level. Assuming that the demand for abortion is constrained in part by price and income, then even if the wantedness of a birth is randomly distributed, abortions will vary systematically across counties. Areas which provide financing and accessibility to abortion should have fewer unwanted births than counties in which abortion is less readily available. Therefore, where the abortion rate is higher, the proportion of unwanted births will be smaller, and the probability of unfavorable birth outcomes should be reduced.

C. Health and Schooling

The empirical association between parental schooling and measures of child health has been consistently observed to be positive. Attempts to explain this regularity have focused on the enhanced non-market efficiency which parents with higher levels of schooling bring to the production of child health (Michael, 1973). More formally, given equal amounts of health inputs, parents with more education can achieve greater output of child health than their less educated counterparts holding all else constant. For example, given the same information from an obstetrician concerning the significance of diet during pregnancy, parents with higher levels of schooling will more effectively use that knowledge to heighten the probability of a favorable birth outcome. In short, schooling increases the marginal product of an input and is consequently termed the "efficiency effect."

The impact of education may be neutral or non-neutral. In the former case the marginal product of all inputs is enhanced by the same percentage; a non-neutral change alters the various marginal products differently. When education has a neutral effect on the inputs, the relative price of the health inputs is unchanged; in this case the demand for child health increases, decreases, or remains the same depending on whether its income elasticity is greater than, less than or equal to one respectively (Grossman, 1972; Michael, 1973). When schooling has a non-neutral effect on inputs, the demand for child health may not necessarily rise given an increase in the marginal product of one input relative to others. Furthermore, if the input which experiences a relative increase in efficiency is also a utility-augmenting commodity, then the effect of schooling on the demand for health is made more ambiguous (Rosenzweig and Schultz, 1981).

Alternatively, education may enhance parents' ability to correctly perceive the true relationship between an input and child health. As such, parents choose the mix of inputs which most effectively produces child health. Rosenzweig and Schultz (1981), following Welch (1970), call this the allocative effect. Hence, more educated women may smoke less during pregnancy because they are more aware of the potential hazards. Therefore, if education has a purely allocative effect, then women with different levels of education, but using the same amount of identical inputs, should produce the same output of child health, all else held constant.

Theoretically, then, it should be possible to distinguish between a pure efficiency effect and an allocative effect of parental schooling on infant health. If mother's education is entered in a well-specified structural equation of infant health, then evidence of a purely allocative effect would be a small and insignificant coefficient. The opposite result, a large and significant coefficient for mother's education, would suggest that an efficiency effect was present; this, however, would not rule out the possibility of an allocative effect as well.

The dangers of leaning too heavily on such a test are rather obvious. For example, rarely can a production function be well-specified in that all the relevant health inputs are included. In the case of birth outcomes, data on nutritional inputs are often lacking. Consequently, entering a women's education in a production function of infant health where information on diet is not included may give misleading results if education is positively correlated with good nutrition.

Conclusion

The models just reviewed have all set forth the propositon that birth outcomes are in part determined by parental choices. This insight was put forth by Grossman (1972) in his general model of health. However, not until Rosenzweig and Schultz (1981,1982,1983a,1983b) has this structure clearly been implemented such that the epidemiological relationships, or the association between medical care, nutrition, etc., and health, have been clearly distinguished from the economic relationships, or the impact of constrained opportunities on the demand for the health inputs.

The distinction is more than nomenclatural. All the models acknowledge the importance of a genetic component in determining birth outcomes. For Rosenzweig and Schultz, however, the genetic component is exogenous, yet partially known by the parents, which creates a correlation between the inputs and the residuals in the health production function. This assumption instantly binds the epidemiological relationships (the dose-response function) to the economic ones (the demand functions) because the former can no longer be estimated unbiasedly without the latter. In other words, since the health endowment is not expected to vary with the determinants of the input use, these price and resource variables can be used as instruments to predict that portion of the health inputs which is independent of the genetic component.

The upshot then is that the estimation of the birth outcome production function should be done by simultaneous equation methods. Variables which enter the infant health production function as the result of parental decisions should be treated as endogenous while the remaining variables in the model can be reasonably defended as exogenous. These criteria will be used in estimating the neonatal mortality and birth weight production functions presented in Chapter IV. They also provide a standards by which other research in this area can be evaluated.

CHAPTER II--Empirical Review

This chapter reviews the empirical work on birth outcomes. The literature is voluminous and much of it comes from the medical sciences. This chapter, however, is confined to research done by economists in the field of infant mortality. Since this study has emphasized the importance of the appropriate framework in the analysis of birth outcomes, this section pays particular attention to the distinction between reduced form versus structural models; the endogeneity of various inputs; and the appropriateness of the data for measuring what the author claims is being captured. As in any study of health, the epidemiological relationships are critical. This aspect of the literature will be refered to in Chapter III which discusses the actual variables used and why.

Williams (1976) was one of the first to emphasize the importance of estimating infant mortality and fertility simultaneously, even though she herself was unable to do so. Nevertheless, aware of these endogenous relationships, she emphasizes the biological determinants of neonatal mortality and birth weight over the medical and socioeconomic inputs. For example, among the regressors in her neonatal mortality equation are the race and sex of the child, the number of previous infant deaths and fetal losses prior to the present birth, the completed family size, whether the mother was less than 19 years old and the legitimacy status of the child.

Of the above regressors only the race and sex of the child should be treated as strictly exogenous. Williams treats all the inputs as exogenous. She argues that fetal losses and previous infant deaths may reflect in part the health endowment of the parents and therefore can be justifiably considered exogenous. However, she acknowledges that to the extent that fetal losses and previous infant deaths are a function of a woman's health endowment, they could be viewed as endogenous. Births to women 18 years or less and illegitimate births should be treated as endogenous risk factors because they represent life-cycle decisions by parents or mothers as to the timing of their offspring. Williams, however, assumes these inputs to be exogenous as well. A justification for treating these varibles as exogenous, which Williams doesn't make, is that the data is from a retrospective study of women born between 1910 and 1925. Since abortion and contraception were not readily accessible or reliable, the argument can be made that the timing of children was much less of a choice than it is today.

Williams includes income and education measures in her neonatal mortality production function. Neither of the measures is significant. Nevertheless, Rosenzweig and Schultz (1983b) have shown that the inclusion of reduced form variables may bias the estimates of the other inputs. Furthermore, since the equation already includes indicators of teenage births and legitimacy status, the insignificance of education may imply that schooling is a better proxy for high-risk births than as a measure of the efficiency in the production of healthy infants.

The criticisms of the neonatal equation can also be applied to the estimated birth weight function. First, many of the variables should be treated as endogenous. Second, the mixing of resource measures such as income and health insurance with the appropriately included biological determinants yields a hybrid function that may be misleading. Finally, the potential colinearity between inputs like the number of previous live births and births to women over thirty-four jeopardizes the stability of those estimates.

Lewit's (1977,1983) work on birth outcomes is derived from a similar theoretical model, but his empirical application has several advantages. First, Lewit's data has none of the time dimension problems that Williams felt hindered her estimates. Second, Lewit has a much larger sample of births. Third, his data is restricted to a single metropolitain area which serves to minimize the common problem of missing data on medical fees. Finally, Lewit has well-defined measures of medical care use. Lewit's production function, however, is also a hybrid including both health inputs and determinants of health inputs. Lewit defends this procedure by arguing that income and education may be proxies for such lifestyle variables as nutrition, smoking and alcohol consumption.

The use of proxies is an essential part of social science reasearch. However, within a particular context, it is important to ask whether a proxy, broadly defined, enlightens as much as it obscures. Rosenzweig and Schultz (1982,1983b) have noted that one of the appealing aspects of the application of household production theory to health is that the concept of a health technology is so well accepted. Moreover, the technology is more biological or epidemiological than it is economic. What economics brings to the study of health is the notion that choices individuals make concerning the use of these health inputs is constrained by prices, resources, tastes and information as to the correct relationship between inputs and outcomes. To obfuscate this distinction in the hope of achieving a better specified equation may be to forfeit the advantage of economic theory to the empirically more appealing yet theoretically more ad hoc approach of epidemiology.

For example, how does one interpret mother's education? As an efficiency variable? As a lifestyle variable? Or as a risk factor? In Lewit's birth weight equation the coefficient of mother's education switches signs across specifications and is never significant. Surely one cannot conclude that neither smoking nor nutrition have an impact on birth weight. Lewit also includes legitimacy status and mother's age; certainly teenage mothers are much more likely to have illegitimate children and relatively fewer years of schooling. The point is that education may be insignificant because it is a proxy for such risk factors as births to young mothers and illegitimate births.

Furthermore, there is good justification for assuming that such lifestyle variables may well be correlated with a mother's endowed health. Information acquired during prenatal care visits may reveal potential difficulties with the fetus. Part of this may be picked-up in the birthing experience variables but there is a stochastic component to any pregnancy. If a women alters her behavior because of information so acquired, then the relevant inputs and the residuals should be correlated. This is especially true for the number of prenatal care visits. Pregnancies which have been diagonsed as problematic are more likely to receive additional attention resulting in extra visits. In such cases, the birth may be premature or light weight, but it will be paired with an above average number of prenatal care visits thus potentially masking the beneficial effect of extra care.

Unlike Williams, Lewit never discusses the possible simultaneity between the fertility measures and birth weight. This is in accord with his model which made survival a function of expenditures and reproductive efficiency. However, it is difficult to argue that the number and timing of births are not parental choices conditioned on such life-cycle variables as earnings. Instead, Lewit views the first birth, all previous births, and the number of fetal and/or infant deaths as exogenous measures of reproductive efficiency. However, as Williams (1976) points out, previous fetal or infant deaths may be determined by unobserved family characteristics which also affect the birth weight of the child under study.

A major advantage of Lewit's specifications to those of Williams' (1976) is the inclusion of a well-defined measure of medical care. In addition to the number of prenatal care visits, Lewit added the trimester in which care began. Unlike the number of visits, there is less of a mechanical relationship between the length of the pregnancy and the trimester in which care began. However, as Harris (1982) indicates, women who initiate care late, if at all, may constitute a group with more homogenous birthing characteristics than women who begin care earlier. These characteristics may confound the relationship between prenatal care and birth outcomes. More specifically, during a pregnancy weaker fetuses are naturally selected out. Women whose pregnancies have lasted to the third trimester with no prenatal care may be endowed with latent characteristics that compensate for the lack of care. Not adjusting for what Harris (1982) terms, "fetal selection" tends to obscure the causal relationship between prenatal care and birth outcomes.

This caveat aside, Lewit's estimates indicate that prenatal care has a substantial impact on birth weight holding gestation constant. For example, a woman with twelve prenatal care visits beginning in the first trimester will give birth to an infant 303 grams heavier than a women with no care at all. Lewit plays down the results a bit by acknowledging that the biological mechanism by which prenatal care impacts on birth weight has not been elucidated. Harris (1982) adds that prenatal care may indirectly affect birth weight by lengthening gestation but again, the link between prenatal care and intrauterine growth has not been explained biologically.

In sum, the major criticism of Lewit's work on birth outcomes is the potential endogeneity of the variables. As such, his distinction between a "demand, production or outcome equation" versus a "production or outcome equation" is not well delineated. The use of this hybrid specification tends to merge the economic determinants with the epidemiological relationships, blurring their distinctions and hence their indiviudal strengths. However, the inclusion of a well-defined medical input, and the result that its impact is substantial and significant, underscores the importance of medical care measures for explaining the determinants of birth outcomes.

The first study to examine the role of abortion and family planning services on birth outcomes in a multivariate context was conducted by Grossman and Jacobowitz (1981). A striking finding was that the increase in legal abortion was the most important factor in the decline in the United States neonatal mortality rate since 1964 for both whites and nonwhites. The increased subsidization and availability of abortion and family planning services, the authors hypothesized, not only reduced the number of high-risk births, but may have been responsible for the decline in risk-specific deaths as well. In particular, by lowering the cost of contraception the optimal number of births should fall while the optimal resources devoted to each birth should rise.

Grossman and Jacobowitz use the county as their unit of observation to estimate a reduced form "demand for survival" function. This enables them to investigate the impact of government programs, such as Medicaid, on infant deaths. The disadvantage of a reduced form as opposed to a structural equation, is that the mechanisms through which a specific program operates cannot be explored. For example, an increase in the number of states whose Medicaid benefits cover first-time pregnancies for all financially eligible women may increase the use of prenatal care by the poor. This, in turn, may improve the probability of an infant surviving by lowering the likelihood that the birth will be of low weight. Another possibility is that the expansion of Medicaid coverage results in better perinatal care and thereby increases the survival rate of low-birth-weight births. A reduced form specification is unable to address these issues.

Except for abortion, the programs examined in the Grossman and Jacobowitz study are aimed at poor women. Consequently, they employ a specification that interacts the policy measures with the estimated percentage of births to poor women within a county. In other words, the greater the proportion of women who are poor, the greater should be the impact of a poverty-specific program on the total neonatal mortality rate. This is an important insight that is exploited in a later study by Corman and Grossman (1984), and which is also used in the present study. This interactive specification is further discussed in Chapter III.

A variable used by Corman and Grossman (1984) that may be endogenously determined is the number of active non-federal physicians per 1000 population which is used as a proxy for the price and availability of medical care. Counties with poor health, as measured by high infant mortality, may attract physicians because the demand for their services is greater. If this is the case, then the demand for doctors is determined simultaneously with the demand for survival and the ordinary least squares estimates are biased. It should be noted that in all of the neonatal mortality regressions presented by Grossman and Jacobowitz, the coefficient on physicians has the wrong sign.

Hadley (1982), using Grossman's (1972) health model, investigated the importance of medical care on neonatal mortality averaged over the five-year period from 1969 to 1973. Using county groups in the U.S. he also examined how the liberalization of abortion and the Medicaid financing of first-time pregnancies affected neonatal mortality.

Unlike Grossman and Jacobowitz (1981), Hadley estimated a structural health production function. However, given data limitations his measures of medical care usage were crude, and in general, a better indicator of medical care availability than use. The distinction is important. As will be argued in the next chapter, the availability of an input helps determine the indirect costs, and hence, the shadow price of obtaining that good. This is especially relevant with medical care in which health insurance may cover part or all of the actual fees. Ideally then, variables which measure the use of an input belong in the structural equation while indicators of availability, which determine use, should appear in the reduced form.

Hadley uses Medicare expenditures per enrollee in each county group as a proxy for prenatal and perinatal care use. He assumes that the consumption of medical care for pregnancies and births is a fixed proportion of Medicare expenditures. Aside from its loose link to medical care usage, the assumption of a fixed relationship between Medicare expenditures and birthing expenditures is potentially problematic in states with a large percentage of very old residents, but with relatively ungenerous benefits to poor mothers. His other measure of medical care usage is the number of obstetricians and pediatricians per 1000 live births. Not only is this variable more appropriate as an availability measure but, as discussed above, its exogeneity is questionable.

Following Williams (1979), Hadley controls for high risk births by estimating the expected neonatal mortality rate within a county group. He multiplied the sex and race birth weight-specific neonatal mortality rate obtained from 1960 data by the fraction of births averaged over the period 1969 to 1973. This measure of risk is the single most important factor explaining the variation in neonatal mortality for both race and sex. Another result of interest is that neither of his two proxies for the consumption of prenatal and perinatal care are affected substantially when expected mortality is held constant. Hadley interprets this to mean that even though the association between prenatal care and birth weight is well-established at the individual level, the aggregated medical care measure and expected mortality at the county group level are distributed independently.

Another interpretation is that Hadley's medical measures are poor proxies for prenatal care. Using county data Corman, Joyce and Grossman (1984) have shown that the direct effect of initiating prenatal care in the first trimester, holding birth weight and gestation constant, is essentially zero. What Hadley's results do imply is that relatively high levels of medical expenditures and large numbers of health professionals enhance the survivability of risk-specific births.

Finally, Hadley's results support the findings by Grossman and Jacobowitz (1981) on the significance of abortion as a factor in reducing neonatal mortality. With respect to Medicaid financing of first-time pregnancies, Hadley reports that states which do not cover unborn children have a neonatal mortality rate for males that is 4 to 5 percent higher. Grossman and Jacobowitz's results were less definitive in this respect. The Medicaid measures have the correct sign only for blacks.

Corman and Grossman (1984) updated the study by Grossman and Jacobowitz (1981) to determine whether their findings and those of Hadley (1982) still prevailed. Also at issue was the effectiveness of federal programs specifically aimed at improving birth outcomes in the U.S. Specifically, they explored whether the Supreme Court decision in 1973 legalizing abortion nationally and the subsequent growth in the abortion rate, continued to explain the variation in neonatality mortality rates across large counties in the U.S. in 1977. Other programs of interest were the Maternal and Infant Care projects, the Supplemental Food program for Women Infants and Children (WIC), the expansion in family planning clinics and the increase in community health centers.

Unlike previous studies Corman and Grossman were able to measure the contribution of neonatal technology in preventing infant deaths. They combined information from the American Hospital Association and from Ross Laboratories on the state-specific number of hospitals with Level II, or Level III, or Level II and Level III neonatal intensive care units. Level III represents the most sophisticated facility for managing ill neonates, but owing to the rather ambiguous distinction between a Level II and Level III unit, the two were combined.

Corman and Grossman once again estimated a race-specific, reduced form demand for survival equation. However, the data in the updated version represents a significant improvement. First, the distinction between use and availability is followed strictly. For instance, the number of aboriton providers, family planning clinics, and the sum of the maternal and infant care projects and community health centers per women 15 to 44 are used as proxies for the indirect costs of obtaining an abortion, family planning services and prenatal care respectively. The appropriateness of using availability measures to capture indirect costs is supported by survey evidence (Chamie et al., 1982) which shows that proximity and accessibility are major factors in determining the use of these clinics by teenagers. This is not surprising since many of the clinics, which are heavily subsidized, charge nominal and often no fees for their services.

Another improvement in the updated version is the elimination of the number of physicians per population as a measure of prenatal and perinatal care availability. As a partial substitute for the quality of perinatal care and also to control for differences in neonatal technology, the number of state-specific hospitals with Level II and

Level III neonatal intensive care units is also included.

In the updated study, the availability of abortion is a powerful and significant determinant of the variation in neonatal mortality across counties. The coefficient for blacks, it should be noted, is four times the coefficient for whites. Furthermore, when the black coefficients are applied to national trends in abortion they account for ten percent of the decline in neonatal mortality since 1964. For blacks, therefore, the accessibility of abortion is the single most important factor explaining the decline in early infant deaths.

A similar difference between races exists with respect to neonatal intensive care. The black coefficient is two to four times greater, depending of the specification, than the white one. Other differences between blacks and whites were found for the programs developed specifically to combat high infant mortality. For instance, the WIC program and Medicaid financing of first-time pregnancies have important negative effects on white neonatal mortality only, while the Maternal and Infant Care projects and community health centers retard neonatal mortality for blacks.

In sum, Corman and Grossman have made an important contribution to the empirical study of infant deaths. First, their results underscore the gains to be made from estimating race-specific functions whenever possible. Unexplained differences between races are often attributed to genetic dissimilarities (Lewit, 1983). Although this may be the case, their study indicates that the impact coefficients on the various programs differ by race. Second, the rapidly evolving advances in neonatal technology makes time series analysis extremely problematic. Consequently, impact parameters estimated from a cross-sectional analysis are an important means of gauging the effect of medical technology. As their results indicate, neonatal technology plays a major role in the survivability of newborns. Finally, Corman and Grossman convincingly establish the strong association between abortion and infant health. A major focus of this thesis will be to explain in detail the various mechanisms through which abortion operates to affect infant health.

Perhaps the most important contribution in recent years to the economic analysis of infant health has been made by Rosenzweig and Schultz in a series of papers (1981,1982,1983a,1983b). The significance of their work lies less in their estimates and more in the empirical framework they have imposed on the study of newborn health. As noted in Chapter II, the concept of a health production function was rigorously put forth by Grossman (1972) and applied to the study of fertility and mortality by Willis (1973), Ben-Porath and Welch (1973), Williams (1976) and Lewit (1977,1983). However, along the way economists clouded the distinction between a structural and a reduced form equation of health. The primary reason was simple: the data was never available to adequately fit a well-specified structural equation. Instead, researchers estimated hybrids, a mixture of demand and production variables, sometimes termed a quasi-structural equation.

The main contribution of Rosenzweig and Schultz is the resurrection of the distinction between tastes and technology. They argue that of all the areas to which household production has been applied, health remains one of the few areas where the concept of a health production process is well-accepted. For instance, the epidemiological literature has shown that there is a strong association between the number, spacing and timing of births, nutritional and medical consumption, and birth outcomes. As Schultz (1984) points out, what economic analysis brings to the study of infant health is an understanding of parental behavior with regard to the demand for the inputs which have been shown to be important epidemiologically.

However, Rosenzweig and Schultz's most important insight is that the estimation of the epidemiological associations cannot be separated from the underlying economic relationships. To understand why, one must return to the notion of a genetic component in the determination of an individual's health. Rosenzweig and Schultz term this "health heterogeneity." They hypothesize that many individuals have some information or expectation about the relationship between this genetic component and the health outcome at issue. In response to this knowledge individuals alter their behavior which then sets up a correlation between this unobserved element and the observed consumption of the health inputs. In econometric terms, the inputs on the right hand side of a health production function are correlated with the residuals, thus rendering ordinary least squares estimates biased and inconsistent.

For instance, because of past experience with drug abuse, a pregnant women may be concerned that her fetus will be adversely affected. As a result, she initiates prenatal care much earlier than she might otherwise. The women delivers prematurely, but due to early supervision of her pregnancy, the doctor is able to prolong gestation and improve the likelihood of survival. In this instance, early prenatal care is associated with prematurity masking the ameliorating effect of prompt care.

To overcome this bias, Rosenzweig and Schultz suggest that the determinants of the health inputs be used as instruments to purge the correlation between the inputs and the residuals. More specifically, since price and income measures are assumed to vary independently of the health endowment, the predicted values from the health input equations should be used to obtain unbiased estimates of the epidemiological relationships between the health inputs and the birth outcome.

The results from their recent work point to a substantial difference between structural equation coefficients estimated by OLS and those obtained by simultaneous equation methods. For example, they (1983b) report that neglecting to correct for heterogeneity bias leads to OLS estimates that understate by a factor of forty the effect of delaying prenatal care on birth weight. Although the magnitude of the change differs across specifications, their finding that heterogeneity bias exists and causes direct correlations to understate the effect of key health variables is an important finding.

Rosenzweig and Schultz (1982,1983a,1983b) and Schultz (1984) use individually matched birth and death certificates from the 1968 and 1969 National Natality Followback Surveys to estimate birth outcomes: infant mortality, birth weight and gestation. The latter two birth outcomes are continuous variables and thus the authors are able to control for gestation when estimating birth weight. This allows them to examine the direct effect of an input on birth weight holding gestation constant. It also produces a clearer understanding of how various inputs operate and lessens the bias due to heterogenity dramatically.

In this series of studies, for example, the two-stage least squares estimates of prenatal care fall by a factor of thirty when gestation is held constant.[1] In addition, the magnitude of the change atttributed to heterogeneity bias falls. More specifically, the 40-fold increase in the coefficient of prenatal care "due" to heterogeneity bias becomes a factor of two when gestation is held constant. Using the same data set, Schultz (1984) reports that due to heterogeneity bias the change in the prenatal care coefficient is twelve-fold in the birth weight equation, five-fold in the gestation equation, while in the infant mortality equation the coefficient changes sign and becomes significant. Yet in neither the birth weight nor the mortality regression is any attempt made to control for the corresponding intermediate birth outcome.

This omission is important. Heterogeneity bias appears to exist, but the degree of the bias seems questionably large. This is especially true in the infant mortality equations presented by Rosenzweig and Schultz (1983a) and Schultz (1984) because there is little concensus that prenatal care has an independent effect on infant mortality holding birth weight constant. Conventional wisdom maintains that prenatal care improves birth weight by prolonging gestation (Harris, 1982), but even this argument lacks solid biological evidence. What this implies is that the large effects attributed to heterogeneity bias may in fact be a result of omitted varible bias. To obtain a more accurate estimate of how much the direct association between inputs

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[1] These results are from their 1983b paper. The functional form is Cobb-Douglas.

and outcomes is biased, it is important that estimates be obtained from specifications that capture the direct effects of health inputs on health outcomes.

In a related paper, Rosenzweig and Schultz (1982) test whether the frequently observed association between education and health is due to an efficiency effect or an allocative one. The former augments the productivity of an input while the latter lowers the cost of obtaining information concerning the correct relationship between the health inputs and the outcome. According to the authors, if education has an allocative effect then one would expect the coefficient on the schooling variable in a structural equation of health to be zero. A significant effect on health holding the appropriate inputs constant, would suggest that education increased the marginal product of the inputs. This, however, would not eliminate the possiblitiy that an allocative effect exists simultaneously with an efficiency effect.

Their results on this point are as ambiguous as their test. In both the birth weight and the birth weight standardized for gestation equations, the coefficient on the schooling variable is positive and significant. Nevertheless, the authors argue that since the residual sum of squares always increases with the inclusion of education, one cannot reject the null hypothesis that education should be excluded from the structural health production function. In others words, specifications which incorporate neutral and non-neutral efficiency effects are not "statistically preferable to the 'pure' allocative models."

There are a number of problems with this procedure. First, the test requires that the production function be well-specified. Nutrition, for instance, is an important input in birth outcomes, yet rarely is this information available in data sets such as the one used by Rosenzweig and Schultz. Further, there is a strong possibility that nutrition and education are positively correlated. If this is the case, then a significant efficiency effect could be implied from a result in which education was really a proxy for some other factor such as a healthy diet.

Second, as discussed above, controlling for intermediate birth outcomes is an important means of ascertaining the direct effect of an input. The association between low levels of education and low-birth weight have been frequently noted in the literature (Chase et al., 1973; Taffel, 1980). Therefore, in order to determine whether there are efficiency effects in the production of survival, it would be erroneous to include education on the right hand side without controling for birth weight because many of the risk factors which operate through birth weight, such as teenage and illegitimate births, are highly correlated with low levels of schooling.

In sum, the work of Rosenzweig and Schultz has called attention to the self-selection of health inputs and the need, therefore, to treat them endogenously. This is an important issue because medical diagnostic technology is advancing far more rapidly than the speed with which the results from these tests are being included in data sets. Procedures such as ultrasound and amniocentesis supply information that is often very precise regarding the genetic make-up of the fetus. Thus, parents are able to adjust their behavior even to the point where they choose to abort.

This study will incorporate these insights in estimating structural health production functions for neonatal mortality and birth weight. Since the unit of observation is the county, the neonatal mortality rate and the percentage of low-birth-weight births will be the relevant health outcomes. An advantage of aggregate data in the present context is that the proportion of light births can easily be controlled for when fitting an infant mortality equation. As discussed above, this will permit an examination of the extent to which heterogeneity bias is a factor when estimating the direct and indirect impact of an input on the birth outcomes. Furthermore, controlling for these and other risk factors will provide a measure of the possible efficiency and/or allocative effects when education is included.

CHAPTER III--Empirical Implementation

This chapter describes the data employed and the estimation procedures used in the specification of the neonatal mortality and birth weight production functions as well as the reduced form input demand equations. Descriptions of the three types of variables--birth outcomes, health production inputs, and income and availability measures--are presented in four subsections. Wherever possible, race-specific variables are used in the regressions. Such variables are denoted with an asterisk. Except for abortion, Medicaid and neonatal intensive care measures (which are available only for states), all variables are county-specific. Table 1 contains definitions and acronyms of the variables while Table 2 contains the means and standard deviations.

The basic data set used in this study is the Area Resource File (ARF), a county data service prepared by Applied Management Sciences, Inc. for the Bureau of Health Professions, U.S. Department of Health and Human Services. It incorporates information from different sources for the 3,077 counties of the U.S. Race-specific data on neonatal deaths, birth weight and gestational age, as well as birth by various demographic characteristics for 1976 through 1978, are from the National Center for Health Statistics (NCHS) Natality Tape.

Data on health manpower comes from the American Medical Association. Socioeconomic characteristics are taken from the Census of Population, and estimates on smoking are from Eugene Lewit of the National Bureau of Economic Research. Measures pertaining to the

Table 1

Definitions of Varibles[a]

Variable	Definitions
Neonatal mortality rate* (NMR*)	Three-year average neonatal mortality rate centered on 1977; deaths of infants less than 28 days old per 1,000 live births
Low birth weight* (LBW*)	Three-year average percentage of low-birth weight (2,500 grams or less) live births centered on 1977
Gestational age*[d] (GEST*)	Three-year average percentage of premature (gestational age of 36 weeks or less) live births centered on 1977
Teenage family planning users* (FPTEEN*)	Percentage of women age 15-19 who used organized family planning clinics in 1975
Abortion rate (ABOR)	Three-year average state-specific resident abortion rate centered on 1976; abortions performed on state residents per 1,000 women age 15-44
Prenatal care*[d] (PRECARE*)	Three-year average percentage of live births for which prenatal care began in the first trimester (first three months) of pregnancy centered on 1977
Neonatal intensive*[g] care (NICU*)	Sum of state-specific hospital inpatient days in Level II, Level III, or Levels II and III neonatal intensive care units in 1979 per state-specific three-year average number of births centered on 1977
Cigarettes (SMOK)	State-specific daily number of cigarettes smoked per adult 18 years and older in 1976
Births to teenagers* (YOUNG*)	Three-year average percentage of live births to women age 15-19 centered on 1977
Illegitimate births*[d] (ILEGIT*)	Three-year average percentage of live births to unmarried women centered on 1977

Table 1 (continued)

Parity*[d] (PARITY*)	Three-year average percentage of fifth and higher order births centered on 1977
Births by mother's education*[d] (EDBIR*)	Three-year average percentage of live births to women with at least a high school education centered on 1977
POV*[b]	Percentage of women age 15-44 with family income less than 200 percent of the poverty level in 1980
HSP*[c]	Percentage of women age 15-49 who had at least a high school education in 1970
MPA[e]	Dichotomous variable that equals one if county is in state that covered first-time pregnancies under Medicaid to financially eligible women in the period 1976-1978
MPU[e]	Dichotomous variable that equals one if county is in state that covered first-time pregnancies under Medicaid only if no husband was present or if the husband was present but unemployed and not receiving unemployment compensation in the period 1976-1978
MPN[e]	Dichotomous variable that equals one if county is in state that covered first-time pregnancies under Medicaid only if no husband was present in the period 1976-1978
MNEW	Dichotomous variable that equals one if county is in state in which Medicaid paid for newborn care under the mother's number but allowed pregnant women to register their "unborn child-ren" with Medicaid in 1981
MBEN	State specific average annual Medicaid payment per adult recipient in AFDC families in fiscal 1976
FPCLIN[f]	Number of organized family planning clinics in 1975 per 1,000 women age 15-44 with family income less than 200 percent of the poverty level in 1975

Table 1 (continued)

BCHSP[f]	Sum of maternal and infant care (M and I) projects and community health centers (CHCs) in 1976 per 1,000 women age 15-44 with family income less than 200 percent of the poverty level in 1975; numerator termed Bureau of Community Health Services (BCHS) projects
ABPROV	Three-year average of abortion providers centered on 1976 per 1,000 women age 15-44 in 1975
NEOH	Sum of state-specific number of hospitals with Level II, Level III, or Levels II and III neonatal intensive care units in 1979 per 1,000 women age 15-44 in state in 1975
AFDC	Three-year average AFDC payment per recipient centered on 1977
Ln population density (POPDEN)	The natural logarithm of the ratio of the population in 1975 to the area per square mile

Notes to Table 1

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

[b]Variable is available for nonblacks and blacks as opposed to whites and blacks

[c]Variable is available for whites and nonwhites as opposed to whites and blacks

[d]There are unknowns for this variable in some counties. The exact number that are missing and the instrumental procedure used to estimate values for the missing counties is presented in Appendix A.

[e]The variables MPA, MPU, and MPN characterize the eligibility of first-time pregnant women for prenatal care under Medicaid. The omitted category pertains to states that cover no first-time pegnancies because their AFDC programs do not recognize "unborn children."

[f]Since the numerator of this variable is not race-specific, the also denominator is not race-specific. The denominator is obtained by applying the race-specific percentage of women age 15-44 with family income less than 200 percent of the poverty level in 1980 to the race-specific number of all women age 15-44 in 1975.

[g]This variable, the number of inpatient days per low-birth weight birth, has been multiplied by the percentage of low-birth weight births in accord with the interactive model described below. The resulting variable is the number of inpatient days per birth.

Table 2

Means and Standard Deviations of Variables[a]

	Whites		Blacks	
	Mean	Standard Deviation	Mean	Standard Deviation
NMR*	8.550	1.574	15.648	3.425
LBW*	5.946	.739	12.890	1.317
GEST*[b]	7.375	.838	15.611	1.983
FPTEEN*	9.186	6.296	24.178	9.832
ABOR	25.150	8.825	24.916	8.724
NICU*	.644	.383	1.523	1.071
PRECARE*[b]	78.625	8.297	60.183	10.923
YOUNG*	13.741	4.037	29.037	4.844
ILEGIT*[b]	8.477	2.345	52.665	8.679
PARITY*[b]	4.097	2.126	7.132	2.177
SMOK	7.399	.538	7.477	.358
EDBIR*[b]	76.312	9.950	62.302	8.005
ABPROV	.058	.044	.059	.038
FPCLINxPOV*	.072	.057	.151	.130
BCHSPxPOV*	.005	.011	.014	.020
NEOH	.011	.004	.010	.003
MPAxPOV*	.112	.146	.146	.237
MPUxPOV*	.034	.089	.052	.157
MPNxPOV*	.023	.078	.099	.227
MNEWxPOV*	.247	.109	.521	.154
MBENxPOV*	119.477	55.323	239.900	69.293
HSED*	63.107	7.371	44.389	9.322
POV*	26.507	8.763	54.871	9.505
AFDC	76.032	24.466	69.232	28.537
POPDEN	6.295	1.485	7.176	1.664
Sample size	677.		357.	

[a]An asterisk (*) next to a variable means that it is race-specific. Means and standard deviations are weighted $(b_i/(p_i(1-p_i))^{1/2}$ where b_i is the sum of births from 1976-1978 in county i, and p_i is the probability of an infant dying in the first 27 days in county i. These are the same weights used to estimate the neonatal mortality equations as explained in Chapter III.

[b]The mean is calculated for the sum of known and estimated values (see Appendix A).

policies and programs used in the reduced form input demand equations are from Corman and Grossman (1984). Finally, data on family planning and abortion are from the Alan Guttmacher Institute.

Separate regressions are fitted for white and black birth outcomes. One rationale for race-specific regressions is that the neonatal mortality for blacks is almost twice the corresponding rate for whites. Moreover, the percentage of black births of low-birth weight was more than double the white figures nation-wide in 1977. By fitting race-specific regressions, the coefficients on inputs are allowed to vary between races. In addition, differences in the demand for inputs can also be examined.

Counties are used as the unit of observation instead of states or SMSA's because they are more homogenous with respect to socioeconomic characteristics and medical resources. However, small counties present a number of potential problems. First, people may travel outside the county for medical services; second, small counties with few births may show large fluctuations in birth outcomes due to random movements. To minimize these difficulties, only counties with a population of 50,000 or more are included. For the black sample, there is the additional criterion of at least 5,000 blacks in the county. There are 677 counties in the white regressions and 357 counties in the black regressions. The counties used in the white regressions account for approximately 80 percent of the white population of the U.S. in 1970; the counties in the black regressions account for a similar percentage. In addition to selecting large counties, random elements are attenuated by employing three-year averages of the race-specific neonatal mortality rate and percentages of low-birth weight births for the period 1976-1978 as the dependent variables. Finally, weighted regressions are used where the set of weights depends on the functional form that is imposed.

A. Birth Outcomes

Since the data are at the aggregate level, the outcomes are the neonatal mortality rate and the percentage of low-birth weight births. A third outcome, the percentage of births for which gestational age is less than 37 weeks, is used as a right-hand-side variable in the birth weight regressions. Due to measurement problems and missing observations, a gestional age production function is not estimated. More will be said concerning the role of gestational age below.

The neonatal mortality rate is used instead of the more frequently encountered infant mortality rate for several reasons. First, the neonatal mortality rate, as opposed to the postneonatal rate (death between 28 days to 365 days after birth) accounts for 71 percent of the all infant deaths in 1977. In short, the neonatal mortality rate dominates the infant mortality rate. Second, most neonatal deaths are caused by congenital abnormalities, prematurity and complications at birth. These conditions are more sensitive to prenatal, perinatal and neonatal care than are infectious diseases and accidents, the primary causes of postneonatal mortality.

Birth weight is the most accurate and most proximate cause of neonatal mortality. The probability of a baby dying in the first 27 days of life in 1976 was forty times greater for a light baby (less than 2500 grams or 5.5 pounds) than for an infant of normal weight.

Moveover, since 1965 the rate of decline in the neonatal mortality rate has far outstripped the corresponding decline in the proportion of low-birth weight births.

Further, McCormick (1985) notes that in many regions of the country, birth weight-specific mortality rates for high and low risk groups are becoming increasingly more similar. This suggests that if the declining trend in neonatal mortality is to be sustained, then substantial efforts will have to be made to reduce the proportion of light births. Consequently, understanding the determinants of low-birth weight, and the degree to which various inputs impact on mortality through birth weight is of major policy significance.

In 1977 gestational age was not reported on birth certificates in eight states. Moreover, this variable is subject to potential measurement error due to the difficulty of determining the data of a woman's last menstrual period. Consequently, in 100 white counties and 65 black counties gestational age is missing for at least 70 percent of all births. Therefore, gestational age is estimated in unknown counties by instrumental variable estimation described in the appendix.

Due to the missing data, a gestational age production function is not computed. However, this measure of prematurity is used as a right-hand-side variable in the birth weight production function because some of the inputs which impact on birth weight may operate through gestation. More specifically, a great deal of controversy revolves around the issue of whether prompt initiation of prenatal care increases birth weight because the physicians's instructions concerning nutrition, smoking, drinking, etc., are more likely to be followed;

or, due to careful monitoring of the pregnancy, a potentially premature birth can be delayed, thereby increasing intrauterine growth and hence birth weight. By controlling for gestational age such issues can be directly addressed.

A major caveat to the use of gestational age is the potential measurement error which renders the coefficients biased and inconsistent. If prematurity is orthogonal to the other regressors then only its coefficient is potentially problematic. However, due to the argument just made above, this is unlikely to be the case. Therefore, the estimates from a specification which includes gestational age must be interpreted with caution.

B. Health Production Inputs

As mentioned above, many researchers have suggested that the increased use of contraception may be responsible in part for the accelerated decline in neonatal mortality since the late 1960's. To examine this proposition, the percentage of teenagers within a county who used family planning services in 1975 is included in both the birth weight and mortality production functions. It is included in both structural equations to reflect the fact that relatively high utilization of family planning services may operate in two distinct ways. First, contraceptive use may lower the proportion of births in such high-risk categories as births to young mothers and illegitimate births. For example, Forrest (1981) estimates that for every ten adolescents enrolled in family planning clinics in 1975, three pregnancies were averted the following year. Thus, despite the increase in teenage sexual activity, the adolescent birth rate fell by 18 percent between 1970 and 1975. Hence, if in a structural equation of birth weight, the effect of teenage family planning use is reduced significantly when such risk factors as adolescent and illegitmate births are added, then this would support the proposition that family planning impacts on mortality by improving the distribution of high-risk births. On the other hand, if teenage contraceptive use has a significant effect on neonatal mortality holding birth weight constant, then this would suggest that due to better planning, more teenagers may delay births so that more resources are being devoted to pregnancies which are not averted.

This study focuses on teenage family planning use as opposed to the utilization of family planning clinics by all women of childbearing age (15-44) for several reasons. First, teenagers are disproportionate users of family planning clinics. Estimates by Corman, Joyce and Grossman (1984) indicate that while nonwhite adolescents represent only 23 percent of the nonwhite women of childbearing age, the average ratio of nonwhite teenagers who used family planning clinics in 1975 to the race-specific number of users ages 15-44 is .40. Second, adolescent births and adolescent births out-of-wedlock are two groups with a well-documented, above normal risk of low-birth weight and neonatal mortality (Taffel, 1980). Consequently, reductions in teenage pregnancies may bring about a relatively large reduction in infant mortality. Third, Corman, Joyce and Grossman (1984) find that when family planning use by teenagers, and total family use are entered as separate variables in a regression predicting neonatal mortality, the measure of adolescent use is negative and consistently dominates the broader indicator of family planning use. Fourth, teenage family planning use is available by race whereas use by all women is not.[1]

The number of abortions by state of residence per woman age 15 to 44 operates in a manner similar to family planning, but its impact is expected to be greater. First, the abortion rate is not restricted to teenagers. Second, the epidemiological evidence supporting the association between increased legalized abortions and reductions in fertility, illegitimacy, prematurity and births to adolescents 15 years or younger has been noted by numerous researchers (Sklar and Berkov, 1974; Bauman et al., 1977; Lanman et al., 1974; Shelton, 1977). Moveover, Tietze (1984) reports that an often overlooked benefit of legalized abortion is that "women with medical contraindications to continued pregnancy, especially poor and minority women, now have better access to legal pregnancy termination." (p. 26). He goes on to show that the relative number of abortions performed on medical indication rose dramatically during the 1970's especially when compared to the previous decade. These findings support the hypothesis that abortion impacts on birth weight and mortality by reducing the proportion of births within high-risk categories. Finally, abortion, perhaps more so than family planning use, may be responsible for what David and Siegel (1983) refer to as the trend towards "better babies,"

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[1] The Alan Guttmacher Institute provided the data on teenage family planning use. Their estimates were adjusted to compensate for cross-county utilization of family planning services. However, in 26 counties the figures on family planning use by nonwhite teenagers appeared abnormally high (above 50 percent or approximately 2.5 standard deviations from the mean adjusted for the outliers). In the 26 black counties where this problem was relevant, the state mean was substituted.

a reference to the increased healthiness of newborns within risk-specific groups due to their being better planned and more "wanted."

Lewit[2] however, questions the link between abortions and declines in neonatal mortality rates on the grounds that abortion may be a substitute for other forms of contraception. As such, abortion may increase the fertility rate by enhancing the level of sexual activity. In fact, sexual activity among adolescents rose substantially between 1970 and 1975 (Forrest, 1981). Whether abortion was responsible in part for the increase is certainly debatable. However, if abortion were a substitute for other forms of birth control, then one would expect an increase in pregnancy rates and possibly the birth rate as well. Yet, from 1970 to 1977 the birth rate fell across age groups. In addition, Forrest (1981) cites evidence indicating that pregnancy rates actually declined over the first half of the 1970's.[3] Furthermore, Grossman and Jacobowitz (1981) point out that by controlling for family planning use the problem of abortion serving as a proxy for other forms of contraception is mitigated.

The use of prenatal medical care in this study is given by a three-year average of the percentage of live births in which prenatal care began in the first trimester of pregnancy centered on 1977. This is a more desirable measure of prenatal care use than the number of

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[2] See Grossman and Jacobowitz, 1981, footnote number 4.

[3] The illegitimate birth rate has risen 9 percent since 1972. Interestingly, however, if one looks at the birth rate of out-of-wedlock births as measured at conception, then the illegitimate birth rate has fallen over the same time period by 8 percent. The difference is due to the fact that more and more women who conceive premaritally choose not to get married before delivering their baby.

prenatal care physician visits per birth because the latter variable is mechanically related to the length of the pregnancy. That is, longer pregnancies, which in general are associated with more favorable birth outcomes, are also associated with more visits.

Prenatal care is entered in both the neonatal mortality and low-birth weight production functions. Conceptually this can certainly be disputed, but empirically there is much to recommend it. The primary reason relates to the inability to account for the quality and quantity of perinatal care, a common problem in the study of birth outcomes. Yet, if women who initiate prenatal care in the first trimester are correlated to women who have ready access to high quality perinatal care, then this measure of prenatal care may have a significant effect on the survivability of a newborn. Another reason for suspecting a relationship between early initiation of prenatal care and neonatal mortality is that women who monitor their pregnancies closely, as represented by those who begin care at the onset of their pregnancy, may also be women who eat nutritiously and avoid the use of alcohol and drugs, other inputs for which data at the county level is not available.

The conventional wisdom, however, is that prenatal care impacts primarily on birth weight. But even this association has been questioned due to the absence of a convincing biological link between the care generally administered during a routine visit and intrauterine growth. As Stanley (1977) notes, the "function of prenatal care is often no more than educational and selective." (p. 269). Hence, prenatal care may operate on birth weight by simply lengthening gestation (Harris, 1982). Other researchers, however, report a significant effect of prenatal care on birth weight holding gestational age constant (Gortmaker, 1979; Lewit, 1983). Showstack et al. (1984) have recently argued that by not controlling for the nonlinear relationship between length of gestation and birth weight the true impact of prenatal care on birth weight holding gestational age constant may be underestimated. In particular, they note that after 40 weeks of gestation the effect of additional gestation on birth weight is minimal. Consequently, in a regression of birth weight on prenatal care and gestational age the coefficient on prenatal care is biased downwards if this nonlinearity is not taken into account. The authors ran the above regression with additional maternal characterisitcs excluding births for which gestational age was greater than 40 weeks. Their results indicate that prenatal care has a substantial impact on birth weight especially for blacks.

In this study the direct and indirect effect (through gestation) of prenatal care on birth weight will be analyzed holding constant the proportion of births of gestational age less than 37 weeks. This gestational measure is well below the point at which nonlinearity becomes a factor, and thus the results should be free of the bias mentioned above.

The use of neonatal intensive care services is measured by the sum of the state-specific number of hospital inpatient days in Level II, Level III, or Level II and III neonatal intensive care units in 1979 divided by the state-specific three-year average of the number of low-birth weight births centered on 1977. The denominator pertains to light neonates because they are the primary users of the neonatal intensive care units. Hospitals that provide neonatal care are generally divided into three levels based on the intensity of care they are equipped to deliver. Level I hospitals provide minimal or normal newborn care; Level II hospitals provide intermediate care; and Level III hospitals provide the most intensive care (Budetti et al. 1981).

As mentioned above, the dramatic decline in neonatal mortality over the the past two decades has been attributed largely to reductions in birth weight-specific mortality. Favorable changes in the distribution of prematurity have rarely accounted for more than 30 percent of the decline in neonatal mortality (Lee et al., 1980; Williams and Chen, 1983; David and Siegel, 1983). Because of this, many researchers have pointed to advances in the management of the newborn as a primary reason for the increased survivability of low-birth weight infants (Paneth et al. 1982). This suggests that there may be important interactive effects between the use of neonatal intensive care and the proportion of low-birth weight births within a county.[4] More formally, let r_{kj} be the neonatal mortality rate of of low-birth weight babies and r_{nj} be the corresponding rate for normal weight babies. As an identity

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[4] With the exception of teenage family planning use none of the other inputs are targeted at a specific population. Family planning clinics are designed to serve areas of high fertility and infant mortality. However, the use of these clinics is not restricted to women from low-income families. Therefore, if neonatal mortality were available by family income, teenage family planning use would not be restricted to an equation predicting the neonatal mortality rate of low-income women. This is not the case with neonatal intensive care use. As outlined in the text, if mortality rate were available by birth weight, the model would limit its impact to babies of low-birth weight.

$$r_j = k_j r_{kj} + (1-k_j) r_{nj} \qquad (1)$$

where r_j is the observed neonatal death rate and k_j is the percentage of light births. Ignoring other inputs specify production functions for r_{kj} and r_{nj} as follows:

$$r_{kj} = a_o + a_1 m_j + e_{1j} \qquad (2)$$

$$r_{nj} = b_0 + e_2 \qquad (3)$$

In these equations m_j is neonatal intensive care use and e_{1j} and e_{2j} are the residuals. Equation (3) makes the simplifying assumption that the mortality rate of normal weight babies is a function of random factors only; this can be altered with little loss in generality. Since birth weight specific death rates are unavailable at the county level substitute (2) and (3) into (1) to obtain

$$r_j = b_0 + (a_0-b_0)k_j + a_1 k_j m_j + v_j \qquad (4)$$

where $$v_j = k_j e_{1j} + (1-k_j)e_{2j} \qquad (5)$$

Thus, in order to correctly specify an equation that attempts to capture the possible interactive effects between birth weight and neonatal intensive care, the proportion of low-birth weight births (k_j) should be included along with the measure of neonatal care (m_j) as right-hand-side regressors. Furthermore, the larger the proportion of low-birth weight births the greater the marginal effect of an increase in neonatal care use $(dr_j/dn_j) = a_1 k_j)$. Consequently, to fit the above model, the neonatal care intensive measure is multiplied by the fraction of low-birth weight births. The resulting variable can be interpreted as the race-specific number of inpatient days in neonatal intensive care units per birth.

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

The studies linking maternal smoking to birth outcomes have consistently showed a dose-response type gradient between the amount of cigarettes consumed and the heightened probability of a low-birth weight birth (Meyer et al., 1976). The impact of smoking on gestational age has also been noted but the finding remains less consistent and less significant than the relationship between smoking and birth weight. By estimating a birth weight production function that holds gestation constant, the differential impact of smoking on birth weight and gestational age can be directly addressed. Moreover, since the evidence suggests that smoking during pregnancy effects neonatal mortality by increasing prematurity (Meyer and Tonascia, 1977), cigarette consumption is only included in the birth weight production function. This restriction has the additional benefit of providing identification in the estimation of the neonatal mortality and low-birth weight production functions as a system of structural equations. This will be discussed further in Chapter Four.

The next three inputs can be categorized under the rubric of endogenous risk factors because they represent life-cycle decisions concerning the timing and spacing of births as well as the mother's marital status at delivery. In particular, the race-specific, three year average (1976-1978) of the percentage of births to teenagers 15 to 19, the percentage of fifth and higher order births and the percentage of illegitimate births are used to capture the impact of these life-cycle choices on birth weight.

A well-documented result of adolescent pregnancies is the increased risk of low-birth weight infants and neonatal deaths (Chase, 1972). One explanation for this heightened risk is that the average teenage diet is nutritionally deficient. In a survey cited by Taffel (1980), 90 percent of the teenagers interviewed had less than the recommended iron, and more than half lacked the advised amounts of protein, vitamin A and vitamin C as part of their daily intake. These deficiencies are most likely compounded by the additonal nutritional demands of a growing fetus. Futhermore, pregnant youths, on average, have lower levels of schooling which may be associated with not only a poor diet, but also the use of cigarettes, alcohol and drugs.

In previous work Corman, Joyce and Grossman (1984) made the assumption that endogenous risk factors, such as births to teenagers, impacted on neonatal mortality indirectly through birth weight and gestation. This restriction is retained for the mortality production function but relaxed in the birth weight equation. Consequently, It is possible to more explicity map out the mechanisms by which abortion and family planning, for example, effect infant deaths. More specifically, if the use of family planning clinics by teenagers effectively reduces adolescent pregnancies, then one should expect the coefficient of the family planning variable in the birth weight regression to fall in absolute value when the proportion of teenage births is held constant. The same is true of the abortion variable since teenagers are disproportionate users. Moreover, one can address the question of whether teenage mothers have an above normal rate of low-birth weight births because they deliver prematurely or, because of a poor diet, for example, they give birth to full-term, light infants. Examination of this issue requires that gestational age be controlled for when regressing birth weight on the percentage of newborns delivered to adolescents. A small direct effect of teenage births on birth weight

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[5] To clarify the notion of a direct and indirect effect consider the following model:

$$b = a_0 + a_1X + a_2g$$

$$g = B_0 + B_1X$$

where b is the percentage of low-birth weight births, g is the percentage of births for which gestational age is less than 37 weeks and X is an input such as abortion, family planning or a risk factor such as births to teenagers. The direct effect of X on b is a_1. The indirect effect is obtained by substituting for g in the birth weight equation; it equals B_1a_2. The total effect is simply the sum of the direct and indirect effects $(a_1+B_1a_2)$. Even though a gestational age production function is not estimated, it is easy to see that since a_1 and B_1 are negative (when X measures abortion or family planning) would strongly suggest that young mothers give birth prematurely.[5]

Children born out-of-wedlock face substantially higher risks of neonatal mortality and low-birth weight (Chase et al., 1973; Berkov and Sklar, 1976). Since most illegitimate births are to adolescents, this risk factor will be highly correlated with the percentage of teenage births. For instance, Berkov and Sklar (1976), using a two-year average of births from 1971 to 1972 show that there is little difference in the death rates between legitimate and illegitimate births for women less than twenty. In fact, black teenagers who give birth out-of-wedlock have lower infant death rates than their legitimate counterparts. However, for all age groups above twenty, black and white, the infant mortality rates are substantially higher for illegitimate children. Therefore, despite the high correlation between the percentage of teenage and illegitimate births, the latter variable is expected to capture the separate effect of marital status on birth outcomes.

Lewit (1983) has commented that there is no biological link between legitimacy status and low-birth weight. Instead, he considers marital status as a proxy for the "wantedness" of a birth. Williams (1976) argues in a similar manner. The reasoning seems to be that births which are better planned are more wanted and less likely to be out-of-wedlock. Given that both Lewit and Williams used data that pertained to individuals before the liberalization and subsequent legalization of abortion, the argument has some merrit. However, since

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while a_2 is positive, the greater the indirect effect of X on b, the larger will be the decline in the coefficient of X when g is held constant.

the 1973 Supreme Court decision on abortion, the percentage of unwanted pregnancies has surely risen, but as Fuchs (1983) notes, three-fourths of all unintentional pregnancies end in abortion. Consequently, many of the unmarried women who carry to term do so because they want the child. Fuchs points to the excess demand for adoption as an indication of the decline in unwanted births. Legitimacy status, therefore, may pick-up the effect of poor nutrition, a poor health endowment or less access to high quality prenatal and perinatal care.

As in the case of births to teenagers, legitimacy status may lessen the impact of abortion on low-birth weight especially if the effect of abortion is to lower the proportion of births within high-risk categories. As mentioned above, approximately 75 percent of all abortions are performed on unmarried women. Moreover, O'Connell and Rogers (1984) indicate that from 1955 to 1973 the percentage of all first-time births conceived out-of-wedlock rose from 17 to 37 percent. However, since 1973 the figure has fallen, despite the rise in teenage sexual activity, such that the percentage of all first-time births conceived premaritally stood at 30 percent in 1981.

The third risk factor to be considered is the race-specific percentage of fifth and higher order births. The effect of birth order on infant mortality is somewhat mixed. Rosenzweig and Schultz (1983a,1983b) report a positive and significant effect of birth order on newborn survival and birth weight. Shapiro et al. (1968), on the other hand, find survival to be negatively related to parity. A better measure of the risk associated with high fertility and short intervals between births would be parity conditional on age as used by Chase et al. (1973). For example, teenage births of a second and higher order, or fourth and higher order births to women 20 to 24 years old are each associated with a dramatically enhanced likelihood of an unfavorable birth outcome. Given the aggregated nature of the data in this study, the best that can be done to capture the risk of poor spacing and high fertility is to interact age- and race-specific birth rates with the proportion of births within that category. As a result, counties with high teenage birth rates but where adolescents make-up a relatively small proportion of the women of childbearing ages should have a lower neonatal mortality rate than a county with a similar teenage birth rate but where teenagers account for a disproportionately large percentage of the women between the ages of 15 to 44. This interactive variable would have the additional benefit of picking up the potential nonlinear effects of fertility on birth outcomes (Rosenzweig and Schultz, 1982).[6] I will experiment with various forms of fertility along these lines.

As discussed in Chapter III, education has been used to examine whether mother's schooling plays an allocative or efficiency role in the production of infant health (Rosenzweig and Schultz, 1981). A major difficulty with the attempt to distinguish between these two functions is that levels of schooling are highly correlated with legitimacy status, teenage births, and other high-risk categories. Stated differently, teenage and unmarried mothers are less likely to pursue an education and low levels of schooling may be indicative of

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[6] Let TB_j be the number of births to teenagers, T_j the total number of teenagers and B_j the total number of births all in county j. The interactive variable discussed in the text would be $TB_j^2/(T_j*B_j) = (TB_j/B_j)*(TB_j/T_j)$.

women with less knowledge of contraception and the risks associated with smoking and drug use during pregnancy. Given this two-way causality, mother's education is more correctly treated as an endogenous risk factor. Rosenzweig and Schultz (1982), for example, estimate two birth weight production functions in which gestational age is controlled for in one specification and not the other. Mother's education is included in both equations but is significant only when gestation is held constant. In other words, lower levels of schooling are associated with shorter gestation. The authors control for some risk factors by excluding all illegitimate births and including mother's age. Nevertheless, lower levels of schooling are probably correlated with inadequate nutrition and possibly alcohol or drug use. The point is that unless a production function is well-specified, the use of education without controlling for other risk factors or intermediate birth outcomes is misleading and difficult to interpret.

This study will examine the effect of mother's education in the production of infant survival and birth weight. Regressions including the race-specific, three-year average of the percentage of births to women with at least a high school education will be run with and without the endogenous risk factors discussed above. This same specifications will be examined controlling for the relevant intermediate birth outcome. In this way, the role of education can be more clearly mapped out.

Finally, the natural logarithm of the population density in 1975 is included in all structural specifications to capture the exogenous impact of environmental factors. Using data from the 1976 national natality tape, Taffel (1980) shows that the black and white percentage of low-birth weight births is highest in large urban areas and that it falls progressively as one moves to less urban and more rural areas. This relationship holds despite controlling for variations in the educational attainment of the mother. Rosenzweig and Schultz (1982) use individual data and find that residing in an SMSA and the population of an SMSA are positively related to prematurity. There are a number of possible explanations for this inverse association. For example, Lave and Seskin (1973,1977) report a significant effect of air pollution on neonatal and infant mortality across SMSA's in the U.S. In addition, Bakketieig et al. (1985) make the point that overcrowding associated with poor housing conditions may be related to poor pregnancy outcomes.

In the sample used in this study the population density ranges from a low of 7 to a high of 62,130. Given this spread, it is difficult to conceive of the relationship between neonatal mortality and population density being linear. Consequently, the natural log of the population density is used instead.

C. Income and Availability Measures

The area characteristics utilized to predict input use consist of variables which measure the availability of health inputs, income and tastes. The distinction between input use and input availability is important to this study and merits further discussion. Acton (1975) has shown that nonmonetary factors such as traveling and waiting time act as prices in determining the demand for medical services where the cost of the service is wholely or partially covered by insurance.

Hence, increased availability of an input ought to lower the shadow price of a service and thereby increase the quantity demanded. On the other hand, use of an input such as prenatal care or abortion is a choice variable determined by the availability of the service, command over resources, and tastes. Measures of input use belong in the structural equations [equation (23), Chapter Two] whereas variables representing availability should be used in the reduced-form input demand equations [equations (24)-(26), Chapter Two].

For example, the availabilty of abortion is measured by the three-year average number of abortion providers per 1,000 women age 15 to 44 in 1975. This variable is expected to be positively related to the number of abortions performed between 1975 and 1977. As noted in Chapter Two, the accessibility of family planning clinics has been cited as a major determinant of their use by teenagers. The use of neonatal intensive care as measured by the number of inpatient days should also be positively correlated with the number of hospitals with Level II or Level III intensive care units in 1979 per 1,000 women ages 15 to 44. Finally, the sum of maternal and infant care projects and community health centers per women 15 to 44 with income less than 200 percent of the poverty level in 1975 should be positively related to the use of prenatal care.[6]

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[7] Many of the program measures used in the reduced form are interacted with the proportion of women 15 to 44 with income less than 200 percent of the poverty level. The reason is similar to the one used in the case of inpatient days being interacted with the proprotion of low-birth weight births. In short, the greater the proportion of poor women within a county, the greater the potential effect of these programs on birth outcomes. For a detailed discussion see Grossman and Jacobowitz (1981), and Corman and Grossman (1984).

In addition to differences in availability, the price variations of medical inputs are captured by four dichotomous variables which distinguish between states that covered all first-time pregnancies and all newborn care and those which offered more restricted prenatal and perinatal coverage between 1976 and 1981. The income measures used are the race-specific percentage of women age 15 to 44 with family income less than 200 percent of the poverty level in 1980; the state-specific average annual Medicaid payment per adult recipient in AFDC families in 1976; and the three-year average AFDC payment per recipient (1976-1978).

It is not clear a priori how each of these resource measures will impact on the demand for the various inputs. The poverty level should be negatively related to the initiation of early prenatal care (Gortmaker, 1979), abortion and neonatal care intensive use, yet positively correlated with measures of fertility such as the percentage of teenage births and fifth and high order births. Generous Medicaid benefits should have a positive effect on prenatal and neonatal care use since physicians in states with relatively low reimbursement schedules under Medicaid are less likely to treat Medicaid patients (Sloan et al. 1978). However, relatively high average AFDC payments may be negatively related to abortion if families that are more able to provide for children because of state support have more children. For the same reason, AFDC payements may be positively associated with the other measures of fertility.

Parental tastes are captured by the race-specific percentage of women age 15 to 49 in 1970 with at least a high school diploma. This measure differs from the schooling variable used in the production functions in that the latter variable is the ratio of births to women with at least a high school education between 1976 and 1978 over the total number of births for the same period. Due to this difference there should be less bias owing to the possible feedback from levels of education to the percentage of births to teenage mothers. This measure of schooling should have a positive effect on prenatal care (Chase et al. 1973; Gortmaker, 1979), and a negative effect on abortion if more educated women are more likely to use contraception to avoid unwanted pregnancies. Since higher levels of education are associated with more human capital and hence, a higher opportunity cost of time, this schooling variable ought to be negatively related to parity.

D. Estimation

This section describes the various estimation problems germane to this study. In particular, issues such as simultaneous equation bias, identification, the appropriate functional form, and the proper weights to correct for the heteroscedasticity common to aggregation are each discussed.

Following Rosenzweig and Schultz (1982,1983a,1983b) it is anticipated that the residuals in the structural equations are correlated with the health inputs. This expectation is based on the assumption that individuals have some information concerning their genetic health endowment which is unobservable to the researcher but which causes the parents to alter their behavior with respect to their choice of inputs. As a result, ordinary least squares estimates are biased and inconsistent. I make the assumption that the income and health input availability measures are uncorrelated with the disturbance term in the structural equation in order to be able to use them as instruments in a two-stage least squares estimation procedure.

To test whether a significant correlation between the production function residuals and the health inputs does in fact exist, Wu's T_2 statistic (Wu, 1973) as described by Nakamura and Nakamura (1981) is be applied.[8] If the null hypothesis of zero correlation between the error term and the regressors is rejected, then two-stage least squares will be used to estimate the birth weight and neonatal mortality equations. To increase efficiency, the two structural equations will be estimated as a system of simultaneous equations allowing shocks which impact on birth weight to affect mortality as well. In essence, this is a quasi-three-stage least squares (3SLS) procedure because information across the input demand equations is not included due to severe multicolinearity in the full system.

The dependent variable in the neonatal mortality and the low-birth weight equations ranges between zero and one. Moreover, there may be a nonlinear relationship between the probability of survival and the health inputs. Consequently, a logistic function is estimated as well as a linear specification for each of the structural equations. A Cobb-Douglas specification is also fitted following the work of Rosenzweig and Schultz (1983b).

In the case of the logistic function, the equation to be es-

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[8] A more detailed description of the Wu test can be found in Appendix B.

timated can be written as a log of the odds ratio

$$\log((p_i/(1-p_i)) = B'X_i + u_i \qquad (6)$$

where p_i is the probability of an infant dying in the first 27 days of life in county i; X_i is a set of regressors; B is a vector of coefficients and u_i is the disturbance term. P_i can be written as follows:

$$P_i = d_i/b_i \qquad (7)$$

d_i is the number of neonatal deaths within county i and b_i is the total number of births. Maddala (1983) shows that as long as b_i is large then the variance of u_i can be expressed as

$$1/(b_i p_i(1-p_i)) \qquad (8)$$

Hence, B in equation (6) can be estimated by weighted least squares where the appropiate weight is

$$(b_i(p_i(1-p_i))^{1/2} \qquad (9)$$

This method is termed the minimum chi-square method.

The linear specification is treated as a linear probability model on grouped data. Again, following Maddala (1983), weighted least squares can be used to obtain estimates of B. The weights in this case are as follows:[9]

$$(b_i/(p_i(1-p_i))^{1/2} \qquad (10)$$

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[9] The weights for the Cobb-Douglas equation are similar and can be found in Maddala (1983).

To explicitly trace out the actual equations that are estimated and to verify that identification is achieved when the structural equations are estimated simultaneously, the model can be written in a general functional form as follows:

NMR* = f_1(FPTEEN*,ABOR,NICU*,PRECARE*,LBW*,LPOPDEN) (11)

LBW* = f_2(FPTEEN*,ABOR,SMOK,PRECARE*,YOUNG*,ILEGIT*, PARITY*,GEST*,LPOPDEN) (12)

FPTEEN*,ABOR,NICU*,PRECARE*,YOUNG*,PARITY*) = f_i(POV*,HSED*, MPA,MPN,MPU,MNEW, MBEN,FPCLIN,ABORPR,NEOH, BCHSP,AFDC,LPOPDEN) (13)-(18)

In the first stage, the health inputs are regressed on a set of exogenous price, income and taste measures [equations (13)-(18)] using OLS. The predicted values are then inserted into the appropriate structural equation. This two-stage or instrumental variable procedure provides consistent and unbiased estimates of the structural coefficients. A third step is added whereby the equations (11) and (12) are estimated jointly allowing the covariance between the residuals in each equations to increase the efficiency of the estimates.

If viewed in the context of an expanded structural model, identification is achieved by the exclusion of smoking, gestation, and the endogenous risk factors from the neonatal mortality equation while the neonatal intensive measure, and the endogenous risk factors are restricted to have no impact on birth weight holding gestation constant. Restricting smoking to the birth weight equation is justified by the epidemiological literature linking the effects of smoking during pregnancy to an increased probability of a low-birth weight infant as discussed above. The use of neonatal intensive care could never be a determinant of birth weight since such care is only administered after an infant is born. As described in the text, the above average incidence of low-birth weight among births to teenage and unwed mothers is due primarily to preterm delivery. Therefore, the risk factors are modeled to impact on birth weight by shortening gestation. This restriction is examined by the estimation of "quasi-structural" equations which are described in Appendix D.

A few issues remain to be clarified. Ideally both gestational age and illegitimacy should be treated as endogenous variables. As is discussed above gestational age had a number of missing observations. The same is true for legitimacy status which is not reported in 12 states. In both cases, as described in Appendix A, an instrumental variable procedure was used to obtain predicted values for the missing observations. The instruments utilized were from the reduced form set of variables. Consequently, they could not be used again to "repredict" these inputs. As a result, the coefficient on legitimacy status may understate its true impact. The smoking variable, as mentioned above, was already a predicted value obtained from a study done by Lewit and Coate (1982). As such, it is assumed to be uncorrelated with the residuals.

CHAPTER IV -- Results

A. Input Demand Equations

Tables 3 and 4 present the results from the first-stage input demand equations. The relationship between input use and input availability is well-supported by the data. Increases in family planning services, abortion providers and neonatal intensive care (NIC) hospitals are strongly associated with increased family planning use by teenagers, the abortion rate and the number of inpatient days in a NIC unit respectively. In short, the own shadow price effects are as hypothesized. This is a noteworthy result for it reinforces the notion that accessibility to medical services, the fees for which are often covered by insurance, plays a major role in the utilization of these resources.

Moreover, the use of NIC by black infants is much more sensitive to the availability of a NIC hospital than it is for whites. In particular, a one percent increase in the number of hospitals with a NIC unit per woman age 15 to 44 results in a .7 of a percent increase in the number of inpatient days per birth in a NIC unit. The corresponding white elasticity is .12.

Other noticeable differences between races are the effect of schooling on the demand for contraceptive services and abortion. In the former case, counties with a relatively large proportion of women 15 to 49 with at least a high school education is strongly and positively associated with the utilization of family planning services by teenagers. For blacks, however, education evidences little relationship

Table 3

Ordinary Least Squares Input Demand Equations--Whites[a]

Independent Variables	Prenatal Care	Abortion Rate	Teenage Family Planning users	Neonatal Intensive Care	Births to Teens	Parity	Low-Birth Weight
ABORPR	-17.807	62.343	40.978	1.590	5.628	-7.894	.640
	(-2.90)	(11.14)	(7.96)	(4.39)	(2.18)	(-4.77)	(1.00)
FPCLINxPOV*	3.993	4.971	34.653	-.462	-3.586	1.300	-.529
	(.82)	(1.11)	(8.42)	(-1.60)	(-1.74)	(.98)	(-1.03)
BCHSPxPOV*	-42.203	-25.907	90.424	1.660	-8.114	-6.215	10.032
	(-1.77)	(-1.18)	(4.46)	(1.16)	(-.80)	(-.95)	(3.98)
NEOH	67.518	-907.918	224.241	7.347	33.566	170.927	6.791
	(.91)	(-13.31)	(3.57)	(1.67)	(1.07)	(8.47)	(.87)
MPAxPOV*	.022	20.572	.066	.335	4.009	3.228	-.209
	(.01)	(7.98)	(.03)	(2.01)	(3.38)	(4.24)	(-.71)
MPUxPOV*	13.153	-13.796	.927	.807	.264	-3.511	1.066
	(3.98)	(-4.52)	(.33)	(4.09)	(.19)	(-3.89)	(3.05)
MPNxPOV*	-.342	1.272	5.556	.886	6.771	-2.009	1.117
	(-.12)	(.46)	(2.19)	(4.97)	(5.33)	(-2.46)	(3.54)
MNEWxPOV*	9.476	11.527	-15.228	-.522	.472	-1.007	.192
	(2.56)	(3.37)	(-4.84)	(-2.36)	(.30)	(-1.00)	(.49)
MBENxPOV*	-.028	.057	.013	.002	-.001	-.001	.002
	(-4.06)	(9.27)	(2.19)	(5.03)	(-.41)	(-.70)	(2.57)
HSED*	.246	-.053	.117	.001	-.236	-.069	-.021
	(5.84)	(-1.36)	(3.26)	(.43)	(-13.18)	(-6.01)	(-4.66)
POV*	-.487	-.473	.088	-.010	.090	.098	.003
	(-8.57)	(-9.01)	(1.83)	(-2.93)	(3.71)	(6.32)	(.54)
AFDC	.022	.001	.007	-.0004	-.054	.015	-.005
	(1.29)	(.04)	(.48)	(-.38)	(-7.44)	(3.26)	(-2.98)
POPDEN	-.781	.367	.341	.030	-.455	.137	.153
	(-4.25)	(2.17)	(2.19)	(-2.72)	(-5.83)	(2.74)	(7.91)
CONSTANT	80.115	32.706	-8.914	.707	32.250	2.588	6.182
	(23.56)	(10.41)	(-3.08)	(3.48)	(22.30)	(2.78)	(17.21)
F	47.07	78.95	27.10	7.72	77.27	35.16	18.81
R^2	.47	.60	.33	.11	.59	.40	.26
Sample size	677	677	677	677	677	677	677

[a]The t-ratios are in parentheses. The critical t-ratios at the 5 percent level are 1.64 for a one-tailed test and 1.96 for a two-tailed test. The F-ratio associated with each regression is significant at the 1 percent level. The goodness of fit measure is an adjusted R^2.

Table 4

Ordinary Least Squares Input Demand Equations--Blacks[a]

Independent Variables	Prenatal Care	Abortion Rate	Teenage Family Planning users	Neonatal Intensive Care	Births to Teens	Parity	Low-Birth Weight
ABORPR	10.768	31.025	1.870	5.036	.925	-12.230	1.622
	(.70)	(3.77)	(.13)	(3.00)	(.16)	(-4.25)	(.87)
FPCLINxPOV*	6.278	5.735	16.170	-.104	2.194	-1.150	.746
	(1.51)	(2.56)	(4.06)	(-.23)	(1.42)	(-1.47)	(1.47)
BCHSPxPOV*	39.430	-20.706	73.696	-.194	-14.115	9.575	-2.907
	(1.49)	(-1.46)	(2.92)	(-.07)	(-1.44)	(1.93)	(-.90)
NEOH	95.510	-1102.650	-306.200	106.730	87.518	39.843	37.800
	(2.51)	(-10.40)	(-1.62)	(4.93)	(1.20)	(1.07)	(1.57)
MPAxPOV*	-4.480	12.625	1.043	-.155	-7.338	-3.093	-1.876
	(-1.20)	(6.28)	(.29)	(-.38)	(-5.30)	(-4.40)	(-4.12)
MPUxPOV*	5.504	-11.367	1.514	.478	-5.257	.409	-.331
	(1.99)	(-7.66)	(.57)	(1.58)	(-5.14)	(.79)	(-.98)
MPNxPOV*	9.176	-3.332	-8.592	1.734	1.466	-3.998	.362
	(2.83)	(-1.91)	(-2.77)	(4.86)	(1.22)	(-6.55)	(.92)
MNEWxPOV*	-15.342	1.924	.041	-1.152	-.699	2.349	.574
	(-3.67)	(.86)	(.01)	(-2.51)	(-.45)	(2.99)	(1.13)
MBENxPOV*	-.034	.034	-.018	.001	-.008	-.003	.001
	(-4.15)	(7.58)	(-2.32)	(.96)	(-2.79)	(-1.84)	(1.20)
HSED*	.320	.189	-.013	-.010	-.156	-.066	-.047
	(3.38)	(3.70)	(-.14)	(-.94)	(-4.43)	(-3.70)	(-4.08)
POV*	-.098	-.166	.103	.001	.213	.055	.014
	(-1.02)	(-3.21)	(1.12)	(.11)	(5.97)	(3.03)	(1.20)
AFDC	.051	-.037	-.125	.016	.028	.032	.007
	(1.17)	(-1.58)	(-3.02)	(3.36)	(1.76)	(3.87)	(1.42)
POPDEN	-1.552	-.041	1.391	-.068	-.406	-.006	.421
	(-4.17)	(-.21)	(3.91)	(-1.66)	(-2.95)	(-.09)	(9.28)
CONSTANT	67.625	27.704	21.802	.186	28.073	5.341	9.827
	(7.77)	(5.92)	(2.62)	(.20)	(8.72)	(3.26)	(9.26)
F	14.92	64.76	10.20	6.54	32.94	20.00	14.06
R^2	.34	.70	.25	.17	.54	.41	.32
Sample size	357	357	357	357	357	357	357

[a]The t-ratios are in parentheses. The critical t-ratios at the 5 percent level are 1.64 for a one-tailed test and 1.96 for a two-tailed test. The F-ratio associated with each regression is significant at the 1 percent level. The goodness of fit measure is an adjusted R^2.

to the consumption of family planning services by adolsecents. For abortion, the results are practically reversed. Higher levels of educational achievement by nonwhite women is positively and significantly related to the use of abortion. For whites, on the other hand, education is negatively related to abortion.

Schooling plays a significant role in the demand for prenatal care by blacks and whites. For example, a one standard deviation increase in the percentage of white women with at least a high school education increases the percentage of women who begin prenatal care in the first trimester by 1.8 percentage points.[1] In the case of blacks, the analogous change brings about a 2.5 percentage point increase in the percentage of black women who initiate care in the first trimester. This is an important finding that will be discussed further when the effect of prenatal care on birth outcomes is examined in more detail below.

As expected, the fertility measures, the percentage of fifth and and higher order births and the percentage of births to teenagers, are negatively related to schooling and positively correlated with poverty. In addition, the more generous the average AFDC payment per recipient, the greater the proportion of births to black teenagers, and fifth and higher order births. For whites, parity also has a strong and positive relationship to relatively generous welfare pay-

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[1] Beta coefficients are used in place of elasticities when the independent variable is measured as a percentage. As explained in Corman, Joyce and Grossman (1984), even though the slope coefficient is the same in absolute value when, for example, the percentage of women with at least a high school education (p_{hs}) is used instead of its complement ($1-p_{hs}$), the elasticities will differ (except when $p_{hs}=.50$).

ments, but the proportion of births to teenagers responds negatively to the AFDC measure. Also of note is that the number of abortion providers per woman age 15 to 44 has a substantial and significant impact on the black and white percentage of fifth and higher order births. This supports the link between increases in abortion and declines in fertility reported elsewhere (Sklar and Berkov, 1974; Bauman et al., 1977). The effect of family planning clinics on the fertility measures is mixed. FPCLIN is negatively associated with births to white teenagers and higher order births to black women.

Generous Medicaid benefits, as measured by counties that cover all first-time pregnancies (MPA) and average benefits per adult (MBEN) have very a significant relationship to the abortion rate. The magnitute of the t-ratios should be viewed with caution since there is substantial multicolinearity among the five Medicaid measures. Nevertheless, the null hypothesis that the coefficients of all five program measures is zero is rejected at the .01 level in all fourteen input demand equations.

Finally, there is evidence that the increased availabilty of contraception results in a greater proportion of women obtaining prenatal care in the first trimester of pregnancy. Some caution is advised in interpreting this result since some family planning facilities also serve as community health centers that offer a range of medical services including prenatal care. Hence, increased availability of family planning may in part represent easier access to prenatal care and not a decline in the cost of contraception. Nevertheless, the result merits further attention.

B. Birth Outcome Production Functions

Estimates for the linear white and black neonatal mortality and low-birth weight production functions are presented in Tables 5 through 8. Comparison of the results of equations estimated by ordinary least squares (OLS) and two-stage least squares (2SLS) generally confirms the bias present when heterogeneity is not corrected.[2] The coefficient of prenatal care increases by 73 percent when 2SLS is used instead of OLS as shown by regressions 5A and 5B and by 92 percent when regressions 6A and 6B are compared. The change in the abortion coefficient is not as substantial when comparing OLS and 2SLS over the same specifications as prenatal care. Heterogeneity bias appears to be most prominent with neonatal intensive care in both the black and white regressions. The 2SLS estimates are at least four times as large as their corresponding OLS estimates as seen by comparing regressions 5A to 5B, 5C to 5D, 6A to 6B and 6C to 6D.

The same pattern holds for the birth weight production functions. The coefficients of prenatal care and abortion are smaller in absolute value when estimated by OLS (Tables 7 and 8). The coefficients of teenage family planning use follow this same pattern in the black equations while for whites the changes are less consistent. In

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[2] Heterogeneity bias, as described in Chapters II and III, refers to the fact that each individual has a unique genetic birthing component that may play a major role in determining the health of a newborn. This endowed health may be known by parents who use this information, or expectation, to alter their demand for health inputs. This may generate a correlation between the health inputs and the residuals that could cause the estimates to be biased and inconsistent. The bias generated in this manner is labeled heterogeneity bias by Rosenzweig and Schultz (1982, 1983a,1983b) and is so referred to in this study.

Table 5

Linear Neonatal Mortality Rate Production Functions--Whites[a]

Independent Variables	OLS (5A)	2SLS (5B)	OLS (5C)	2SLS (5D)	3SLS (5E)
Teenage family planning*[b]	-.028	-.059	-.031	-.048	-.049
	(-3.01)	(-2.97)	(-3.47)	(-2.43)	(-2.44)
Abortion rate[b]	-.038	-.043	-.026	-.017	-.033
	(-5.36)	(-4.40)	(-3.77)	(-1.28)	(-2.70)
Prenatal care*[b]	-.037	-.064	-.020	-.019	-.023
	(-5.11)	(-5.68)	(-2.74)	(-.99)	(-1.22)
Neonatal intensive care*[b]	.020	-.163	-.132	-.964	-.619
	(.13)	(-.30)	(-.89)	(-1.62)	(-1.02)
Low birth weight*[b]			.625	.830	.876
			(7.71)	(2.79)	(2.90)
Ln population density	.262	.270	.188	.139	.103
	(6.46)	(6.02)	(4.67)	(2.18)	(1.65)
Constant	10.995	13.601	6.252	5.740	6.426
	(16.48)	(14.23)	(7.04)	(1.93)	(2.16)
F	15.73	17.14	24.16	16.43	
R^2	.098		.170		
Sample Size	677	677	677	677	677
Wu's T_2,F=		4.447		2.176[c]	

[a]Asymptotic t-ratios in parentheses. The critical asymptotic t-ratios a the 5 percent level are 1.64 for one-tailed and 1.96 for a two-tailed test. In this table and the others that contain regression results, the F-ratio associated with each regression is significant at the 1 percent level unless otherwise indicated.

[b]Endogenous

[c]Not significant (p>.05)

Table 6

Linear Neonatal Mortality Rate Production Functions--Blacks[a]

Independent Variables	OLS (6A)	2SLS (6B)	OLS (6C)	2SLS (6D)	3SLS (6E)
Teenage family planning*[b]	-.020	-.180	-.026	-.204	-.189
	(-1.10)	(-3.35)	(-1.51)	(-3.60)	(-3.63)
Abortion rate[b]	-.102	-.190	-.056	-.110	-.090
	(-4.43)	(-4.98)	(-2.54)	(-2.10)	(-1.83)
Prenatal care*[b]	-.004	-.033	.005	.066	.064
	(-.27)	(-.91)	(.36)	(1.18)	(1.25)
Neonatal intensive care*[b]	-.353	-1.430	-.410	-1.986	-1.809
	(-2.19)	(-2.56)	(-2.72)	(-3.17)	(-2.92)
Low birth weight*[b]			.996	1.384	1.405
			(7.42)	(2.34)	(2.56)
Ln population density	.730	.876	.392	.477	.222
	(6.21)	(5.71)	(3.31)	(2.04)	(1.00)
Constant	14.241	22.590	2.330	1.070	2.194
	(9.27)	(7.59)	(1.08)	(.11)	(.24)
F	9.42	10.26	18.24	8.86	
R^2	.100		.220		
Sample Size	357	357	357	357	357
Wu's T_2,F=		6.446		4.652	

[a]See footnote a to Table 5

[b]Endogenous

Table 7

Linear Low-Birth Weight Production Functions--Whites[a]

Independent Variables	OLS (7A)	2SLS (7B)	OLS (7C)	2SLS (7D)	3SLS (7E)
Teenage family planning*[b]	.006	-.001	-.005	-.004	-.005
	(1.36)	(-.11)	(-1.32)	(-.60)	(-.70)
Abortion rate[b]	-.015	-.021	-.010	-.012	-.012
	(-4.90)	(-4.93)	(-3.57)	(-2.83)	(-2.93)
Prenatal care*[b]	-.025	-.047	-.006	-.018	-.018
	(-7.87)	(-9.75)	(-1.84)	(-1.73)	(-1.75)
Cigarettes[b]	.188	.190	-.185	.002	.016
	(3.91)	(3.79)	(-3.65)	(.03)	(.34)
Births to teenagers*[b]			.021	.033	.038
			(2.66)	(1.88)	(1.61)
Illegitimate births*			.044	.048	.052
			(4.08)	(4.43)	(4.80)
Parity*[b]			-.115	-.103	-.103
			(-8.53)	(-3.82)	(-3.85)
Gestation*			.421	.337	.352
			(12.34)	(9.80)	(10.29)
Ln population density	.121	.134	.091	.112	.105
	(6.85)	(7.04)	(5.06)	(5.03)	(4.73)
Constant	6.039	7.926	4.203	3.996	3.882
	(12.80)	(13.55)	(6.99)	(3.15)	(3.08)
F	25.36	30.43	52.42	42.76	
R^2	.153		.406		
Sample Size	677	677	677	677	677
Wu's T_2,F=		11.753		3.975	

[a]See footnote a to Table 5

[b]Endogenous

Table 8

Linear Low-Birth Weight Production Functions--Blacks[a]

Independent Variables	OLS (8A)	2SLS (8B)	OLS (8C)	2SLS (8D)	3SLS (8E)
Teenage family planning*[b]	.007	-.002	.003	-.011	-.012
	(1.06)	(-.11)	(.55)	(-.92)	(-.93)
Abortion rate[b]	-.040	-.052	.005	-.011	-.009
	(-4.33)	(-3.72)	(.63)	(-.71)	(-.58)
Prenatal care*[b]	-.010	-.060	.008	-.023	-.021
	(-1.66)	(-5.51)	(1.57)	(-1.44)	(-1.29)
Cigarettes[b]	.213	.372	.236	.312	.328
	(1.10)	(1.80)	(1.42)	(1.93)	(2.04)
Births to teenagers*[b]			.069	.029	.024
			(3.78)	(1.00)	(.83)
Illegitimate births*			.021	.044	.045
			(2.05)	(5.31)	(5.48)
Parity*[b]			-.012	-.110	-.099
			(-.45)	(-1.39)	(-1.26)
Gestation*			.280	.271	.285
			(8.51)	(7.35)	(7.76)
Ln population density	.342	.316	.368	.247	.243
	(7.92)	(5.96)	(7.51)	(4.42)	(4.36)
Constant	10.246	12.795	.407	4.079	3.593
	(6.27)	(6.75)	(.24)	(1.61)	(1.42)
F	15.83	19.36	38.28	32.41	
R^2	.172		.485		
Sample Size	357	357	357	357	357
Wu's T_2, F=		13.951		1.061[c]	

[a]See footnote a to Table 5

[b]Endogenous

[c]Not significant (p>.05)

short, this initial review of the results tends to support the contention by Rosenzweig and Schultz (1982,1983a,1983b) that analysis of the effect of health inputs on birth outcomes by direct correlational methods underestimates their true impact.

However, closer inspection reveals that the difference between the coefficients estimated by OLS and 2SLS narrows in many instances as the specification becomes more complete. For example, when birth weight is included in the neonatal mortality equation, there is no difference between the OLS and 2SLS estimates of prenatal care in regressions 5C and 5D. The abortion coefficient actually declines in absolute value between these same equations. In the birth weight production functions, the abortion and family planning coefficients evidence more change in the specifications that exclude the risk factors when comparing OLS and 2SLS. This suggests, perhaps, that what Rosenzweig and Schultz (1982,1983a,1983b) claim is heterogeneity bias may in fact be omitted variable bias.

Their own results reveal instances of the same pattern. In particular, the coefficient of prenatal care in the birth weight production function (1982) that does not control for gestation increases by a factor of 25 when 2SLS is applied instead of OLS. In the same specification, but with gestation held constant, the 2SLS estimate is only 1.6 times as large in absolute value as the corresponding OLS estimate.

To formally test for the presence of heterogeneity bias Rosenzweig and Schultz (1983b) use a Wu statistic (1973) to determine whether the regressors in the Cobb-Douglas specification estimated by OLS are correlated with the disturbance term. In both the birth weight and the birth weight standarized for gestation equations the null hypothesis of zero correlation between the regressors and the residuals is rejected at the .01 level in the former case and at the .05 level in the latter. This test, coupled with the seemingly downward bias of the OLS estimates, leads the authors to conclude that heterogeneity bias is present and must be corrected.

Rosenzweig and Schultz also present results using a translog function to estimate the two birth outcome equations. In this case, there is no systematic tendency for the coefficients estimated by OLS to underpredict the impact of the health inputs. Nevertheless, the Wu statistic for each birth outcome exceeds its respective critical value at the .01 level by very wide margins, indicating substantial correlation between the regressors and the residuals when the translog specification is estimated by OLS. However, the authors use an F-test to reject the translog specification in favor of the nested Cobb-Douglas form. In other words, it is not clear whether the Wu test is picking-up a correlation between the right-hand-side variables and the disturbance term due to heterogeneity, as the authors conclude, or whether the correlation is due to missing variable bias or an incorrect functional form. As Judge et al. (1985) note, specification tests such as those by Durbin (1954), Wu (1973), or Hausman[3] (1978) can detect regressors that are not orthogonal to the residuals, but are essentially unable to say anything about the exact cause.

Results from this study using a linear specification suggest that

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[3] See Nakamura and Nakamura (1981) for the similarity among these three specification tests.

missing variable bias may be generating the correlation between the right-hand-side variables and the disturbance term. Tables 5 through 8 show the results from applying the Wu T_2 statistic as described by Nakamura and Nakamura (1981) to test whether the orthogonality assumption is violated when OLS is used to estimate the neonatal mortality and birth weight production functions. As is evident from the figures, the magnitude of the Wu statistic falls as the specification is expanded to include the relevent risk factors and intermediate birth outcomes. In fact, in two of the four completely specified equations, the null hypothesis of zero correlation between the regressors and the residuals cannot be rejected.

Nothing definitive can be concluded from this discussion; the results are obviously mixed. Heterogeneity remains a plausible hypothesis for explaining the non-orthogonality between the regressors and the residuals. For example, the OLS estimates of the neonatal intensive care use coefficient are consistently smaller in absolute value than the corresponding 2SLS estimates. It is reasonable to hypothesize that the advances in perinatal care have enabled physicians to more effectively predict, prepare for, and manage a problematic birth. Nevertheless, as the results from the Wu test suggest, there may be other reasons for the correlation between the health inputs and the disturbance term besides heterogeneity bias. However, since the results of this study are based on aggregate data, there is always the possiblity that conclusions, or suggestions, generated at this level are not applicable at the micro level. This caveat aside, these results do suggest that a greater effort be made to more completely specify the health production function at issue before pronouncements are made as to the definitive presence of heterogeneity bias.

The effect of abortion on birth outcomes differs somewhat by race. For both blacks and whites the coefficient of abortion in the neonatal mortality equation is negative and significant ($p<.0001$) as displayed in regressions 5B and 6B. Yet, when the percentage of low-birth weight births is held constant, the effect of abortion falls for both races. In the case of blacks the coefficient remains significant ($p<.04$), but in the white specification, the null hypothesis cannot be rejected. This result is tempered somewhat by the 3SLS estimate which is not only larger in absolute value than the 2SLS coefficient, but is significant as well ($p<.01$). Moreover, the OLS estimate is also greater than the 2SLS one which further suggests that the variability of the coefficient across the three estimating procedures may be due to multicolinearity.

The black results imply that abortion operates in part to lower the percentage of low-birth weight births. Examination of the estimated birth weight production function (regression 8B) confirms just this. However, in specification 8D, which includes the full set of risk factors, the abortion coefficient is essentially zero. In other words, for blacks, abortion improves the distribution of low-birth weight births by lowering the incidence of prematurity. A step-wise presentation of risk factors is given in Tables C-1 and C-2 in Appendix C. Of note is the dramatic drop in the magnitude and the significance of the abortion coefficient when the the percentage of births to teenagers is included. In short, abortion operates on birth weight by reducing the fraction of births to adolescents. This is consistent with the fact that in 1977, 31 percent of all abortions were performed on teenagers (Henshaw and O'Reilly, 1983). This analysis can be taken one step further by incorporating the results from regression 8D. These estimates suggest that infants born to teenagers have a greater risk of being delivered preterm, which accounts for increased likelihood of a low-birth weight birth.

The results for whites with respect to abortion follow much the same pattern as do the black results. In particular, the inclusion of births to teenagers in the birth weight equation causes the abortion coefficient to drop by 62 percent (see Table C-1). Where the races differ is that abortion has a significant impact on the birth weight of white infants (equation 7D) holding the full set of risk factors constant. Corman, Joyce, and Grossman (1984) obtain qualitatively the same result from which they argue that the use of abortion by whites lowers the percentage of full-term light births whereas abortion use by blacks lowers the percentage of premature light births. Because the former case is associated with life-long health problems (Beck and van der Berg, 1975), they conclude that white women have a greater tendency to identify and abort defective fetuses than do blacks. One indication of this comes from the growing data on the use of amniocentesis. A recent analysis found that the application of prenatal cytogenetic diagnosis is positive and significantly associated with income and to a lesser extent education (Hook et al., 1981). Since whites, on average, have higher levels of education and income, it seems reasonable to assume that white women make use of this procedure more often than blacks.

A related explanation for the significance of abortion in the black neonatal mortality equation and the white birth weight equation is that the abortion rate is a proxy for the "wantedness" of a birth as discussed in Chapter II. The more abortions that are performed in a county, the fewer unwanted births, and the lower the birth rate should be for unintended pregnancies. Furthermore, if on average, births which are better planned (more "wanted") occur at a time when a woman is emotionally and financially more prepared to have a child, then a pregnant woman may receive more of the unmeasurable factors (better nutrition, better quality care, less stress) that improve the survivability of an infant.

The teenage family planning variable presents some anomolies. In the black and white neonatal mortality equation its coefficient becomes more negative and significant (Tables 5 and 6) when the percentage of low-birth weight births is held constant. If family planning effectively helps teenagers avoid pregnancy and hence unwanted births, then controlling for this dominant risk factor should diminish its effect. A closer look at the operation of family planning clinics, however, offers some insight into this discrepancy.

In a comparison of counties that served a high proportion of teenagers at risk of pregnancy (75 percent on average) than those that serve a much smaller percentage (28 percent on average), Chamie et al. (1982) noted major differences between the delivery of services. Using counties that shared the same economic and demographic characteristics, the authors found that counties that served a high proportion of adolescents at risk were more likely to provide additional services such as prenatal care, abortion and other gynecological care than counties that served a smaller proportion of teenagers at risk. In addition, the family planning clinics in the well-served counties tended to have greater continuity of care with their clients.

Other researchers in this field have documented that the reason most often given by teenagers for going to a family planning clinic is the fear that they are pregnant (Zabin and Clark, 1981). Furthermore, in a large metropolitan clinic more than half of the first-time adolescent clients had previously been pregnant (Jones et al., 1982). In sum, a higher proportion of family planning users may be indicative of a population that has been integrated into a network of prenatal and perinatal care. The births to these young women may still be problematic (i.e. premature or low-birth weight), but with better support and care their children are more likely to survive.

An additional explanation is that teenage family planning use may be a proxy, in part, for abortions by teenagers. Jones et al. (1982) note that of the young women who became pregnant while attending the clinic under study, 61 percent requested an abortion. In other words, teenagers who are more likely to seek out contraception, may also have a greater probability of aborting an unintended pregnancy.

An important result is the magnitude and the significance of the neonatal intensive care (NIC) use variable in the black neonatal mortality equation (regressions 6B and 6D). As argued in Chapter III, interacting the NIC inpatient days with the percentage of low-birth weight births is the only means[4] by which the differential effects of

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[4] As noted in Chapter III, the best way to pick-up the birth weight-specific effect of NIC use is to specify separate equations for low-

this technology measure on the fraction of light births as opposed to normal weight births can be captured. As anticipated, the NIC coefficient increases by 39 percent when the percentage of low-birth weight births is added. In the case of whites, the coefficient increases in absolute value by a factor of almost 6 when birth weight is included in the specification. However, its coefficient is half the size of the corresponding black one and its significance is borderline at best.

The association between prenatal care and neonatal mortality is in general aggrement with the literature. When birth weight is held constant (regressions 5D and 6D) the effect of prenatal care drops dramatically, becoming positive for blacks while remaining negative but insignificant for whites. Recent studies by Lewit (1983) and Showstack et al. (1984) have emphasized the important effect of prenatal care on birth weight holding gestational age constant. Results from equations 7D and 8D offer less than convincing support for their findings.

These findings also differ from those of Corman, Joyce and Grossman (1984) who found that prenatal care had a significant effect on birth weight despite controlling for prematurity. Their specification makes the assumption that the effects of births to teenagers, illegititmate births and fifth and higher order births on birth weight operate through gestation. Equations 7D and 8D indicate that the independent impact of the aforementioned risk factors, especially illegitimate births, on full-term light babies makes such an assump-

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birth weight and normal weight newborns. Given the lack of data on birth weight-specific mortality rates by county, the interactive procedure attempts to achieve the same result.

tion problematic.[5]

Of the three risk factors, legitimacy status is the most stable and significant predictor of low-birth weight. This is true for both races even when gestational age is held constant (regressions 7D and 8D). It is unlikely that marital status impacts on fetal growth directly. Therefore, tracing the etiological path from legitimacy status to birth outcomes remains a speculative exercise especially in an ecologocial study such as this. Nevertheless, a number of variables, for which data is not available at the county level, may be operating through marital status. For example, illegitimacy is strongly associated with social class, which in turn may be related to poor diet, stress and a lack of resources to cope with it, as well as drug and alcohol use (Bakketeig et al., 1985).

Births to teenagers have long been associated with prematurity (Taffel, 1980; Chase et al., 1972). The more salient issue, however, is whether the risks are medical or social. Recent work (Zukerman et al., 1983; Elster, 1984) has pointed out that when confounding variables such as inadequate prenatal care and the consumption of cigarettes, alcohol and drugs are held constant, the effect of maternal age on birth weight is insigificant. Furthermore, in 1977 over 80 percent of all births to black teenagers were to unmarried mothers. It is not surprising, therefore, that the introduction of marital status in a birth weight regression reduces the coefficient of births to

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[5] Corman, Joyce and Grossman do attempt to control for illegitimacy by including the percentage of poor women ages 15 to 44 on AFDC. This variable increases the explanatory power of their black birth weight equation substantially, but it has little effect on the coefficient of prenatal care.

black adolescents by 57 percent (Table C-2). Moreover, the impact of births to teenagers on birth weight falls still more with the inclusion of gestational age whereas legitimacy status does not decline at all (regressions 7D and 8D).

Without estimating a gestational age production function, the possibility that births to adolescents has an independent effect on prematurity cannot be eliminated. However, the finding that marital status tends to dominate births to young mothers suggests that the younger side of the maternal age spectrum represents a social problem as opposed to a biological one.

The percentage of fifth and higher order births is negatively related to low-birth weight in the black and white production functions, but is significant in the case of whites only (regressions 7C, 7D, 8C and 8D). The results support the findings of Rosenzweig and Schultz (1982,1983a,1983b) who obtained a positive effect of parity on birth weight using individual data. The data from this study imply that mothers of large families, in which the mother's age is unknown, may be genetically well-endowed. Such women, therefore, may be self-selected in that they desire, and can achieve large families. Attempts to capture the effects of poor spacing by interacting birth rates with maternal age proved unsuccessful due to severe multicolinearity. In short, unless aggregate data on birth order is categorized by age, the percentage of fifth and higher order births seems an inadequate proxy for areas where high fertility is indicative of poor spacing at young ages.

Finally, the natural logarithm of population density is positive and significant for both races and both birth outcomes in all specifications except the 3SLS estimates of neonatal mortality. It is clear, therefore, that poor birth outcomes are related to urbanization. Whether this is due to pollution or stress, for example, is impossible to say. However, it is noteworthy that the inclusion of the endogenous risk factors, births to unwed mothers and teenagers as well as fifth and higher order births, have no impact on the coefficient of population density. This implies that the effect of urbanization on birth outcomes is probably not related to socioeconomic status as captured by the risk factors, but rather is assocated with more exogneous factors such as pollution or stress.

To gauge the magnitude of the estimated relationships between infant health inputs and outcomes, the impact of a one standard deviation increase in each input on the race-specific neonatal mortality rate is shown in Table 9. The magnitude of the effects are summarized in this manner to highlight the cross-sectional variablility in the inputs. The direct, the indirect, and the total effect of each input are computed. The direct effect is obtained from the neonatal mortality rate production function that includes birth weight as a regressor. The indirect effect of any input (x) is its regression coefficient in the birth weight equation multiplied by the birth weight coefficient in the neonatal mortality regression, all multiplied by the standard deviation of x. Thus, the indirect effect shows the reduction in the neonatal mortality rate due to a one standard deviation increase in x that operates via a reduction in the percentage of low-birth weight births. The total effect is the sum of the

Table 9

The Effect of One Standard Deviation Increase in Inputs on Neonatal Mortality Rate[a]

Input	Panel A: Whites[b] Direct	Indirect	Total	Panel B: Blacks[c] Direct	Indirect	Total
Teenage family planning	.178	.006	.184	1.059	.100	1.159
Abortion rate	.117	.057	.174	.809	.387	1.196
Prenatal care	.109	.100	.209	-.433	.418	-.015
Neonatal intensive care	.129	---	.129	.949	---	.949
Smoking	---	-.012	-.012	---	-.258	-.258
Births to teenagers	---	-.234	-.234	---	-.290	-.290
Illegitimate births	---	-.111	-.111	---	-.637	-.637
Parity	---	.101	.101	---	.267	.267

[a] Reduction in deaths per 1,000 live births. Negative sign denotes a predicted increase

[b] The direct effect is from regression 5D. The indirect effect is from regression A-1D. Total effect equals the sum of the direct and indirect effects.

[c] The direct effect is from regression 6D. The indirect effect is from regression A-2D. Total effect equals the sum of the direct and indirect effects.

direct and indirect effects.[6]

For whites, a one standard deviation increase in the four basic inputs, teenage family planning, abortion, prenatal care, and neonatal intensive care, causes the mortality rate to fall by .7 of a death per 1000 live births. This accounts for 8 percent of the mean white neonatal mortality rate (8.5 deaths per 1000 live births). Teenage family planning, abortion, and prenatal care all contribute about equally to the decline in neonatal mortality. The impact of the risk factors implies that a one standard deviation decrease in births to teenagers, out-of-wedlock births, and per capita smoking, plus a one standard deviation increase in fifth and higher order births results in a decline of .5 of a death per 1000 live births. All told, therefore, a one standard deviation change in the appropriate direction in the health inputs and risk factors combined would result in a decline of 1.2 deaths per 1000 live births.

The results for blacks are far more dramatic. A one standard deviation increase in the basic inputs causes a reduction of 3.3 neonatal deaths per 1000 live births. When this is added to the decline in neonatal deaths caused by changing the risk factors in the relevant direction, the explanatory variables account for 30 percent of the mean black neonatal mortality rate (15.6 deaths per 1000 live births). Of the inputs, teenage family planning, abortion and neonatal intensive care are the most significant whereas legitimacy status has more than double the impact of the other risk factors.

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[6] See footnote 4 in Chapter III for a detailed explanation of the direct and indirect effect of an input on mortality.

Some caution should be exercised in interpreting the results in Table 9 because an increase in abortion due, for example, to an increase in abortion availability is likely to cause organized family planning use to fall as well as cause a decline in the percentage of illegitimate births. Stated differently, these computations do not provide the reduced form effects that are required to evaluate the potential impacts of alternative policies to lower the neonatal mortality rate. Nevertheless, they do provide insights with regard to the benefits of expanding the use of one input or risk factor with all other inputs or risk factors held constant.

The results of including the race-specific percentage of births to women with at least a high school education in the birth outcome production functions are presented in Tables 10 and 11. For whites, this schooling measure is never significant except in the fully specified birth equation in which case it has the wrong sign. The insignificance and instability of this variable implies that at the aggregate level, education is most likely a proxy for socioeconomic status and is more accurately measured by the risk factors. The simple correlations between the education variable and the percentage of births to teenagers, the percentage of out-of-wedlock births and the percentage of fifth and higher order births are -.66, -.32, and -.45 respectively. Given the apparent multicolinearity and probable simultaneous equation bias, it is very difficult to draw any conclusions as to the efficiency versus the allocative effect of education in the production of white birth outcomes.

For blacks, the results are conceptually more consistent with the further distinction that the schooling measure has a negative and

Table 10

Linear Neonatal Mortality Rate Production Functions Including Education: Whites and Blacks[a]

Independent Variables	Whites 2SLS (10A)	Whites 2SLS (10B)	Blacks 2SLS (10C)	Blacks 2SLS (10D)
Teenage family planning*[b]	-.057	-.048	-.206	-.217
	(-2.84)	(-2.44)	(-3.60)	(-3.65)
Abortion rate[b]	-.043	-.016	-.132	-.090
	(-4.37)	(-1.20)	(-2.92)	(-1.62)
Prenatal care*[b]	-.057	-.021	.069	.116
	(-3.56)	(-1.04)	(1.31)	(1.82)
Neonatal intensive care*[b]	-.015	-.998	-1.758	-2.067
	(-.29)	(-1.64)	(-2.94)	(-3.16)
Low birth weight*[b]		.861		.924
		(2.73)		(1.40)
Education	-.005	.003	-.118	-.096
	(-.57)	(.31)	(-2.77)	(-2.05)
Ln population density	.268	.135	.899	.628
	(6.00)	(2.06)	(5.56)	(2.47)
Constant	13.437	5.544	23.357	8.848
	(13.50)	(1.82)	(7.43)	(.82)
F	14.44	13.98	9.01	7.57
Sample Size	677	677	357	357

[a]See footnote a to Table 5

[b]Endogenous

Table 11

Linear Low-Birth Weight Production Functions Including Education: Whites and Blacks[a]

Independent Variables	Whites 2SLS (11A)	Whites 2SLS (11B)	Blacks 2SLS (11C)	Blacks 2SLS (11D)
Teenage family planning*[b]	-.002	-.010	-.010	-.014
	(-.20)	(-1.34)	(-.64)	(-1.01)
Abortion rate[b]	-.021	-.006	-.028	-.008
	(-4.92)	(-1.30)	(-2.02)	(-.49)
Prenatal care*[b]	-.050	-.026	-.019	-.018
	(-6.96)	(-2.59)	(-1.43)	(-1.01)
Smoking[b]	.195	.016	.380	.305
	(3.80)	(.33)	(2.14)	(1.91)
Teenage births*[b]		.082		.035
		(4.43)		(1.17)
Illegitimate births*		.066		.044
		(6.00)		(5.15)
Parity*[b]		-.084		-.091
		(-3.13)		(-1.04)
Gestation*		.474		.273
		(12.55)		(7.40)
Education*	.002	.034	-.053	.0001
	(.51)	(8.69)	(-4.47)	(.01)
Ln population density	.135	.145	.331	.258
	(7.01)	(6.46)	(7.19)	(5.65)
Constant	7.957	-.266	13.020	3.442
	(13.44)	(-.20)	(7.89)	(1.24)
F	25.07	46.23	24.94	29.96
Sample Size	677	677	357	357

[a]See footnote a to Table 5

[b]Endogenous

significant effect on neonatal mortality holding birth weight constant (regression 10D). However, given that legitimacy status has a stable and siginificant effect on full-term light infants, and that over 52 percent of all black births in the sample are born out-of-wedlock, it was suspected that, as in the case of whites, education is actually a measure of social class; as such, it may be a less accurate proxy for poor socioeconomic status than legitimacy status. Yet, when the percentage of illegitimate births is included in the neonatal mortality production function, the effect of education falls, but it is just beyond conventional levels of significance ($p<.11$). The percentage of illegitimate births has no appreciable impact on mortality.

Thus, for blacks, it may be true that education enhances the marginal product of the health inputs. However, if this were the case, it is difficult to explain why schooling has no explanatory power in the birth weight equation. Education could be correlated with some other unobserveable variable that impacts on birth outcomes, but this input would have to have a direct effect on survival independent of birth weight. Such restrictions suggest the quality of perinatal or neonatal care as potential confounding variables, but such notions are purely speculative requiring further research.

The last issue to be addressed is whether the estimates from the linear model are robust with respect to the functional form. Tables C-3 through C-6 (in the Appendix) show the results from the logistic transformation and the Cobb-Douglas specification for both birth outcomes. The magnitude and significance of the coefficients are very similar to the linear estimates. In particular, the OLS coefficients for family planning use and neonatal intensive care underestimate their 2SLS counterparts considerably. Moreover, when Wu's T_2 statistic is applied to the logistic and Cobb-Douglas production functions including all risk factors, the same specifications that in the linear case could not reject the null hypothesis of zero correlation between the regressors and the residuals, also fail to reject the null hypothesis with these different functional forms. Thus, as suggested above, when testing for heterogeneity bias it is crucial that the equation be as well-specified as possible because omitted variable bias may be a confounding factor.

Conclusion

The theoretical and empirical framework in this study of infant health represents a substantial improvement over the more ad hoc approaches used in the past. First, holding to the notion of a health technology helps to clarify the distinction between a health input and the determinants of that input. Moreover, this distinction between production and demand serves to delineate the borders separating epidemiology and economics. Rosenzweig and Schultz have argued that attempts to estimate the production process by direct correlation, as is done by epidemiologists, generates biased coefficients regardless of how well-specified the equation is. However, results from this study suggest that such a claim is too strong. What Rosenzweig and Schultz have labeled "heterogeneity bias" cannot definitively be distinguished from omitted variable bias. Before the existence of the former can confidently be asserted, care has to be taken to more completely specify the health technology.

Despite the questionable presence of heterogeneity bias and the need for more completely specified equations, the findings from this study do not support the rather facile use of proxies to compensate for missing inputs. Education, for example, is often utilized as a "catch-all" substitute when data on smoking, alcohol, drugs and nutrition are lacking. In addition, human capital theory has argued for a more narrowly defined meaning of schooling. This dissertation has pointed out the difficulty of interpreting education as a measure of any of these factors at the aggregate level.

This study has also raised the issue of sample selection bias in the study of birth outcomes. This arises because the increased accessibility of contraception and abortion which permits women a choice regarding the decision to give birth. If one assumes that the genetic component, which in part determines the health of a newborn, is imbedded in the residuals of a health production function, then the further assumption that this error term is randomly distributed with zero mean is extremely problematic. The majority of women who choose to abort either have diagnosed medical risks or fall within high-risk categories due to age or marital status. Stated differently, women who abort have, on average, a genetic birthing component that is less favorable than women who do not abort. Moreover, regressors that appear in the structural birth outcome equation, such as age, would also appear in the equation predicting the probability of aborting, thereby insuring that the right-hand-side variables in the structural function are correlated with the disturbance term. Although this proposition could not be tested in an ecological study such as this, it points to a potentially fruitful area of future research.

From the perspective of a national health policy, the results from this study have a number of implications.[1] First, the effect of abortion and neonatal intensive care on neonatal mortality are dramatic for blacks. This is of particular interest since unlike WIC, maternal and infant care projects, and community health centers, neither of these inputs has been specifically targeted to improve the

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[1]The reader is reminded that although this study examined only twenty-two percent of the counties in the U.S., this represented approximately eighty percent of the white and black population in the country.

birth outcomes of the poor. However, the cutoff of federal funds for Medicaid financed abortions has inadvertantly become a public health issue since the evidence now accumulating points to the availability of abortion as a major factor in the accelerated rate of decline in neonatal mortality experienced in this country since the Supreme Court decision in 1973.[2] This takes on added significance if it is true, as McCormick (1985) suggests, that the use of neonatal technology may be reaching the point of diminishing returns. In other words, if the U.S. infant mortality rate is to continue declining, then gains will have to be made to reduce the incidence of prematurity--an outcome for which abortion has its greatest impact.

Another result germane to public health policy is the impact of family planning clinics on black birth outcomes, and to a lesser extent on white outcomes. As mentioned in Chapter IV, the mechanism by which this program operates on infant health remains unclear. However, if future research indicates that teenage family planning users are more effectively integrated into a network of medical care than non-users, then the expansion of this relatively inexpensive service could be an important tool for achieving a continued decline in infant mortality.

Finally, substantial gains in the reduction of infant mortality can be achieved by lowering the percentage of births to young and unmarried mothers. In the case of whites, births to teenagers have more of an impact on neonatal mortality than any other health input.

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[2] The effect of the Hyde Ammendment on abortions to poor women is unclear from evidence to date (Cates, 1981; Henshaw et al., 1984; Nestor and Gold, 1984)

More specifically, a one standard deviation decline in the percentage of births to adolescents (approximately 3 percentage points) would reduce neonatal mortality by a greater amount than a 5 percentage point increase in the percentage of women who initiate care in the first trimester. In the case of blacks, the proportion of births born out-of-wedlock has almost 6 times the impact on neonatal mortaltiy that marital status has on white birth outcomes. In sum, as important as the expanded availability and utilization of health resources is to the continued decline in neonatal mortality, many causes of poor birth outcomes are fundamentally social in origin, requiring solutions that reflect this broader perspective.

Appendix A

As noted in Table 1 a number of variables had missing values because (1), the state in which the county is located did not report the relevant information on birth certificates, or (2), the information was not collected at the time of birth. In the latter case, if less than 30 percent of the average number of births in a county between 1976 and 1978 recorded the relevant data, then a missing value was entered for that county. The table below presents the number of missing values that had to be estimated in the case of whites and blacks.

	Whites (N=677) number missing	Blacks (N=357) number missing
prenatal care	16	10
illegitimate births	192	113
parity	0	1
education	52	31
gestation	100	65

The following regression results are from the equations that generated the estimates for the missing values for each of the above inputs. The instrumental variables used were the race-specific percentage of women 15-44 with family income less than 200 percent of the poverty level in 1980 (POV*); the race-specific percentage of women 15-49 with at least a high school education in 1970 (HSP*); the race-specific percentage of women 15-49 employed in 1970; and the race-specific percentage of women who were married with spouse present in 1975 (MSP*).

Whites[1]

prenatal care = 81.703 + .182xHSP* − .541xPOV* − .006xEMP*
R^2=. (24.13) (4.58) (−16.69) (−.13)

illegitimate births = 20.418 − .056xHSP* + .091xEMP* − .212xMSP*
R^2=.24 (11.39) (−3.77) (4.44) (−10.09)

gestation = 9.102 − .053xHSP* + .022xEMP* + .027xPOV*
R^2=.29 (22.90) (−11.18) (3.61) (6.92)

education = −29.627 + .957xHSP* + .122xEMP* − .127xPOV*
R^2=.60 (−9.56) (25.74) (2.70) (−4.19)

Blacks

prenatal care = 74.276 + .064xHSP* − .358xPOV* + .055xEMP*
R^2=.13 (7.09) (.73) (−3.91) (.56)

illegitimate births = 82.370 − 1.259xMSP* + .290xEMP* + .113xHSP*
R^2=.49 (20.81) (−14.94) (4.29) (1.96)

gestation = 18.724 − .127xHSP* + .020xPOV* + .033xEMP*
R^2=.41 (10.76) (−8.75) (1.28) (2.01)

education = 6.349 + .524xHSP* − .093xPOV* + .088xEMP*
R^2=.62 (1.43) (13.88) (−2.40) (−2.13)

parity = 13.018 − .092xHSP* + .022xPOV* − .063xEMP*
R^2=.29 (7.19) (−5.98) (1.42) (−3.72)

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[1] T-ratios in parentheses

Appendix B

This appendix summarizes Wu's T_2 statistic following the lucid description by Nakamura and Nakamura (1981). As mentioned in the text, this is a test of whether the right-hand-side regressors are correlated with the residuals. This is more formally refered to as a test of the orthogonality assumption of ordinary least squares (OLS). The model is as follows:

$$M = HB + X_1C + u \qquad \text{(structural equation)} \qquad (1)$$

$$H = X_1P_1 + X_2P_2 + V = XP + V \qquad \text{(reduced form equation)} \qquad (2)$$

where M is an Nx1 vector of observations on the neonatal mortality rate; H is an NxG matrix of stochastic health inputs; X_1 is an NxK_1 matrix of exogenous health determinants; X_2 is an NxK_2 matrix of demand determinants; and u and V are an Nx1 and NxG matrices of disturbances; B, C, P_1, P_2 are Gx1, K_1x1, K_1xG, K_2xG matrices of unknown disturbances. The covariance matrix of (u,V) is

$$S = \begin{matrix} s & d' \\ d & S_{22} \end{matrix} \qquad \text{where } E(X_1,u) = E(X_2,u) = 0$$

Wu's T_2 statistic is a test of whether E[Hu] = d = 0. Hausman presents a more convenient test of whether d=0 which can be expressed as follows:

$$M = \hat{H}B_2 + X_1C + eB_3 + u \qquad (3)$$

where $\hat{H}$ + e has been substituted for H; $\hat{H} = X\hat{P}$ and $\hat{P}$ is the matrix of reduced form coefficients estimated from (2); $B_2 = B_3 = B$. Moreover,

$\hat{H}$ is asymptotically orthogonal to e under both the null (H_o: d=0) and the alternative (H_a:d≠0). Therefore, B_2 is consistent under either hypothesis. However, the plim of B_3 equals the plim of B_2 only if e is orthogonal to u, and herein lies the crux of the test. Following Hausman (1978), (3) can be rewritten by adding and subtracting HB_2 as follows:

$$M = HB_2 + X_1C + eD + u \qquad (4)$$

where $D = B_3 - B_2$. Hence, as Nakamura and Nakamura show, Wu's T_2 test, Hausman's I test, and Durbin's (1954) test can all be expressed as a test of whether D=0. This is a straight forward F-test on a set of linear restrictions. Consequently,

$$L = [(RRSS - URSS)/URSS] x [N-2g-K_1)/G]$$

is the test of whether the health inputs (H) are correlated with the structural residuals (u). RRSS is the sum of squared residuals from the OLS estimation of (1); URSS is the sum of squared residuals from (4).

Appendix C

Table C-1

Linear Low-Birth Weight Production Functions--Whites[a]

Independent Variables	2SLS (A-1A)	2SLS (A-1B)	2SLS (A-1C)	2SLS (A-1D)	2SLS (A-1E)
Teenage family planning*[b]	-.001	.011	.006	-.002	-.004
	(-.11)	(1.53)	(.78)	(-.27)	(-.60)
Abortion rate[b]	-.021	-.008	-.008	-.010	-.012
	(-4.93)	(-1.75)	(-1.61)	(-2.00)	(-2.83)
Prenatal care*[b]	-.047	-.011	.0005	-.021	-.018
	(-9.75)	(-1.30)	(.05)	(-1.85)	(-1.73)
Cigarettes[b]	.190	.116	.080	.027	.002
	(3.79)	(2.38)	(1.60)	(.52)	(.03)
Births to teenagers*[b]		.090	.101	.090	.033
		(5.00)	(5.56)	(4.86)	(1.88)
Illegitimate births*			.055	.057	.048
			(4.55)	(4.68)	(4.43)
Parity*[b]				-.092	-.103
				(-3.04)	(-3.82)
Gestation*					.337
					(9.80)
Ln population density	.134	.208	.193	.181	.112
	(7.04)	(9.00)	(8.26)	(7.66)	(5.03)
Constant	7.926	3.486	2.391	5.141	3.996
	(13.55)	(3.34)	(2.18)	(3.64)	(3.15)
F	30.43	33.39	31.39	28.80	42.76
Sample Size	677	677	677	677	677
Wu's T_2, F=	11.753				3.975

[a]See footnote a to Table 5

[b]Endogenous

Table C-2

Linear Low-Birth Weight Production Functions--Blacks[a]

Independent Variables	2SLS (A-1A)	2SLS (A-1B)	2SLS (A-1C)	2SLS (A-1D)	2SLS (A-1E)
Teenage family planning*[b]	-.002	.008	-.013	-.011	-.011
	(-.11)	(-.60)	(-.98)	(-.78)	(-.92)
Abortion rate[b]	-.052	-.016	-.026	-.038	-.011
	(-3.72)	(-1.19)	(-1.93)	(-2.30)	(-.71)
Prenatal care*[b]	-.060	-.028	-.026	-.046	-.023
	(-5.51)	(-2.51)	(-2.36)	(-2.57)	(-1.44)
Cigarettes[b]	.372	.339	.498	.506	.312
	(1.80)	(2.00)	(2.92)	(2.77)	(1.93)
Births to teenagers*[b]		.139	.060	.058	.029
		(5.00)	(1.95)	(1.75)	(1.00)
Illegitimate births*			.050	.053	.044
			(5.84)	(5.64)	(5.31)
Parity*[b]				-.135	-.110
				(-1.50)	(-1.39)
Gestation*					.271
					(7.35)
Ln population density	.316	.424	.257	.224	.247
	(5.96)	(8.73)	(4.59)	(3.50)	(4.42)
Constant	12.795	5.535	5.455	8.003	4.079
	(6.75)	(2.60)	(2.58)	(2.83)	(1.61)
F	19.36	28.17	29.39	22.64	32.41
Sample Size	357	357	357	357	357
Wu's T_2, F=	13.951				1.061

[a]See footnote a to Table 5

[b]Endogenous

Table C-3

Cobb-Douglas Neonatal Mortality Rate Production Functions: Whites and Blacks[a]

Independent Variables	Whites OLS (A-3A)	Whites 2SLS (A-3B)	Blacks OLS (A-3C)	Blacks 2SLS (A-3D)
Teenage family planning*[b]	-.003	-.006	-.002	-.007
	(-2.99)	(-3.12)	(-1.99)	(-2.94)
Abortion rate[b]	-.074	-.108	-.047	-.089
	(-4.01)	(-3.58)	(-1.50)	(-1.62)
Prenatal care*[b]	-.093	-.283	.052	.157
	(-1.66)	(-2.18)	(1.01)	(1.20)
Neonatal intensive care*[b]	-.050	-.039	-.084	-.100
	(-2.41)	(-.74)	(-3.18)	(-1.91)
Low birth weight*[b]	.548	.449	.811	1.036
	(9.64)	(2.38)	(7.49)	(3.25)
Ln population density	.010	.013	.004	.007
	(2.42)	(2.43)	(.60)	(.66)
Constant	1.788	4.838	.712	-.046
	(5.78)	(12.04)	(1.89)	(-.03)
F	27.72	17.03	13.17	5.43
R^2	.192		.170	
Sample Size	677	677	357	357
Wu's T_2, F=		2.232[c]		2.123[d]

[a]See footnote a to Table 5

[b]Endogenous

[c]Significant at (p.<.05)

[d]Not significant (p>.05)

Table C-4

Cobb-Douglas Low-Birth Weight Production Functions: Whites and Blacks[a]

Independent Variables	Whites OLS (A-4A)	Whites 2SLS (A-4B)	Blacks OLS (A-4C)	Blacks 2SLS (A-4D)
Teenage family planning*[b]	-.001	.0001	.0002	-.001
	(-1.05)	(.15)	(.58)	(-1.35)
Abortion rate[b]	-.024	-.042	.024	.010
	(-2.11)	(-2.17)	(1.66)	(.44)
Prenatal care*[b]	-.120	-.239	.034	-.015
	(-3.06)	(-2.24)	(1.60)	(-.28)
Smoking[b]	-.136	.016	.173	.195
	(-2.39)	(.30)	(1.78)	(2.12)
Teenage births*[b]	.027	.059	.152	.077
	(1.62)	(1.56)	(4.28)	(1.48)
Illegitimate births*	.078	.061	.056	.140
	(5.03)	(4.10)	(1.64)	(5.18)
Parity*[b]	-.090	-.100	-.001	-.006
	(-7.79)	(-4.06)	(-.07)	(-.18)
Gestation*	.541	.460	.336	.339
	(12.30)	(10.35)	(8.53)	(7.96)
Ln population density	.010	.015	.025	.018
	(3.50)	(4.03)	(7.35)	(4.86)
Constant	1.407	1.767	.164	.372
	(5.55)	(2.89)	(.60)	(.90)
F	52.80	46.63	33.52	29.66
R^2	.410		.451	
Sample Size	677	677	357	357
Wu's T_2,F=	3.265		.899[c]	

[a]See footnote a to Table 5

[b]Endogenous

[c]Not significant (p>.05)

Table C-5

Logistic Neonatal Mortality Rate Production Functions: Whites and Blacks[a]

Independent Variables	Whites OLS (A-5A)	Whites 2SLS (A-5B)	Blacks OLS (A-5C)	Blacks 2SLS (A-5D)
Teenage family planning*[b]	-.003	-.006	-.002	-.009
	(-3.40)	(-2.29)	(-1.89)	(-3.17)
Abortion rate[b]	-.004	-.004	-.002	-.005
	(-5.07)	(-2.81)	(-1.66)	(-1.67)
Prenatal care*[b]	-.002	-.003	.001	.004
	(-2.10)	(-1.17)	(.72)	(1.42)
Neonatal intensive care*[b]	-.021	-.090	-.029	-.100
	(-1.26)	(-1.22)	(-2.76)	(-2.67)
Low birth weight*[b]	.081	.089	.067	.086
	(8.81)	(2.48)	(7.49)	(2.72)
Ln population density	.012	.010	.001	.008
	(2.79)	(1.46)	(.12)	(.68)
Constant	-4.997	-4.880	-4.848	-5.024
	(-11.41)	(-13.32)	(-34.21)	(-9.42)
F	26.65	17.17	12.50	5.36
R^2	.185		.162	
Sample Size	677	677	357	357
Wu's T_2, F=		3.860		3.153

[a]See footnote a to Table 5

[b]Endogenous

Table C-6

Logistic Low-Birth Weight Production Functions: Whites and Blacks[a]

Independent Variables	Whites OLS (A-4A)	Whites 2SLS (A-4B)	Blacks OLS (A-4C)	Blacks 2SLS (A-4D)
Teenage family planning*[b]	-.001	.0002	.0001	-.001
	(-1.39)	(.16)	(.40)	(-.80)
Abortion rate[b]	-.002	-.002	.001	-.0003
	(-3.07)	(-2.50)	(.93)	(-.25)
Prenatal care*[b]	-.002	-.003	.001	-.002
	(-2.49)	(-1.93)	(1.62)	(-1.22)
Smoking[b]	-.031	.003	.026	.032
	(-3.34)	(.39)	(1.69)	(3.32)
Teenage births*[b]	.003	.005	.006	.002
	(2.35)	(1.68)	(3.63)	(1.01)
Illegitimate births*	.009	.009	.002	.004
	(4.73)	(4.45)	(2.04)	(5.22)
Parity*[b]	-.022	-.020	-.001	-.006
	(-8.75)	(-3.73)	(-.26)	(-1.10)
Gestation*	.078	.065	.024	.024
	(12.76)	(10.46)	(8.33)	(7.52)
Ln population density	.012	.016	.029	.017
	(3.82)	(3.96)	(6.71)	(3.46)
Constant	-3.044	-3.112	-3.002	-2.788
	(-28.13)	(-14.01)	(-19.86)	(-13.44)
F	57.28	47.44	32.74	28.55
R^2	.430		.445	
Sample Size	677	677	357	357
Wu's T_2,F=	3.860		1.090[c]	

[a]See footnote a to Table 5

[b]Endogenous

[c]Not significant (p>.05)

Appendix D

The full conceptual model used is this study can be presented as follows:

$$d = d(a,c,p,n,b,u) \quad (1)$$
$$b = b(a,c,p,s,g,u) \quad (2)$$
$$g = g(a,c,p,i,t,f,u) \quad (3)$$
$$i = i(a,c,t,u) \quad (4)$$
$$t = t(a,c,i,u) \quad (5)$$
$$f = f(a,c,u) \quad (6)$$

$$a = a(y,z,e,u) \quad (7)$$
$$p = p(y,z,e,u) \quad (8)$$
$$c = c(y,z,e,u) \quad (9)$$
$$n = n(y,z,e,u) \quad (10)$$
$$s = S(y,z,e,u) \quad (11)$$

where d is neonatal mortality, b is birth weight, g is gestational age, i is legitimacy status, t is births to teenagers, f is parity, a is abortion, c family planning services, p is prenatal care, n is neonatal intensive care, s is smoking, u is the health endowment, y is income, z is price and availability measures, and e is reproductive efficiency and parental tastes. Equations (1)-(6) are the structural equations because they show the relationships among the endogenous variables. Equations (7)-(11) are the reduced form input demand functions. It is straight forward to show that only equations (1), (2), and (6) satisfy the rank condition for identification.

Substitution of equation (3) into equation (2) results in

$$b = b(a,c,p,s,i,t,p,u) \quad (2a)$$

which in the text is termed a quasi-structural equation. The advantage of of this specification is that it permits one to examine the impact of the endogenous risk factors (i,t,f) on birth weight. If gestation (g) is also added to to (2a), then one can compare the direct effect of the endogenous risk factors on birth weight with their total effect. The difference between the total and direct effect indicates the extent to which the risk factors impact on birth weight through gestation.

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