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Demographic analysis of the determinants of postneonatal mortality in the U.S.: Inferences from risk specific infant mortality

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DEMOGRAPHIC ANALYSIS OF THE DETERMINANTS OF POSTNEONATAL MORTALITY IN THE U.S.: INFERENCES FROM RISK-SPECIFIC INFANT MORTALITY

by

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

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Abstract

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by

Jae-Il Myoung

Advisor: Professor Michael Grossman

This paper presents the first postneonatal mortality production functions and their reduced form equations on the basis of the economics of the family and household production. Its aim is to contribute to an understanding of the determinants of the variation in the race- and birthweight-specific postneonatal mortality rates among states in the U.S. in 1980. The results of a reduced-form model underscore such factors as neonatal intensive care availability, pediatrician availability, MIC project availability, poverty, the female unemployment rate, and the female labor force participation rate in explaining the variation in the postneonatal mortality rates regardless of race. Abortion availability which is an important determinant of neonatal mortality does not have any explanatory power, however. Likewise, this study fails to support the findings of earlier studies regarding the effects of schooling and the public programs such as family planning clinics, Medicaid, and WIC program on the infant mortality rate. The estimation of the production function corroborates the importance of prenatal care use especially for the normal-birthweight infants regardless of race. The use of pediatrician services also exerts a negative impact. Of the public program inputs considered, the use measure of AFDC support for teenagers turns out to affect the survival prospects greatly in an adverse manner.

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Finally, I wish to thank my parents and wife for their support, understanding and patience. My two kids also deserve special thanks for tolerating their father who could not play with them. After all, their life here in the States was not chosen by their will, and I am very sorry about that.

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CHAPTER I

Introduction

1.1 Background

The infant mortality rate of the United States has been a cause of long-standing policy concern for its notoriously high level compared to those of other developed countries such as the countries of Scandinavia. Also, both of its substantial time trends and cross-sectional variations have induced considerable research effort of the academics of various fields including economists. Especially, the time-series behavior of the infant mortality rate over the past several decades has attracted extensive attention.

From 1955 through 1964, there had been no significant decrease in the infant mortality rate which fell by only 0.6 percent per annum. The slowdown in the decline in the infant mortality rate could not be expected at the time in light of the sharp decline during the first half of this century. In spite of the extensive studies in this area, the factors which contributed to this event are still unidentified, however.

After the period of relative stability, the long downward trend in infant mortality resumed again and continued to decline with the rate of 4.0 percent per annum for two decades between 1964 and 1984. This impressive progress in reducing infant mortality rate also has stimulated researchers to direct considerable attention towards exploring the determinants of infant mortality. The focus of attention has been on the role of prenatal care, legal abortion, technological advances in medical treatments for infants with health problems in the neonatal period, and a variety of public programs aimed at the poor. As a result, we do know much about the factors which has contributed to the sharp decline in the infant mortality rate during this period.

Nevertheless, our knowledge about the determinants of infant mortality is no doubt far from being satisfactory in view of the conflicting empirical results reported in the received literature. To be sure, there are myriad factors that affect the infant's survival prospect in one way or another, and some influential factors that need to be taken into account when the determinants of infant mortality are to be investigated should be separated from those innumerable factors. However, any attempt to assess the relative importance of plausible factors is bound to be impeded by numerous obstacles from measurement problems to illegitimate statistical techniques. In light of these empirical problems, the ambiguities and controversies in the literature and in policy formation are not surprising.

What is worse, the recent behavior of infant mortality has added to troubles of policy-makers and researchers due to its failure to sustain rapidly declining trend enjoyed for the past two decades. Indeed, if the remarkable achievement in reducing the infant mortality rate in the U.S. will continue is seriously doubted recently, although the evidences are not clear-cut yet. But, it is enough, at least, to draw attention and, to be sure, the situation cannot be gone unnoticed.

The signs of slowdown in the decline in the infant mortality rate, which reminds us of the static picture of the rate of the late 1950s and early 1960s, began to appear in the early 1980s. Between 1981 and 1984, infant mortality in the U.S. fell by 3.4 percent per annum and the postneonatal mortality rate among blacks which has been tending downward at a faster pace than the white actually rose between 1982 and 1983. Moreover, the black infant mortality rate declined by only 1.1 percent per annum between 1984 and 1986, whereas the counterpart for the white was 2.8 percent. Thus, although both whites and blacks suffer from the deceleration, the situation is worse for blacks.

The problems are not confined to racial differences in the rate of decline. For a long time, policy-makers and researchers have worried over the seemingly everlasting differential in the absolute level of infant mortality between whites and blacks. It is a widely recognized fact that the infant mortality rate of blacks has been twice as large as that of whites since early 1960s. The reasons for this surprising regularity, however, have not been articulated yet. Thus, it remains an open question why the survival probability differential of black and white infant has not narrowed despite the dramatic decline in mortality for the last two decades. Moreover, there is a problem of large differential in the infant mortality rate across geographical regions at various levels and socioeconomic groups. Therefore, despite the growing number of studies of infant mortality in the United States, the lacunae in our knowledge remain substantial, while the future prospect for further decline is not apparent.

1.2 Objectives and Outline of the Study

In demographic study of infant mortality, it is almost customary to focus on neonatal mortality (deaths of infants within the first 27 days of life) and in consequence less attention has been paid to postneonatal mortality (deaths within the first year of life but beyond the first 27 days). There are some good reasons for that. Above all, the lack of research on postneonatal mortality can be attributed to the fact that it is not of importance relatively compared to neonatal mortality since two-thirds of infant deaths are concentrated on the neonatal period. Therefore, it is enough only to consider neonatal mortality to understand the behavior of infant mortality. Another reason is that the causes of two infant deaths are different. That is, neonatal deaths are usually caused by congenital anomalies, prematurity, and complications of delivery, while most postneonatal deaths are caused by sudden infant death syndrome (SIDS), infectious diseases, accidents, and congenital anomalies, implying that postneonatal mortality is much more sensitive to the standard of living. Thus, as income rises secularly, the importance of postneonatal mortality simply diminishes as it did in the past.

However, given that postneonatal mortality is more responsive to the behavior of income, recent socio-demographic phenomena that are closely associated with poverty may allow to attach more weight on postneonatal mortality than ever before. For example, the increase in the out of wedlock birth rate among teenagers may affect the survival probability of children adversely through the lack of appropriate support given their weak economic condition and unfavorable environment for infant health. In addition, the upward trend in female family headship and resultant changes in living arrangements can also exert a detrimental effect on development of vulnerable children due to the lack of appropriate personal attention, to the extent that the mother, possibly the sole wage-earner, is likely to fall below a poverty line. Perhaps more important, both the relative and absolute increase in people below the poverty line during the early 1980s partly due to the recession of the time might have played some role in the recent slowdown of the infant mortality rate.

Furthermore, the very fact that the further possibility to reduce the infant mortality rate diminishes as it approaches the lowest level attainable implies that it is now very costly to reduce the neonatal mortality rate marginally. Thus, it may be desirable to leave no stone unturned especially for the policy design, and postneonatal mortality still remains an unturned stone, particularly in terms of a multivariate analysis undergirded by an appropriate conceptual model.

Therefore, the major concern of this study is to investigate the proximate determinants of postneonatal mortality and the sources of differences in this rate between the races as an attempt to get an overall picture of infant mortality in the U. S. Specifically, this research is concerned with analyzing the effect of various public programs, prenatal and postnatal medical care availability and use, and socioeconomic factors on the birthweight-specific postneonatal mortality rates taken from the 1980 National Infant Mortality Surveillance.

Some specific questions that will be addressed in this study include: What are the effects of such public programs as Medicaid, Federally subsidized organized family planning services, and the WIC program on the race- and birthweight-specific postneonatal mortality rates? Are there any significant effects of welfare on postneonatal mortality through the moral hazard effect? What are the impacts of prenatal care on birthweight-specific mortality, and how do they vary by race and state cross-sections? What are the impacts of neonatal intensive care availability and use on birthweight-specific postneonatal mortality? How sensitive is postneonatal mortality to pediatrician availability? Do the socioeconomic variables such as the level of education of the mother, the female poverty and income levels, and the female unemployment rate play important roles? Does the female labor force participation affect survival prospects of infant favorably through the contribution to family earnings or negatively through the reduction of nonmarket time spent in child care?

The remainder of this thesis is divided into four chapters. Chapter II provides a review of the literature and specification of the empirical model to be estimated. In reviewing the literature, the attention will be confined to the studies in which the economics of the family and household production is employed as a theoretical framework. This study also derives the equations to be estimated from the same theoretical framework. Chapter III presents the estimation results of the reduced form equations and the postneonatal mortality rate production functions. They are reported separately by birth weight category and race. This is followed by a concluding chapter in which findings are summarized, and policy implications and some suggestions for further study are discussed.

CHAPTER II

SPECIFICATION OF A MODEL

2.1 Review of Literature

Infant mortality of the U. S. has attracted considerable attention from a wide range of disciplines besides demography. It will not be attempted to review all the voluminous literature on the subject in this section, however. Instead, in reviewing the literature, the focus of attention will be confined mainly to the literature whose theoretical underpinnings are the economic models of the family and household production, which have been proved to be fruitful in the analysis of birth outcomes.

Economic models of the family developed by Becker and Lewis (1973) and Willis (1973) has been used as a major framework to undergird the empirical research on birth outcomes such as birth weight and infant death. In the context of the infant survival, it is assumed that, at the household level, the survival probability of each birth is a choice variable along with the number of births and consumption, over which a joint utility function is defined.

The utility function is maximized subject to a household production function along with the resource constraint. The households produce the survival probability by combining the endogenous inputs such as the quantity and quality of medical care, nutrition, and the own time of the mother, according to household production functions. These considerations are not the only ones that play a role in the production process. The biological endowment of fetus which is not observed affects the survival probability production function as well.

The demand function for survival probability yielded by the usual constrained maximization exercises depends on input prices whose direct and indirect costs are negatively related to input availability, efficiency, income, and tastes. The interaction between the survival demand and production functions determines demand functions for medical care and other endogenous inputs, which depend on the same set of variables as the demand function for survival.

Therefore, on the basis of this framework, at least three types of estimable relationships can be formulated: structural production functions, input demand functions, and reduced-form outcome equations. The structural production function relates the survival probability to endogenous risk factors such as low birth weight and to basic health inputs including medical care services. Thus, the coefficients of the structural production function measure the direct effects of the health inputs on mortality with low birthweight held constant.

The set of input demand functions relates the use of certain input to exogenous variables including input price and availability measures, socioeconomic characteristics that reflect command over real resources and tastes, and the biological endowment. The reduced form equation is obtained by replacing input use levels in the structural production functions with the exogenous determinants of these levels.

The empirical literature on birth outcome has centered on the estimation of a part or all of these relationships. For example, based on this analytical framework, Grossman and Jacobowitz (1981) attempt to identify the causes of the sharp decline in the neonatal mortality rate in the United States in the period after 1963, examining the roles of public programs and policies. Using county as a unit of observation, they analyze the cross-section data for 1971 and find that of all the programs and socioeconomic variables considered, the increase in the legal abortion rate is by far the most important factor in reducing neonatal mortality rate regardless of race. They also find a strong negative relation between neonatal mortality and subsidized family planning services for low-income women. The impacts of the public programs such as Medicaid and maternal and infant care projects, however, are estimated to be quite small.

Although Grossman and Jacobowitz take the above-mentioned framework as a starting point of their analysis, the empirical implications of the theoretical model are not fully exploited in their specification of the model in that the equation estimated is neither the production function nor the reduced form equation because endogenous inputs are not distinguished from exogenous variables. As Joyce (1987) indicates, abortion rate should be thought of as an endogenous input in the production process of survival probability since the decision to abort depends on the perception of pregnant woman on the birth outcome prospects, economic resource, and cost and availability of abortion services. Then, the production function, in part, depends upon the biologically endowed probability to survive which the mother and possibly her physician may perceive but the researcher cannot observe. Since the mother and her physician may decide to remove the physical problem by aborting pregnancy, or to try to improve the birth outcomes by consuming more medical care services with better quality, the disturbance term in the survival probability production function is transmitted to the demand for abortion services or other medical care inputs. The estimation without the recognition of the correlation between the disturbance term and the arguments of the production function yields, in general, the biased and inconsistent estimates of the parameters in question. To remove the bias and inconsistency caused by the failure to take account of causality running two opposite directions, the empirical model should be specified in the simultaneous equations context. In other words, the input demand function and production function should be estimated simultaneously (Rosenzweig and Schultz 1982, 1983a, 1983b).

To extend and update the work of Grossman and Jacobowitz in this context, Corman and Grossman (1985) estimate a reduced-form model which can be obtained by inserting the arguments of input demand functions into the structural production function. The arguments of input demand functions include input price and availability measure, socioeconomic characteristics that reflect command over real resources and tastes, and the biological endowment. Based on county data of the United States centered on 1977, they find that, for whites, female's schooling levels, neonatal intensive care availability, poverty, Medicaid, maternal nutrition programs, abortion availability, and organized family planning clinics are important determinants of neonatal infant mortality. For blacks, it turns out that abortion, neonatal intensive care, schooling levels, and Medicaid are of importance.

Using county as a unit of observation again, Corman et al. (1985) estimate the neonatal mortality rate production functions, and get the similar results: abortion, prenatal care, neonatal intensive care, and the WIC program are underscored in explaining the variation in neonatal mortality at the county level for 1977.

Joyce (1987a) explores mechanisms by which the health inputs, especially prenatal care and abortion, affect neonatal mortality other than low birthweight. In particular, he considers the percentage of births in which gestational age was 36 weeks or less (prematurity) and the percentage of births to women in high-risk categories (births to teenagers or to women in their forties). For blacks, an increase in prenatal care lowers the percentage of light births solely by reducing the incidence of prematurity. This is consistent with Harris's (1982) results based on a sample of black births in Massachusetts between 1975 and 1976 and with medical evidence summarized by Harris. For both races, the coefficient of abortion in the low-birthweight production function falls substantially when prematurity is held constant. For whites, abortion prolongs gestational age by lowering the percentage of births to women in high-risk age categories.

Joyce (1987b) also explores mechanisms that can account for the input availability effects in Corman and Grossman (1985) reduced form neonatal mortality equations. His estimates of input demand functions for abortion, neonatal intensive care, and organized family planning reveal that in each case own availability is a powerful predictor of own use. Moreover, they suggest that the negative relationship between the availability of a certain input and mortality reflects causality from an increase in availability to an increase in use and hence to a better birth outcome.

Joyce, Corman, and Grossman (1987) combine the input use effects that emerge from the estimated infant health production functions with data on the costs of expanding the use of these inputs to compare the cost-effectiveness of strategies to reduce infant mortality. Based on the impact of prenatal care on neonatal mortality in a cross-section of counties for 1977, they find the early initiation of prenatal care to be the most cost-effective means of reducing neonatal mortality for blacks and whites. Moreover, blacks benefit more per dollar of input use than whites.

In the attempts to evaluate the influence of various medical care and public programs on neonatal mortality, one problem with all the above studies is the interaction effect between some determinants and low birthweight or weak economic condition of parents. The effect of neonatal intensive care units, for instance, should be much larger on mortality of low-birthweight infants. Likewise, the public programs of which target is the population with low income should have their main effects on the death rates of infants born to poor women. They account for these effects by adding interaction terms to the equation to be estimated. But it involves a large set of regressors and thus cause severe collinearity problems. What is worse, the estimation of the reduced-form model becomes a formidable task. Thus, they are forced to estimate a truncated version of the full model. The most direct way to overcome the problem, however, is to employ the infant mortality rates classified by birthweight and income level of parents.

Grossman et al. (1988) and Corman et al. (1988) can directly control for the interaction effect through the use of the 1980 National Infant Mortality Surveillance (NIMS) data that are cross-classified by race, period of death, and birthweight. Using the same framework of household production functions, they investigate the determinants of variations among states of the United States in white and black neonatal mortality rates in 1980 classified by birthweight, and birthweight and mother's age as a proxy for income. Corman et al. (1988) which is a shortened version of Grossman et al. (1988) find abortion and neonatal intensive care availability to be the most important determinants of neonatal mortality. For whites, the two factors are of approximately equal importance in determining neonatal mortality. For blacks, abortion availability has twice the impact of neonatal intensive care. With respect to the effect of the input use variables, the estimation of the birthweight-specific neonatal mortality production functions reveals that the use of neonatal intensive care services is the key input both for whites and blacks (Grossman et al. 1988). The use of prenatal care services also turns out to be an important factor, particularly for the normal-birthweight infants.

2.2 Specification of the Model

In this study, we also employ the economics of the family and household production as a theoretical framework to investigate the determinants of postneonatal mortality, and are concerned with the estimation of the structural and reduced form postneonatal mortality equations. Although we considered these equations on the basis of the micro production function and demand relationships in the above section, our estimation of the model will be based on the aggregated data at the state level by appealing to micro relationships as in most studies. Following the literature reviewed in the preceding section, we first specify the postneonatal mortality rate production functions.

Suppose that the race and state-specific postneonatal mortality rates are classified by two birthweight categories. The birthweight categories are light (less than 2,500 grams) and normal (2,500 grams and over). The following notation is adopted. The subscript i (i = 1,2) denotes a birthweight cell; and the subscript j pertains to the j^{th} state. Let p_{ij} be the postneonatal mortality in birthweight cell i in state j and let k_j be the fraction of infants who survive the neonatal period in low-birthweight cell in state j.

As an identity, the observed postneonatal mortality rate in the j^{th} state (not birthweight and age-specific) is

$$p_j = k_j p_{1j} + (1-k_j) p_{2j} \qquad (1)$$

where p_{1j} is the postneonatal mortality rate of low-birthweight infants and p_{2j} is the postneonatal mortality rate of normal-birthweight infants in the j^{th} state.

Specify structural production functions for the two postneonatal mortality rates as follows:

$$p_{ij} = \alpha_{0i} + \alpha_{1i} m_{ij} + \alpha_{2i} a_{ij} + \alpha_{3i} mn_{ij} + \alpha_{4i} mp_j + \alpha_{5i} f_j + u_{ij} \quad (2)$$

where m_{ij} denotes prenatal medical care use, a_{ij} denotes the use of abortion services, mn_{ij} denotes neonatal intensive care use, mp_j denotes postneonatal medical care use which is not birthweight-specific due to data limitations, f_j denotes the use of a public program aimed at the poor, and u_{ij} denotes the disturbance term.

Prenatal medical care use is a comprehensive indicator of medical care received in the prenatal period. Neonatal intensive care is less comprehensive because it pertains only to intensive care, but it has the advantage of being specific to the neonatal period. On the other hand, the postneonatal medical care variable such as the use of pediatrician services is neither comprehensive nor specific to the postneonatal period. Consequently, all three medical care use variables are included in the postneonatal mortality structural production functions. One justification for including prenatal medical care use and neonatal intensive care use in the postneonatal mortality production function is that the prompt initiation of prenatal care may raise the quantity and quality of medical care received in the neonatal and postneonatal periods. The measures of these prenatal and postnatal medical care services are expected to have negative relationships with the birthweight-specific postneonatal mortality rates.

The use of abortion services is also an endogenous variable determined by such exogenous variables as the economic resources, price and availability measure of abortion services along with perception of pregnant women on the birth outcome. To the extent that a woman with undesirable prospect on the birth outcome tend to seek abortion, the fetal selection would ultimately translate into the reduction in infant mortality. Thus, we expect that it has a negative association with the postneonatal mortality rates.

The public program inputs pertain to the nutrition input and the use of contraceptive services, which are expected to have negative effects on mortality. The use of contraceptive services is a relevant determinant of birth outcomes because it reflects the wantedness of a pregnancy or birth, which affects the health endowment of fetus or infant favorably through controlling such risk factors as parity, child spacing, legitimacy status, and mother's age at birth.

The inputs in equation (2) depend on a vector of the input price and availability (w_j) in the j^{th} state, public program availability (p_j), and socioeconomic characteristics that reflect command over resources and tastes (y_j). Substitution of input demand functions into the production function generates reduced form postneonatal mortality equation:

$$p_{ij} = f(w_j, p_j, y_j) \qquad (3)$$

These equations constitute the reduced form of the model, together with the input demand functions.

The input price and availability pertain to neonatal intensive care, pediatricians, and abortion. The public program availability involves organized family planning, Medicaid, WIC, and the maternal and child health program. The cost and availability of family planning services and abortion services are relevant reduced form regressors even if the use of these services has no impact on birthweight-specific mortality. The reason is that a reduction in the direct or indirect price of these services will increase the quantity and quality of medical care that parents allocate to births and to their infants.

Socioeconomic determinants include schooling, poverty, the female unemployment rate, and the female labor force participation rate. We expect the schooling variable to have a negative sign, although a positive sign cannot be ruled out in the context of household production approach. The female unemployment rate should be positively related to postneonatal mortality because both the stresses associated with the unemployed status and reductions in income can have unfavorable consequences on the health of infants. However, in the case of the female labor force participation, the direction of the impact on the infant's survival probability is ambiguous because the substitution and income effects have the opposite implications on the health of children.

Reduced form postneonatal mortality rate equations (3) can be obtained by ordinary least squares since the regressors in these equations are limited to exogenous variables. On the other hand, all inputs in the production functions (2) are endogenous variables, but ordinary least squares still may be appropriate because the mortality rate is birthweight-specific. Corman, Joyce and Grossman (1987) indicates that production function regressors are independent of the disturbance term when birthweight is held constant. Nevertheless, we will investigate the endogeneity of production function regressors in this research through the specification error tests.

CHAPTER III

ESTIMATION AND EMPIRICAL RESULTS

3.1 Data and Measurement of Variables

The data required to estimate the reduced form and structural models discussed in Chapter II are postneonatal mortality rates; medical care input availability and use; public program input availability and use; and socioeconomic variables related to the infant's survival probability. These data are taken from various sources. The principal data set employed in this study is the 1980 National Infant Mortality Surveillance (NIMS). This data base has been augmented with information on births by a variety of characteristics from the 1980 National Center for Health Statistics (NCHS) Natality Tape, socioeconomic characteristics from the 1980 Census of Population, and program use and availability measure from other sources.

First, the data on dependent variables, postneonatal mortality rates of the U.S. in 1980, come from NIMS. NIMS was prepared by the Centers for Disease Control (CDC) under an interagency agreement between CDC and the National Institute of Child Health and Human Development.[1] Under the NIMS project each of the fifty states of the U.S. and the District of Columbia linked birth and death certificates for the 1980 birth cohort. These linkages pertain to births by mother's state of residence in 1980 regardless of the state in which the subsequent infant death occurred or the infant's state of residence at death. CDC then obtained data on the

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[1]For more detailed descriptions about the NIMS project, see Grossman, Corman, and Joyce (1988) and the literature cited therein.

number of infant deaths in tabular form from each state. The end result of the project is a state data base with race-specific neonatal, postneonatal, and total infant deaths and death rates by birthweight, by birthweight and mother's education, by birthweight and mother's age, by birthweight and cause of death, and by birthweight and other characteristics such as type of delivery, sex of infant, and so forth.

Among these various classifications just mentioned, Grossman, Corman, and Joyce (1988) analyze the neonatal mortality rates by race (white and black), birthweight (less than 2,500 grams and 2,500 grams or more) and state for the year 1980. As a follow-up study, this research will employ the postneonatal mortality rates classified by the same characteristics as the principal object of analysis.

Table 1 contains definitions of these dependent variables. The NIMS postneonatal data pertain to deaths of infant born in 1980 regardless of whether the death occurred in 1980 or 1981. Therefore, it is natural to define the postneonatal mortality rate as postneonatal deaths per thousand neonatal survivors. If the postneonatal mortality rate is given by deaths per thousand live births, it rises as the neonatal mortality rate falls with all other factors held constant.[2] The postneonatal mortality rates defined in Table 1 are not subject to this problem, which is important in assessing the impact of neonatal intensive care on postneonatal mortality.

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[2]Let B_{ij} be the number of live births in the i^{th} risk category in the j^{th} state, N_{ij} be the number of neonatal deaths, and P_{ij} be the number of postneonatal deaths. The two postneonatal death rates defined in the text are related as follows:

$$p_{ij} = p_{ij}(1-n_{ij}),$$

where $p_{ij} = P_{ij}/B_{ij}$, $p_{ij} = P_{ij}/(B_{ij}-N_{ij})$, and $n_{ij} = N_{ij}/B_{ij}$. With p_{ij} held fixed, p_{ij} rises as the neonatal mortality rate (n_{ij}) falls.

Table 1

Definition of Variables

Variable	Definition
A. Dependent Variables	
Postneonatal mortality rate of low-birthweight neonatal survivors*	Deaths in the second through twelfth months of life of infants born alive in 1980 weighing less than 2,500 grams at birth per thousand neonatal survivors weighing less than 2,500 grams at birth
Postneonatal mortality rate of normal-birthweight neonatal survivors*	Deaths in the second through twelfth months of life of infants born alive in 1980 weighing 2,500 grams or more at birth per thousand neonatal survivors weighing 2,500 grams or more at birth
B. Reduced Form Regressors	
Neonatal intensive care hospitals	Sum of number of hospitals with Level II, Level III, or Level II and III neonatal intensive care units in 1979 per thousand women aged 15-44 in 1980
Pediatricians	Number of pediatricians per thousand population aged 14 or less in 1980
Abortion providers	Number of abortion providers in 1980 per thousand women aged 15-44 in 1980
Family planning clinics	Number of organized family planning clinics in 1980 per thousand women aged 15-44 with family income less than 200 percent of the poverty level in 1980
Medicaid	Medicaid coverage of prenatal care for first-time pregnancies in 1980; dichotomous variable that equals one if state covered at least some first-time pregnancies of financially eligible women in that year
WIC program	Number of projects funded under the Special Supplemental Food Program for Women, Infants, and Children in 1980 per thousand women aged 15-44 with family income less than 200 percent of the poverty level in 1980

Table 1, Continued

Variable	Definition
MIC projects	Fraction of counties in a state served by maternal and infant care projects in 1980
Fraction high school educated*	Fraction of women aged 15-44 who had at least a high school education in 1980
Female poverty*	Fraction of women aged 15-44 with income below 200 percent of the poverty level in 1980
Female unemployment rate*	Percentage of women aged 16-44 in the labor force who are unemployed in 1980
Female labor force participation rate*	Percent of civilian noninstitutionalized women aged 16-44 who are in the labor force in 1980
C. Production Function Regressors	
Prenatal care use, low-birthweight births*	Fraction of low-birthweight (less than 2,500 grams) live births for which prenatal care began in the first trimester (first three months) of pregnancy in 1980
Prenatal care use, normal-birthweight births*	Same as preceding variable for normal-birthweight (2,500 grams or more) live births in 1980
Abortion rate*	Abortions performed on state residents in 1980 per thousand women aged 15-44 in 1980
Neonatal intensive care use, low-birthweight births	Sum of patient days in Level II, Level III, or Level II and III neonatal intensive care units spent by low-birthweight infants in 1979 divided by low-birthweight live births in 1980
Neonatal intensive care use, normal-birthweight births	Sum of patient days in Level II, Level III, or Level II and III neonatal intensive care units spent by normal-birthweight infants in 1979 divided by normal-birthweight live births in 1980

Table 1, Concluded

Variable	Definition
Pediatricians	Same as reduced form regressor
Family planning use	Number of users of organized family planning clinics with family income less than 200 percent of the poverty level in 1980 per thousand women aged 15-44 with family income less than 200 percent of the poverty level in 1980
WIC use	Number of eligible pregnant women served by WIC in 1980 per thousand eligible pregnant women in 1980
AFDC for teen-agers*	Fraction of women aged 15-19 receiving public assistance in 1980
AFDC for adults*	Fraction of women aged 20-44 receiving public assistance in 1980

Note: An asterisk (*) next to a variable means that it is race-specific.

Table 2

Means and Standard Deviations of Variables

Variable	Whites		Blacks	
A. Dependent Variables	Mean	Std. Dev.	Mean	Std. Dev.
Postneonatal mortality rate of low-birthweight infants*a	11.370	2.152	11.479	1.821
Postneonatal mortality rate of normal-birthweight infants*b	2.480	.439	2.434	.323
B. Reduced Form Regressors				
Neonatal intensive care hospitals	.010	.004	.010	.003
Pediatricians	.478	.231	.495	.272
Abortion providers	.051	.028	.047	.024
Family planning clinics	.298	.114	.319	.128
Medicaid	.583	.493	.504	.500
WIC program	.090	.070	.082	.064
MIC projects	.051	.066	.059	.067
Fraction high school educated*	.735	.037	.605	.052
Female poverty*	.266	.043	.568	.081
Unemployment rate*	6.297	1.376	13.410	3.082
Labor force participation rate*	62.590	3.578	61.483	4.177
C. Production Function Regressors				
Prenatal care use, low-birthweight births*a	.732	.058	.587	.073
Prenatal care use, normal-birthweight births*b	.796	.046	.631	.072
Abortion rate*	24.726	7.511	49.609	20.182
Neonatal intensive care use, low-birthweight births[a]	10.175	2.772	9.434	2.653

Table 2, continued

	Mean	Std. Dev.	Mean	Std. Dev.
Neonatal intensive care use, normal-birthweight births[b]	.983	.323	.863	.342
Pediatricians	.478	.231	.495	.272
Family panning use	277.703	58.037	272.490	48.223
WIC use	263.664	84.454	269.440	79.733
AFDC for teenagers*	.010	.002	.044	.010
AFDC for adults*	.037	.013	.172	.045

Notes: (1) An asterisk (*) next to a variable means that it is race-specific.
(2) Unless otherwise indicated, white means and standard deviations are based on 51 observations and black means and standard deviations are based on 39 observations.
(3) Unless otherwise indicated, means and standard deviations are weighted by the race-specific total number of neonatal survivors in 1980.
(4) A superscript ([a]) indicates that it is weighted by the race-specific total number of low-birthweight neonatal survivors in 1980.
(5) A superscript ([b]) indicates that it is weighted by the race-specific total number of normal-birthweight neonatal survivors in 1980.

Table 2 contains their sample characteristics. For whites, the mean values and standard deviations are based on 51 observations (those of fifty states and the District of Columbia). For blacks, however, 12 observations are missing and thus they are based on only 39 observations. They are missing because states with a population of less than 20,000 blacks in 1980 and with less than 600 black births in that year are excluded from the black regressions for the obvious reason that the infant mortality rates of those states are unreliable.[3] The descriptive statistics regarding the dependent variables are weighted by the race and birthweight-specific total number of neonatal survivors.

The mean value of the postneonatal mortality rate of light neonatal survivors is five times larger than that of normal weight neonatal survivors irrespective of race. Although this difference can be considered to be very small compared to the case of neonatal mortality rate, there is still a large gap in the mortality rate between the two birth weight categories in the postneonatal period. This means that birth weight is still an important risk factor which affects the survival probability of infants who survive the first month of life successfully.

However, there is no difference at all in the birthweight-specific mortality rate between whites and blacks. But the overall postneonatal mortality rate which is not birthweight-specific is slightly larger for blacks (= 3.375 per thousand neonatal survivors) than for whites (= 2.880 per thousand neonatal survivors) because the fraction of black neonatal

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[3]States that are not included in the black regressions are following: Alaska, Hawaii, Idaho, Maine, Montana, New Hampshire, New Mexico, North Dakota, South Dakota, Utah, Vermont and Wyoming.

survivors of low birth weight (=0.104) is larger than that of whites (=0.045).[4] These two factors rationalize the fitting of separate regressions for white and black neonatal survivors, and by allowing the coefficients to vary between races and birthweight categories, we can determine which factors are particularly important in explaining the black and white differential in the infant mortality. What is more, in light of the nonrandom nature of postneonatal mortality rates vis-a-vis the total number of neonatal survivors, weighted regressions will be estimated to stabilize residual variances, where the set of weights is the square root of the race and birthweight-specific number of neonatal survivors. That is, to eliminate the influence of the total number of neonatal survivors, we inflate all the variables using those weights since small variances are associated with large number of neonatal survivors and large variances are associated with small number of neonatal survivors.

Table 1 and 2 contain the definitions and sample characteristics of the explanatory variables as well. The means and standard deviations reported in Table 2 are weighted by the race and birthweight-specific number of neonatal survivors in the case of regressors that are available on a birthweight-specific basis. In the case of all other regressors, the variable used for the weighting is the race-specific total number of neonatal survivors.

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[4]Let p_1 be the postneonatal mortality rate of light neonatal survivors and let p_2 be the postneonatal mortality rate of normal weight neonatal survivors. The overall mean (p) is given by

$$p = kp_1 + (1-k)p_2,$$

where k is the fraction of light neonatal survivors.

In these tables, the explanatory variables are divided into two groups, reduced form regressors and production function regressors. Reduced form regressors pertain to input price and availability, public program availability, and socioeconomic variables, while production function regressors pertain to the use of medical care and public programs. Following Grossman, Corman and Joyce (1988), this study employs race-specific regressors, wherever possible, which are set off with an asterisk. Also, this study utilizes quite a few regressors used in their study of neonatal mortality to see whether they exercise a similar or dissimilar influence on postneonatal mortality. Some variables which are relevant only in the postneonatal period are newly introduced in this study. In the case of the variables used by Grossman and his associates in their study of neonatal mortality, detailed descriptions and their sources can be found in Corman and Grossman (1985), and Grossman, Corman, and Joyce (1988). Therefore, the discussion here is confined to the variables that are newly introduced in this study of the determinants of postneonatal mortality rates.

Pediatrician availability is measured by the number of pediatricians per thousand children aged 14 or less in 1980. Data on the number of pediatricians are published in the American Medical Association's Physician Characteristics and Distribution in the U.S., 1981. It is the sum of the number of physicians whose specialties are pediatrics, pediatric allergy and pediatric cardiology. Data on the denominator are taken from Statistical Abstract of the United States, 1981.

Female poverty is measured by the fraction of women aged 15 through 44 with income below 200 percent of the poverty level in 1980. Data on poverty come from the 1980 Census of Population.

The female unemployment rate is the percentage of women aged 16 through 44 in the labor force who are unemployed in 1980. It is taken from the Census Bureau's 1980 Census of Population, Vol. 1 Characteristics of the Population, Chapter D Detailed Characteristics.

The female labor force participation rate is the percentage of civilian, noninstitutionalized women aged 16 through 44 who are in the labor force in 1980. The source of data on the labor force participation rate is the same as that on the unemployment rate. In the case of these two variables, seven observations for whites are missing because labor force status is not reported by race for seven states. Therefore, we can get information on the labor force status of the total population only for those states, which are Idaho, Maine, North Hampshire, North Dakota, Utah, Vermont, and Wyoming. However, the reason for not reporting race-specific labor force status is that the nonwhite populations in those seven states consist of a very small portion of total population. In light of this, the data on labor market variables of the total population for those states are merged with the race-specific data for other states.

In the case of the production function regressors, most of them are used by Grossman and his associates as determinants of neonatal mortality rates. They are employed in this study of postneonatal mortality rates without modifications. This study, however, employs one more medical care use variable, the use of pediatrician services, and one more public program use variable, AFDC use. The definition of the pediatrician use to be used in the production function is the same with the pediatrician availability to be used in the reduced form equations. It is because there are no data on visits per child.

This study employs two AFDC program use variables, that is, AFDC support for teenage mothers and for adult mothers to examine differential responses of mortality to these measures.[5] The AFDC for teenagers is given by the fraction of poor women aged 15 through 19 receiving public assistance in 1980. The denominator pertains to the total number of women aged 15 through 19 with family income less than 200 percent of the poverty level in 1980. Likewise, AFDC for adults is given by the fraction of poor women aged 20 through 44 receiving public assistance in 1980. Also, the denominator of this variable is the total number of women aged 20 through 44 with family income less than 200 percent of the poverty level in 1980. These two AFDC variables come from the five percent A Sample of the 1980 Census of Population and are included to see whether welfare program, which has been subject to a variety of criticisms, has any influence on the postneonatal mortality rate by affecting the behavior of mothers who comprise a part or a potential part of the target population of the program.

[5]We do not include the AFDC availability measure in the reduced form equations because preliminary study shows that it has not any explanatory power when we define the AFDC availability as the maximum amount payable to families with two recipients under state AFDC guidelines.

3.2 Empirical Estimates: Reduced Form Regressions

Using the variables defined earlier, reduced form and structural equations are estimated, and the results of reduced form regressions are presented first in this section. The estimation results of the structural equations will be discussed later in the following section. Table 5 and 6 present the estimated reduced form equations for postneonatal mortality of low-birthweight infants of whites and blacks, respectively. Results for postneonatal mortality of normal-birthweight infants are shown separately by race in Table 7 and 8.

In each table, the results of three regressions are shown because of collinearity problems. In the first equation of each table, the regressors are confined to three medical care availability measures and four public program availability measures. The medical care variables are neonatal intensive care hospitals, number of pediatricians, and abortion providers; the program variables are family planning clinics, Medicaid, Special Supplemental Food Program for Women, Infants, and Children (WIC), and maternal and infant care (MIC) projects. In addition to these regressors, the second equation includes two socioeconomic variables, education and poverty, and the third equation further includes the female unemployment rate and labor force participation rate.

Since the four socioeconomic variables considered in the reduced form equations create considerable collinearity problems, some comments may be in order. In the case of the first model, which includes no socioeconomic variable, the collinearity problem does not matter. However, when the schooling and poverty variables are added to the model, the problem becomes serious in terms of intercorrelation between regressors.

To be specific, in the white regressions, there is an almost exact linear relationship between the schooling variable and the intercept term because there is very little variation in the schooling variable. In the case of the black regressions, there exists a near linear dependence among the intercept term, schooling, and poverty variables. Likewise, including the female unemployment rate and labor force participation rate makes the problem all the more serious. In this model, a systematic relation exists among the intercept term, schooling, and the female labor force participation rate in the white regressions and among the intercept and poverty in the black regressions. Thus, to determine the degree of the problem solely in terms of the correlations among these regressors, it can be said that the collinearity problem is severe.[6]

However, the precision of the estimated coefficients of the variables in question is generally high. Therefore, the collinearity problem in this case is not as serious as it might be at first sight. Therefore, the mere fact that intercorrelations between a few regressors are high does not warrant further effort to moderate its harmful consequences. Nevertheless, in reporting the estimation results, the reader is cautioned not to accept the results blindly.

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[6]The degree of collinearity caused by these interrelationships can be confirmed by such a measure as condition number which is defined as the square root of the maximum to the minimum characteristic root of the moment matrix. In our case, for example, it is 17 for the first model. It jumps up to 134 after adding schooling and poverty in the second model. Adding further the female unemployment rate and labor force participation rate to the model changes the condition number to 165 for the white regressions and to 202 for the black regressions. About the condition number as a diagnostic tool to detect the degree of collinearity, see D. A. Belsley, E. Kuh, and R. E. Welsch, Regression Diagnostics: Identifying Influential Data and Sources of Collinearity, Wiley, New York, 1980, Chapter 3. Fomby et al.(1984) also presents a good discussion on this diagnostics and related topics.

Table 3

Postneonatal Mortality Rate Reduced Form Equations
Low-birthweight Neonatal Survivors, Whites

Explanatory Variable	(W-L-1)	(W-L-2)	(W-L-3)
Neonatal intensive	-117.275	-136.625	-64.228
care hospitals	(-1.34)	(-1.44)	(-.71)
Pediatricians	-3.901***	-3.245**	-1.835
	(-2.78)	(-2.23)	(-1.30)
Abortion providers	15.605	11.144	-11.403
	(1.33)	(.89)	(-.85)
Family planning clinic	2.043	-.274	-.695
	(.78)	(-.09)	(-.26)
Medicaid	-.214	-.329	.105
	(-.35)	(-.51)	(.17)
WIC program	7.842*	4.291	1.847
	(1.82)	(.95)	(.44)
MIC projects	-9.584**	-10.436**	-8.923**
	(-2.04)	(-2.20)	(-2.06)
Education*	-	2.043	-3.181
	-	(.17)	(-.29)
Poverty*	-	19.138**	28.706***
	-	(2.06)	(3.20)
Unemployment rate*	-	-	.601**
	-	-	(2.63)
Labor force	-	-	.314***
participation rate*	-	-	(2.95)
Constant	12.963	7.596	-14.789
	(8.89)	(.80)	(-1.34)
R^2	.246	.332	.475
Adjusted R^2	.123	.186	.326
F	2.000	2.267	3.202

Notes: (1) t-ratios in parentheses. An asterisk (*) next to a coefficient means it is statistically significant at the 10 percent level. ** is significant at the 5 percent. *** is significant at the 1 percent.

(2) An asterisk (*) next to a variable means it is race-specific.

Table 4

Postneonatal Mortality Rate Reduced Form Equations
Low-birthweight Neonatal Survivors, Blacks

Explanatory Variable	(B-L-1)	(B-L-2)	(B-L-3)
Neonatal intensive care hospitals	-253.004**	-262.992**	-195.796*
	(-2.03)	(-2.13)	(-1.84)
Pediatricians	-1.826	-1.710	1.018
	(-1.57)	(-1.33)	(.76)
Abortion providers	-.366	-12.735	-19.707
	(-.03)	(-.81)	(-1.42)
Family planning clinics	7.903***	8.614***	2.387
	(2.75)	(2.84)	(.79)
Medicaid	-.521	-.760	.014
	(-.82)	(-1.17)	(.02)
WIC program	-.933	-1.872	-1.923
	(-.20)	(-.39)	(.48)
MIC projects	-5.085	-3.809	-4.851
	(-.98)	(-.69)	(-1.05)
Education*	-	19.232	20.578**
	-	(1.60)	(2.03)
Poverty*	-	8.339	22.346***
	-	(1.11)	(3.04)
Unemployment rate*	-	-	.356***
	-	-	(2.84)
Labor force	-	-	.380***
participation rate*	-	-	(3.75)
Constant	13.018	-2.821	-39.757
	(8.10)	(.06)	(-2.98)
R^2	.241	.303	.544
Adjusted R^2	.069	.087	.358
F	1.403	1.401	2.922

Notes: (1) t-ratios in parentheses. An asterisk (*) next to a coefficient means it is statistically significant at the 10 percent level. ** is significant at the 5 percent. *** is significant at the 1 percent.

(2) An asterisk (*) next to a variable means it is race-specific.

Table 5

Postneonatal Mortality Rate Reduced Form Equations
Normal-birthweight Neonatal Survivors, Whites

Explanatory Variable	(W-H-1)	(W-H-2)	(W-H-3)
Neonatal intensive	-13.771	-26.178	-16.421
care hospitals	(-.76)	(-1.36)	(-.82)
Pediatricians	-.475	-.388	-.194
	(-1.56)	(-1.27)	(-.60)
Abortion providers	2.798	1.031	-1.172
	(1.14)	(.41)	(-.39)
Family planning clinics	.609	.303	.244
	(1.08)	(.48)	(.39)
Medicaid	-.002	-.073	-.046
	(-.01)	(-.55)	(-.33)
WIC program	1.224	.242	-.017
	(1.35)	(.26)	(-.02)
MIC projects	-2.405**	-2.823***	-2.652***
	(-2.42)	(-2.88)	(-2.71)
Education*	-	2.256	1.945
	-	(.93)	(.78)
Poverty*	-	5.047**	5.981***
	-	(2.65)	(2.98)
Unemployment rate*	-	-	.085
	-	-	(1.63)
Labor force	-	-	.027
participation rate*	-	-	(1.11)
Constant	2.539	-.042	-2.345
	(8.22)	(-.02)	(-.95)
R^2	.201	.321	.366
Adjusted R^2	.071	.172	.187
F	1.545	2.151	2.043

Notes: (1) t-ratios in parentheses. An asterisk (*) next to a coefficient means it is statistically significant at the 10 percent level. ** is significant at the 5 percent. *** is significant at the 1 percent.

(2) An asterisk (*) next to a variable means it is race-specific.

Table 6

Postneonatal Mortality Rate Reduced Form Equations
Normal-birthweight Neonatal Survivors, Blacks

Explanatory Variable	(B-H-1)	(B-H-2)	(B-H-3)
Neonatal intensive	-38.457*	-42.010**	-36.372*
care hospitals	(-1.81)	(-2.14)	(-1.84)
Pediatricians	-.387*	-.215	.015
	(-1.90)	(-1.03)	(.06)
Abortion providers	1.094	-1.507	-2.222
	(.46)	(-.59)	(-.85)
Family planning clinic	1.070**	.979**	.453
	(2.16)	(2.01)	(.80)
Medicaid	-.049	-.113	-.043
	(-.44)	(-1.07)	(-.38)
WIC program	.189	-.309	-.319
	(.23)	(-.39)	(-.41)
MIC projects	-2.238**	-2.474***	-2.557***
	(-2.55)	(-2.83)	(-2.97)
Education*	-	4.498**	4.608**
	-	(2.36)	(2.45)
Poverty*	-	3.240***	4.446***
	-	(2.67)	(3.21)
Unemployment rate*	-	-	.029
	-	-	(1.21)
Labor force	-	-	.033*
participation rate*	-	-	(1.74)
Constant	2.755	-1.616	-4.803
	(9.82)	(-.96)	(-1.91)
R^2	.282	.427	.485
Adjusted R^2	.120	.249	.275
F	1.737	2.401	2.311

Notes: (1) t-ratios in parentheses. An asterisk (*) next to a coefficient means it is statistically significant at the 10 percent level. ** is significant at the 5 percent. *** is significant at the 1 percent.

(2) An asterisk (*) next to a variable means it is race-specific.

Although neonatal intensive care hospital availability is particularly relevant in the neonatal period, the estimation results show that it still exerts a positive influence on the survival probability of infants after the first month of life. As expected, the estimated coefficient of this variable has a negative sign in all models. The size of impact, however, differs between whites and blacks, and between low-weight neonatal survivors and normal-weight neonatal survivors, although it is not so great overall. Not surprisingly, the impact of the availability of these hospitals is larger for low-birthweight infants regardless of race. As to the effect between races, it is stronger for the black neonatal survivors than for the white neonatal survivors, and the magnitude of the estimated coefficients of blacks are roughly two times larger than that of whites, regardless of birthweight category. Moreover, in the case of the black regressions, all the estimated coefficients of neonatal intensive care hospitals are statistically significant at the 5 percent or 10 percent level, whereas the precision of white coefficients are somewhat low. Even so, the t-ratios of three coefficients are greater than 1 for the white infants [(W-L-1), (W-L-2) and (W-H-2)].

Therefore, it seems that the effect of neonatal intensive care availability on the survival probability is not confined to the infants aged less than one month. Indeed, the estimation results suggest that the availability of neonatal intensive care exerts influences to some degree even in the postneonatal period, particularly with black infants. In this regard, it is worth noting that Corman, Grossman and Joyce (1988) report that the impact of this variable on neonatal mortality is sizable and significant for the white births whereas the impact on black births is not clear-cut. Combined with the results of this study, it is likely that the effect of neonatal intensive care hospital availability is exhausted mostly during the neonatal period in the case of white infants while it is distributed over the year after birth in the case of black infants.

To understand the meaning of estimated coefficients more concretely, the impacts of a one standard deviation change in neonatal intensive care availability are evaluated using the coefficient of neonatal intensive care hospitals with the highest t-ratio. For white infants, the estimation result indicates that a one standard deviation increase in neonatal intensive care availability lowers the overall white postneonatal mortality rate by 0.13 deaths per thousand white neonatal survivors, which amounts to 4.5 percent of the mean value in 1980. For black infants, the counterparts are 0.19 deaths per thousand black neonatal survivors and 5.6 percent, respectively.[7]

The signs of pediatrician availability are all negative except in models (B-L-3) and (B-H-3) and thus the number of pediatricians per thousand children as an availability measure has a negative influence on postneonatal mortality in general. Moreover, the coefficient is significant at the 1 percent level in model (W-L-1) and at the 5 percent level in models (W-L-2). But the coefficient of this variable is statistically and practically insignificant in other models. Therefore,

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[7]To translate the effect of a given change in the i^{th} regressor into the overall postneonatal mortality rate, the following formula is used. From note 4,

$$(\delta p/\delta x_i) = k(\delta p_1/\delta x_i) + (1 - k)(\delta p_2/\delta x_i)$$

where x_i is the i^{th} regressor in the reduced form equation.

it is likely that the impact of pediatrician availability on infant mortality is limited to white low-birthweight infants.

The effect of abortion providers is not clear at all. In the case of the white regressions, the sign is negative only in the third model for both birthweight categories. In the case of black regressions, the signs are all negative except in model (B-H-1). No matter what the signs are, all the estimated coefficients are not significantly different from zero. Therefore, unlike the strong, negative relationship between abortion and neonatal mortality which has been repeatedly documented and emphasized by Grossman and his colleagues (Grossman and Jacobowitz 1981; Corman and Grossman 1985; Corman, Joyce and Grossman 1987; Joyce 1987a; Corman, Grossman and Joyce 1988), the relationship between abortion and postneonatal mortality, if any, is very weak.

With respect to the effect of public policy availability variables, the estimation results are somewhat puzzling overall. The coefficient of family planning clinics availability is not significant in all white models, and the sign is negative only in two out of six cases. For black infants of both birthweight categories, the signs are all positive, and the estimated coefficients are significant at the 1 or 5 percent level in the first and second specifications, which is contrary to expectations. The dichotomous measure for Medicaid coverage of prenatal care for first-time pregnancies appears to be linked negatively with postneonatal mortality for the most part, although neither statistical significance nor practical significance can be inferred from the estimates.

The WIC program coefficients are positive in almost all equations of white infants and insignificant except in model (W-L-1) where the coefficient is significant at the 10 percent level. In the case of the black regressions, however, the signs are all negative with one exception, although the precision of the estimates are very low. This result may suggest that the WIC program coverage is associated with higher postneonatal mortality rates or that the influence of WIC program coverage on postneonatal mortality is, at best, negligible and thus such coverage is not qualified as a determinant of postneonatal mortality. Nevertheless, the reason for not omitting the WIC program availability in this research is that the program is regarded by health policy makers and Congress as a major policy instrument along with Medicaid to reduce the infant mortality rate.[8] In light of this, the positive sign or the insignificance of the impact of nutrition program may imply that this variable should be regarded as endogenous from the viewpoint of society, rather than as exogenous from the individual viewpoint. If this is correct, nutrition program coverage would be expanded if infant mortality is higher. In that case, their relationship would be either positive or difficult for researchers to identify.

The availability of maternal and infant care projects measured by the fraction of counties in a state served by the projects exhibits a negative association with the postneonatal mortality in 1980. The

[8] It is not difficult to see why Congress accepts the positive effect of maternal nutrition program on infant survival probability. The researches which report the positive relationship between the two abound. See Kotelchuck et al. (1984). Also Grossman and his associates report the negative and significant impact of nutrition program on neonatal mortality in the literature cited earlier. The issue of nutritional supplements and their effect on birth outcomes and infant survival, however, is controversial and not resolved yet. See, for example, Rush (1984) for the comments on the finding of Kotelchuck et al. (1984).

coefficient of the MIC projects availability has a negative sign in all reduced form equations without exception. In addition, they are highly significant in many cases and very stable in that the size of the impacts are virtually the same in all models in spite of a different number of regressors in each one. MIC availability also seems to have more significant effect on low-birthweight infants than on normal-birthweight infants, and it has a slightly larger impact on the white infants. Specifically, a one standard deviation increase in the MIC availability measure is associated with a reduction of the overall white postneonatal mortality rate by 0.21 deaths, which amounts to 7.2 percent of the mean level in 1980, whereas in the case of blacks the impacts are 0.19 deaths and 5.7 percent, respectively. The estimation result which Corman, Grossman and Joyce (1988) get does not show any decisive picture on the impact of MIC availability on neonatal mortality. In light of this, it seems that the MIC project is more effective in the postneonatal period than in the neonatal period.

Contrary to expectations, the estimation results indicate that the postneonatal mortality rate increases as the fraction of women with 12 or more years of education increases. Moreover, in the black regressions, three out of four are significant at the 5 percent level [(B-L-3),(B-H-2) and (B-H-3)]. In the case of white regressions, preliminary study shows that deleting other three socioeconomic variables which are closely intercorrelated with schooling variable results in negative signs in some cases, although the coefficients are not significant at all. The story is not the same for blacks. No matter what the specification is, the sign of schooling coefficient turns out to be positive. Therefore, the effect of education on postneonatal mortality is not clear or nil at best in the case of whites and it is definitely positive in the case of blacks, although the lack of variation in schooling qualifies this conclusion.[9]

It is well known that poverty and its related problems lie at the heart of the phenomenon of infant mortality. Certainly, the results of this study are quite consistent with this well-known fact. All coefficients of the female poverty measure are positive as expected and are significantly different from zero at the 1 percent or at the 5 percent level except in one model. Like many other determinants of postneonatal mortality, this factor also has a larger effect on postneonatal mortality of low-birthweight infants. But, unlike other cases, the impacts of poverty are not so different between races. The estimated coefficient means that a 1.0 percentage point increase in female poverty measure leads to an increase of 0.07 deaths or 2.4 percent of the white postneonatal mortality and to an increase of 0.06 deaths or 1.9 percent of the black postneonatal mortality in 1980. Thus, in fact, poverty affects white infants a bit more unfavorably.

The female unemployment rate also shows the expected positive sign in all models, and the coefficients are significant at the 1 percent or

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[9]In view of existing evidences on the effect of the level of educational attainment on the infant mortality, these results are puzzling. Using the same specification of schooling as employed in this study, for instance, Corman et al. (1988) find a negative and significant relationship between schooling and the neonatal mortality rate. Also, low educational attainment of parents has been found to be correlated with the postneonatal mortality (McCormick, 1985). In conjunction with the results of this study, however, it may be noted that the possibility of no effect or a negative effect of schooling on infant health is not ruled out in the household production function approach to consumer behavior. See, for example, Grossman (1972).

at the 5 percent in the case of the low-birthweight equations. Although the coefficients in models for the normal-birthweight infants are statistically insignificant, they are estimated with the t-ratios greater than 1.0. Also, the estimation results suggest that the overall impact of female unemployment is larger for white infants than for black infants. To be specific, for whites, a 1.0 percentage point increase in the female unemployment rate raises postneonatal mortality rate by 0.11 deaths or by 3.8 percent relative to the white mean postneonatal mortality rate in 1980. In the case of black infants, the same change leads to an increase in postneonatal mortality of 0.06 deaths or 1.8 percent relative to the black mean postneonatal mortality in 1980.

We get the very similar results regarding the impact of female labor force participation rate on postneonatal mortality. That is, all the coefficients have positive signs and they are highly significant in the models for the low-birthweight equations. However, unlike the unemployment rate, the impact of this variable is larger for blacks than for whites. The estimated coefficient implies that a 1.0 percentage point increase in the labor force participation rate of the white women is associated with an increase of 0.04 deaths or 1.3 percent increase in the white postneonatal mortality rate. The counterpart for blacks is 0.07 deaths or 2.0 percent.

From these results as to the labor market variables, it is likely that, to some extent, the female unemployment rate and labor force participation behavior do affect infant health in general and survival probability in particular. There may be no difficulty accepting the result as to the effect of female unemployment because a high female unemployment rate is ultimately translated into weak economic condition of working mothers, little economic resources to be spent on their children, and low quality of children. In the case of the female labor force participation rate, however, the result reported in this study may not be so easy to be accepted in particular by those who support the view that the labor force participation behavior of women does not affect or favorably affects children. As to the effect of mother's labor supply on children, it has been argued that there is no reason to believe that the mere fact a mother works has any clearly predictable effects on her children.[10] The consequence of the rise in women's labor force participation on the development of their children and on the diverse dimensions of children's well-being is surely a complicated issue which is not amenable to a decisive conclusion. In our own context, the result suggests that a decrease in mother's time spent in child care dominates a beneficial effect on the quality of children including health through her contribution to family earnings. This finding of the larger substitution effect is not consistent with the evidence of the larger income effect reported by Rosenzweig and Schultz (1983a). Although it may be argued that state level cross-section data are not adequate to address this kind of question, the result reported in this study can be viewed as one piece of evidence which qualifies the validity of the argument that the female labor force participation behavior does not affect the development of children, at least with respect to the effect on infant health.

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[10] See Francine D. Blau, and Marianne A. Ferber, The Economics of Women, Men and Work, Prentice-Hall, New Jersey, 1986, chapter 5 and the literature cited therein.

3.3 Empirical Estimates: Production Functions

It is a major aim of this research to estimate production functions of birthweight-specific postneonatal mortality rate. The estimation results of postneonatal mortality rate production functions for low-birthweight infants of whites and blacks are reported in Tables 7 and 8. They are followed by the estimated production functions for normal-birthweight infants in Tables 9 and 10. Shown in each table are the results of the two models that are estimated via both ordinary least squares and two-stage least squares.[11]

In the first specification of structural postneonatal mortality rate production functions, the regressors are limited to the medical care variables, which are prenatal care use, the abortion rate, neonatal intensive care use, and pediatrician service use as defined in Table 1. As mentioned earlier, one justification for including these various medical care variables is that the prompt initiation of prenatal care may raise the quantity and quality of medical care received in the neonatal and postneonatal periods. In addition to these regressors, the second model includes three program use variables, which are family planning use, WIC program use, and Aid to Families with Dependent Children (AFDC) program use. In this model, two AFDC variables are included to examine differential response of postneonatal mortality to AFDC use measures for teenage mothers compared to adult mothers. All variables that are included

[11]In estimating the production functions by TSLS, the regressors in models (W-L-3) and (B-L-3) are employed as instruments in the low-birthweight neonatal survivors equations and, likewise, those in models (W-H-3) and (B-H-3) are employed as instruments in the normal-birthweight neonatal survivors equations.

in the production functions are endogenous.[12]

In estimating the model as formulated in Chapter II, the usual estimation technique is the two-stage least squares method following Rosenzweig and Schultz (1982, 1983a, 1983b), as mentioned earlier. The rationale for that is labeled in the literature as "reverse causality" due to "heterogeneity", which causes estimates obtained by ordinary least squares to be biased and inconsistent. The use of medical care affects health outcome but the causation also flows in the reverse order. That is, undesirable biological endowment of the fetus or poor health of the infant induces greater use of medical services but, in general, they are unobservable to researchers. As a result, the use of medical care is not independent of the disturbance term which includes the unobservable endowment as one component.

Corman and Grossman (1985), however, indicate that the production function regressors are independent of the disturbance term when the infant risk factor of birthweight is held constant. Based on their report, we can surmise that all the medical care and public program use variables that are labeled as endogenous variables are not correlated with the disturbance term since the dependent variables employed in this study are all birthweight-specific. If so, ordinary least squares is appropriate for the estimation of the production functions.

Indeed, the results of Wu-Hausman specification error tests support

[12]The number of pediatricians as a measure of pediatrician service use should be an endogenous variable like all other arguments of production function. However, since a separate measure of pediatrician use is not available, the pediatrician variable is treated as exogenous in the production function.

Table 7

Postneonatal Mortality Rate Production Functions
Low-birthweight Neonatal Survivors, Whites

Explanatory Variable	OLS (W-L-4)	TSLS (W-L-5)	OLS (W-L-6)	TSLS (W-L-7)
Prenatal care use*[a]	-7.038	-31.065**	-3.365	-18.934
	(-1.32)	(-2.27)	(-.68)	(-.94)
Abortion rate*	.023	.015	.052	.072
	(.50)	(.07)	(1.30)	(.61)
Neonatal intensive care use[a]	.025	.633**	-.044	.512
	(.23)	(2.11)	(-.45)	(1.23)
Pediatricians	-3.131**	-3.068	-2.693**	-3.425
	(-2.24)	(-1.54)	(-2.25)	(-1.57)
Family planning use	-	-	-.012**	-.022
	-	-	(-2.46)	(-.92)
WIC program use	-	-	.008**	.019
	-	-	(2.46)	(1.41)
AFDC, teen-agers*	-	-	604.196***	285.031
	-	-	(3.81)	(.46)
AFDC, adults*	-	-	-80.542**	-73.964
	-	-	(-2.38)	(-.51)
Constant	17.212	28.760	12.775	21.074
	(4.37)	(3.50)	(3.25)	(1.64)
R^2	.135	.157	.472	.252
F	1.80	2.15	4.70	1.77
Wu test F		5.678		1.289

Notes: (1) t-ratios in parentheses. For the TSLS, the t-ratio is coefficient divided by asymptotic standard error. An asterisk (*) next to a coefficient means it is statistically significant at the 10 percent level. ** is significant at the 5 percent. *** is significant at the 1 percent.

(2) An asterisk (*) next to a variable means it is race-specific. A superscript ([a]) indicates that the variable is birthweight-specific.

Table 8

Postneonatal Mortality Rate Production Functions
Low-birthweight Neonatal Survivors, Blacks

Explanatory Variable	OLS (B-L-4)	TSLS (B-L-5)	OLS (B-L-6)	TSLS (B-L-7)
Prenatal care use*[a]	-6.846	-15.451	-6.855	-26.519
	(-.87)	(-1.08)	(-.94)	(-1.11)
Abortion rate*	.103**	.077	.152***	.197*
	(2.27)	(1.31)	(3.42)	(1.82)
Neonatal intensive	-.273	-.203	-.361	-1.163
care use[a]	(-.98)	(-.40)	(-1.39)	(-1.29)
Pediatricians	-8.535***	-7.686**	-6.344**	-11.539**
	(-3.15)	(-2.56)	(-2.55)	(-2.09)
Family planning use	-	-	-.012	.064
	-	-	(-.82)	(-1.03)
WIC program use	-	-	-.003	-.012
	-	-	(-.42)	(-.83)
AFDC, teen-agers*	-	-	247.590***	326.230*
	-	-	(3.69)	(1.71)
AFDC, adults*	-	-	-28.486*	9.257
	-	-	(-1.87)	(.20)
Constant	20.086	25.346	15.273	6.729
	(4.14)	(3.59)	(2.74)	(.46)
R^2	.242	.201	.542	.259
F	2.71	2.13	4.44	1.31
Wu test F		.781		.507

Notes: (1) t-ratios in parentheses. For the TSLS, the t-ratio is coefficient divided by asymptotic standard error. An asterisk (*) next to a coefficient means it is statistically significant at the 10 percent level. ** is significant at the 5 percent. *** is significant at the 1 percent.

(2) An asterisk (*) next to a variable means it is race-specific. A superscript ([a]) indicates that the variable is birthweight-specific.

Table 9

Postneonatal Mortality Rate Production Functions
Normal-birthweight Neonatal Survivors, Whites

Explanatory Variable	OLS (W-H-4)	TSLS (W-H-5)	OLS (W-H-6)	TSLS (W-H-7)
Prenatal care use*[a]	-3.046**	-6.252***	-2.243	-4.758
	(-2.25)	(-2.97)	(-1.57)	(-1.32)
Abortion rate*	-.009	-.007	-.009	-.016
	(-.91)	(-.52)	(-.97)	(-.63)
Neonatal intensive	.097	.711*	-.022	.625
care use[a]	(.51)	(1.84)	(-.11)	(1.14)
Pediatricians	-.218	-.193	-.147	-.180
	(-.73)	(-.52)	(-.52)	(-.44)
Family planning use	-	-	-.0006	.002
	-	-	(-.55)	(.31)
WIC program use	-	-	.0007	.0007
	-	-	(.95)	(.29)
AFDC, teen-agers*	-	-	110.183***	65.952
	-	-	(3.03)	(.62)
AFDC, adults*	-	-	-10.218	-3.447
	-	-	(-1.26)	(-.12)
Constant	5.123	7.573	3.884	5.001
	(4.56)	(4.22)	(3.14)	(1.85)
R^2	.132	.188	.350	.262
F	1.74	2.67	2.83	1.86
Wu test F		2.739		.668

Notes: (1) t-ratios in parentheses. For the TSLS, the t-ratio is coefficient divided by asymptotic standard error. An asterisk (*) next to a coefficient means it is statistically significant at the 10 percent level. ** is significant at the 5 percent. *** is significant at the 1 percent.

(2) An asterisk (*) next to a variable means it is race-specific. A superscript ([a]) indicates that the variable is birthweight-specific.

Table 10

Postneonatal Mortality Rate Production Functions
Normal-birthweight Neonatal Survivors, Blacks

Explanatory Variable	OLS (B-H-4)	TSLS (B-H-5)	OLS (B-H-6)	TSLS (B-H-7)
Prenatal care use*[a]	-5.005**	-5.479*	-5.991**	1.664
	(-2.48)	(-1.80)	(-2.63)	(.17)
Abortion rate*	.006	-.005	.006	-.022
	(.52)	(-.33)	(.48)	(-.93)
Neonatal intensive	-.113	.698	-.603	.592
care use[a]	(-.21)	(.82)	(-1.08)	(.36)
Pediatricians	-1.357	-.669	-1.350	.555
	(-1.66)	(-.65)	(-1.61)	(.24)
Family planning use	-	-	.001	-.014
	-	-	(.23)	(-.76)
WIC program use	-	-	-.004*	-.002
	-	-	(-1.86)	(-.43)
AFDC, teen-agers*	-	-	22.270	-37.434
	-	-	(1.20)	(-.84)
AFDC, adults*	-	-	3.101	-.839
	-	-	(.69)	(-.06)
Constant	8.251	8.058	8.502	10.072
	(5.80)	(3.93)	(5.33)	(2.86)
R^2	.220	.178	.408	.236
F	2.40	1.84	2.59	1.16
Wu test F		.655		.971

Notes: (1) t-ratios in parentheses. For the TSLS, the t-ratio is coefficient divided by asymptotic standard error. An asterisk (*) next to a coefficient means it is statistically significant at the 10 percent level. ** is significant at the 5 percent. *** is significant at the 1 percent.

(2) An asterisk (*) next to a variable means it is race-specific. A superscript ([a]) indicates that the variable is birthweight-specific.

this anticipation by and large (Wu 1973; Hausman 1978; Nakamura and Nakamura 1981).[13] For the white regressions, the null hypothesis of no correlation between production function regressors and disturbance term can be rejected only in the first specification of the low-birthweight equations [model (W-L-4) of table 7] in which the right-hand side variables are confined to the medical care variables. In this case, the test value is 5.678, and since the critical F value for 3 and 43 degrees of freedom at the 1 percent level of significance is 4.275, the hypothesis can be rejected at the 1 percent level. In other cases, however, it is impossible to reject the null hypothesis. In the case of the first model in the high-birthweight equations in which the regressors are also confined to the medical care use variables, the computed F value of 2.739 is slightly lower than the 5 percent critical value of 2.825. For the second specification in which the public program variables are included in addition to the medical variables, the test values are obviously insignificant, implying that the biological endowment is not correlated with the regressors. In the case of black regressions, the test values are substantially lower than the critical value in every model. Therefore, all the explanatory endogenous variables in the black regressions are independent of the disturbance term.

These results of specification error tests suggest that, overall, birth weight is a good proxy for the health endowment in the analysis of postneonatal mortality, but there may be risk factors other than birth

[13]In the previous footnote, it is indicated that the number of pediatricians is exogenous, although it is conceptually endogenous. Therefore, in performing the specification error tests, it should be excluded to avoid the perfect collinearity.

weight that cause the reverse causality problem for whites and are not controlled for in this study. Based on these results, the TSLS estimates are emphasized only in model (W-L-4) for whites, and the OLS results alone are considered in the case of all other regressions.

The estimation results show that the use of prenatal care as an important determinant of infant mortality of normal-birthweight infants, which Grossman et al. (1988) document for neonatal mortality, exerts its influence to some degree in the postneonatal period also. This implies that an early detection of physical problems associated wit pregnancy induces greater use of the perinatal and postnatal medical care services. For whites, all the estimated coefficients have negative signs irrespective of birthweight category. But the impacts of the prenatal care use are more significant for infants of normal birth weight in terms of the overall postneonatal mortality rate. For example, a 1.0 percentage point increase in the prenatal care use for light neonatal survivors results in a decrease in the overall postneonatal mortality rate of whites by 0.01 deaths which amounts to only 0.5 percent of the rate in 1980, whereas the same change in this variable for the normal-birthweight infants lowers the rate by 0.06 deaths or 2.0 percent of the rate in 1980. Thus, if the fraction of normal-birthweight births for which prenatal care began in the first trimester of pregnancy rises from 0.8(= the mean in 1980) to 0.9 and, of course, if the effect is linear as is assumed, the white postneonatal mortality rate would reduce by a fifth.

Also, comparing the OLS estimates with the TSLS estimates confirms that the former suffer from downward bias due to reverse causality. The TSLS estimate is roughly four times larger in absolute value than the OLS estimates in the case of low-birthweight neonatal survivors equation. Thus, causality which moves in opposite directions does matter in this case of prenatal care use, although the magnitude of bias is by no means dramatic.

For blacks, the results are quite similar except that the OLS estimates of the impact of prenatal care use are not distorted by reverse causality. Of course, for the low-birthweight infants, all the signs are correctly negative and the TSLS estimates are much larger in absolute value than the OLS estimates. But they are not statistically significant. For black neonatal survivors of normal weight, however, the coefficients of prenatal care use not only have the correct negative signs in both models but are significant at the 5 percent level of significance. In this case, the result suggests that a 1.0 percentage point increase in prenatal care use would result in a 2.5 percent decrease in the normal-birthweight postneonatal mortality rate in 1980, which is translated into a 1.5 percent decrease in the overall rate.

With respect to the abortion rate, the estimated coefficients of the white regressions for infants of low birth weight are not significant and the signs are all positive. In the case of normal-birthweight infants, the coefficients are all negative but insignificant. In the black regressions also, the abortion rate is positively related to the postneonatal mortality of low-birthweight infants but, unlike the white regressions, the impacts are statistically significant at the 1 or 5 percent. Similarly, in the normal-birthweight equations, the signs are positive also but the coefficients are not significant at all. Therefore, based on these results, it can be said that, in general, abortion is not an important determinant of postneonatal mortality rate and this is consistent with the results obtained earlier in the above section.

The effect of neonatal intensive care use on the postneonatal mortality is not clear, particularly for whites. The TSLS estimates have the wrong positive signs and are statistically significant at the 5 percent [(W-L-5]) and at the 10 percent [(W-H-5)]. On the other hand, the OLS estimates in the second model have negative signs but are insignificant by any standards. In the case of blacks, a direction of the impact seems to be clear; all the OLS estimates have negative signs but the precision is not enough to accept that direction with confidence. It is not reasonable to expect a large negative impact of this variable on postneonatal mortality because the effect of the use of neonatal intensive care is more relevant to the neonatal period. Thus, on the whole, the results may not be considered implausible, although positive and significant impact on the white infants is not easy to be rationalized.

With respect to the effect of the pediatrician use variable, the results are not much different from those we get from the estimation of the reduced form regressions in the case of whites. That is, a statistically significant negative relationship between the pediatrician use measure and postneonatal mortality appears to be confined to low-birthweight infants. This is reinforced by the estimation results of production functions because, for blacks also, the production function coefficients of pediatrician use are statistically significant at the 1 percent level or 5 percent level, regardless of the estimation technique. In addition, the estimated coefficients suggest the effect is about two times larger for blacks than for whites. Therefore, for both races, the effects of the pediatrician use measure are concentrated on the infants of low birth weight.

Family planning and WIC program use variables have trivial effects on the postneonatal mortality rates. Some coefficients are statistically significant at the 5 or 10 percent level, but they have no practical meaning irrespective of the sign pattern. For instance, although the estimated coefficient of organized family planning use has a correct negative sign and is significant at the 5 percent level for white infants of low birthweight, a one standard deviation change or a 20 percent change in family planning use lowers the overall postneonatal mortality by 0.03 deaths which is translated into only 1.0 percent of the rate in 1980. Of course, this does not indicate that the public programs particularly aimed at the poor and their children have no effect on infant health. Instead, it should be regarded as a warning of incorrect definitions or specifications of the variables involved. Admittedly, the wrong sign and/or the failure to pin down the relationships among the variables involved are routine in this kind of research which uses a lot of hard-to-measure variables.

Unlike the two program measures mentioned above, the AFDC use variables are estimated in general to exert a statistically and practically significant influence on the mortality of infants who survive the first month. With regard to the AFDC support for teenagers, the signs are uniformly positive and the coefficients are highly significant with an exception of model (B-H-6). In the case of adults, the coefficients have negative signs and are significant at the 5 or 10 percent level in the low-birthweight neonatal survivors equations.

The positive sign of AFDC coefficients for adolescent mothers is somewhat impressive because this result can be interpreted as another adverse side effect of AFDC which has been subject to extensive criticism over the last two decades for a variety of reasons, notably in relation to its effect on the labor supply. Of course, we have to be cautious not to be so quick in concluding that the AFDC program itself has been a drag upon the gains in the survival probability of infants. Clearly, we cannot rule out the possibility that a simple positive sign may mask the complexity and diversity of factors involved. There may be more direct and significant relationships between the postneonatal mortality and other variables that are correlated with AFDC use measure for teenage mothers.

Nevertheless, the response of postneonatal mortality to the change in AFDC use for teenagers cannot be considered to be trivial, particularly for whites. Rather, the impact on the white postneonatal mortality is even dramatic. The estimated coefficients suggests that a 1.0 percentage point change in AFDC use measure for teenage mothers, say, from 0.010 to 0.020 for whites and from 0.044 to 0.054 for blacks, would increase postneonatal mortality by 1.3 deaths per thousand neonatal survivors or by 46.0 percent of the overall postneonatal mortality rate in 1980 for whites and by 0.5 deaths or by 12.2 percent of the overall postneonatal mortality rate in 1980 for blacks. Thus, if we interpret the estimation result somewhat mechanically, the white postneonatal mortality rate would be reduced by a half by restricting the eligibility of the AFDC program to adult mothers, putting the feasibility of such restrictions aside.

One possible and, perhaps, fashionable factor that can be considered in explaining the positive sign and sizable impact of the AFDC use measure for teenagers may be the upward trend in teenage pregnancy outside marriage. That is, AFDC availability may induce at least some teenagers to give birth to unwanted infants. Unplanned pregnancy of teenagers, of course, can be terminated by legal or illegal abortion. However, the decision-making of pregnant teenagers may also be influenced, at least to a limited extent, by the AFDC program since the expectation that the program would be available after birth may render the behavior of unwed teenage mothers more flexible and responsive to this opportunity.

In the case of live births to unwed mothers, several factors can affect the survival probability of infants adversely. First of all, it is well known that the majority of premarital pregnancies are neither planned nor wanted (Wilson and Neckerman 1986, p.243) and that unplanned and unwanted births contribute to high infant mortality. In particular, out of wedlock births have been shown to be associated with higher rates of postneonatal mortality (Taffel 1984). It is well known also that infant and maternal mortality are highest among teenage mothers. Along with these two factors, the poor economic condition of single teenage mothers, who have little economic resource to support children, can contribute to lowering the survival probability of infants. What is more, in light of the fact that the class distribution of unwanted births is highly skewed toward the lower income groups in general, the interaction of the poverty of a single teenage mother and the birth of an unwanted child can seriously affect the infant's survival probability in an adverse manner. Therefore, the view that AFDC support for teenagers may be a contributing factor to the increase of postneonatal mortality is not totally unreasonable if fertility among poor youths aged 15 to 19 does respond to welfare.

At this point, it should be admitted that it is easy to blame welfare but it is not so easy to prove the validity of this assertion. Thus, an "explanation" of the positive sign here is problematic in the sense that it is not gone through a test procedure to confirm whether or not fertility among unmarried youth is really sensitive to the measurable dimensions of welfare, which is difficult to do within the limited scope of this research. For the above assertion to be plausible at least, there should be an established relationship between welfare and teenage fertility. In this regard, Moore and Caldwell (1977) report that there is no relationship between AFDC benefit levels and out-of-wedlock birth based on state level cross-section data.[14] Similarly, Ellwood and Bane (1985) report that they can not find little evidence to support the proposition that welfare and fertility among unmarried women are linked. Thus, according to the results they get in their attempt to identify the relationship between AFDC and family structure, AFDC seems to have little impact on childbearing among unmarried women. Then, the above interpretation of the positive sign and sizable impact is at odds with the evidences they suggest. If their findings which are supported by many researchers, notably by Wilson and Neckerman (1986), do capture the true picture of reality, the above explanation which appeals to the effect of welfare on fertility among youth simply breaks down, of course.

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[14]Although not reported in the text, this study could not find any relationship between AFDC benefit levels and postneonatal mortality in the preliminary research, either, as indicated in note 5.

In a review on this and other related topics, however, O'Neill (1986) criticizes their work on the ground that they did not account for the economic alternatives to cash transfer program and supports the view that welfare have affected significantly the behavior of the population with low schooling attainment and little economic resources. Therefore, at the moment, what can be said is that a further analysis is called for since, as she puts it, no definitive study of the effect of welfare on family structure has been done because of the demanding data requirement.[15]

Another problem related to the above discussion is that it is by no means clear why the effect is much larger for whites than for blacks. If out-of-wedlock births of teenagers which are "induced" by welfare does affect the survival probability adversely, the response of blacks should be expected to be more sensitive, because the illegitimate birth rate of blacks is much higher than that of whites.[16] Again, with a somewhat narrow focus of this research, the question cannot be answered adequately.

With respect to the estimation result for the AFDC for adult mothers, it seems to be a sharp contrast to those for teenage mothers. Although, the impact is much smaller than that of AFDC support for teenage mothers, the welfare program coverage for adult mothers and their children affects favorably the infant's survival probability and, to some extent, contributes to the decrease in the postneonatal mortality. Strictly

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[15]June O'Neill, Transfers and Poverty: Cause and/or Effect?, Cato Journal, Vol. 6, No. 1 (Spring/Summer 1986), p.68.

[16]In 1980, the illegitimacy ratio for black women aged 15 to 19 is 851.5, whereas the counterpart for whites is 329.8 (Wilson and Neckerman, 1986, p. 236, Table 10.2).

speaking, however, the effect of AFDC for adult mothers on postneonatal mortality is not clear because the variation in this variable is not independent of the variation in AFDC for teenagers. Preliminary study shows that the sign pattern of AFDC for adults turns out to be volatile and the coefficients are hardly significant by any standards in most cases when AFDC for teenagers is omitted from the equation. In contrast, the coefficient of AFDC for teenagers retain the positive sign with still high precision and there is no wide swing in the magnitude of coefficient in the estimation without AFDC for adults. In view of this, therefore, the estimated coefficient of AFDC for adults is not reliable, whereas the individual effect of AFDC for teenagers seems to be rather precise, although it is not without difficulty in interpretation and thus its validity can be questioned.

CHAPTER IV

CONCLUSION

4.1 Summary of Findings

Postneonatal mortality has been largely ignored in the analysis of the infant mortality rate for some good reasons. Recent slowdown of the rate of decline in infant mortality coupled with some socio-demographic phenomena, however, requires more meticulous investigations on the determinants of infant mortality. Although it is too soon to judge whether or not the slowdown is a temporary break from the downward movement of the last two decades, it is enough to attract attention. Therefore, in an effort to understand the overall picture of infant mortality in the U. S. more thoroughly, this study analyzes the determinants and correlates of postneonatal mortality using the 1980 National Infant Mortality Surveillance data on the race- and birthweight-specific postneonatal rates. Based on the economic models of the family and household production function, the birthweight-specific postneonatal mortality rate production functions and their reduced form equations are estimated.

In the estimation of the reduced form equations, such exogenous variables as medical care availability, public program availability, and socioeconomic factors are considered to capture the effects of these variables on the birthweight-specific postneonatal mortality rates. Estimates of the coefficients of medical care variables reveal that neonatal intensive care hospital availability and pediatrician availability are important determinants, particularly for the infants of low-birthweight. Abortion availability which is an important determinants of neonatal mortality, however, does not have an explanatory power in the analysis of postneonatal mortality.

Of all the public programs considered in this study, the MIC project availability measure is the only one that exhibits a statistically significant negative relationship with the postneonatal mortality rates. Family planning clinics, Medicaid, and WIC program availability do not play important roles in explaining the variation in postneonatal mortality, regardless of race or birthweight category. This does not necessarily mean that these public programs have not been efficacious against the incidence of infant deaths. Possibly, it may be due to a wrong specification of a variable in question or a wrong functional form, which does not allow to estimate the effects of these programs on the infant's survival probability properly.

In general, socioeconomic variables are estimated to have statistically significant relationships with postneonatal mortality. One exception is the schooling variable which has insignificant coefficients with wrong signs in most cases. The measure of poverty is positively associated with the postneonatal mortality rate for both races, which is consistent with prior expectations. Also, the female unemployment rate is positively related to postneonatal mortality, reflecting the stresses and a reduction of income associated with an unemployed status lower the infant survival probability to some extent. The direction of the impact of female labor force participation rate on the infant survival is ambiguous a priori because the substitution and income effects have the opposite implications on the health of children. The results of this study suggest a positive income effect on the survival probability is outweighed by a negative substitution effect for both races, implying that mother's labor force participation affects infant's health adversely, particularly if an infant is born with low birth weight.

Employing these regressors as instruments, the structural production functions are estimated by two-stage least squares to remove detrimental effects of the heterogeneity bias due to reverse causality. Consistent with the views expressed by Corman and Grossman (1985), however, the specification error tests reveal the bias is not serious for the most part because this study directly controls for birth weight as a proxy for health endowment which is a source of potential bias. Therefore, except the case of the white infants of low birth weight in which reverse causality does matter, the production function regressors are not correlated with the disturbance term and thus ordinary least squares is an appropriate technique to estimate the structural model.

Of the medical care variables considered in the estimation of production functions, the use of prenatal care and pediatrician service show negative relationships with postneonatal mortality. In the case of prenatal care, it appears to be still important as a determinant of infant mortality in the postneonatal period, irrespective of race. In addition, the results show that it has a larger impact on the survival probability of normal-birthweight infants. On the other hand, pediatrician service use has a much larger effect on the low-birthweight infants of both races. Unlike the availability measure of neonatal intensive care hospitals, its use measure has little effect on the infant's survival prospect in the postneonatal period. Likewise abortion rate is not related with the postneonatal mortality rate.

The variation in the use measures of public programs does not explain the variation in the postneonatal mortality rate. The coefficients of family planning and WIC program use variables reveal that they have little effect the infant's survival prospect after the first month of life. AFDC support for teenagers, however, is estimated to exert a great influence upon postneonatal mortality. A strong positive relationship between welfare and postneonatal mortality may be due to the increasing trends in illegitimate births that are allegedly induced by welfare. But this interpretation is hardly definitive in view of the conflicting evidences suggested in the received literature on the relationship between welfare and family structure. Although AFDC for adults is estimated to have a negative effect on the mortality rate, it is not reliable because of a high intercorrelation with the AFDC for teenagers.

4.2 Policy Implications and Suggestions

The estimated reduced form equations and the postneonatal mortality production functions which are race- and birthweight-specific provide useful information on the factors that affect the infant's survival prospects after one month of life. The overall conclusion that appears to emerge from the preceding chapter is that prenatal care use, neonatal intensive care availability, pediatrician availability and use, and socioeconomic variables which reflect command over economic resources are of importance as factors that affect infant's survival probability after one month of life. But public programs which intend to address the problem of infant deaths do not affect postneonatal mortality except in the case of MIC availability. Thus, the determinants of postneonatal mortality are not the same with those of neonatal mortality in many respects.

Surely, one reason for exploring the determinants of infant mortality is to provide a better basis for policy formation. However, implications for policy design that can be derived from the above-mentioned estimation results appear to be compromised by the very fact that postneonatal mortality consists of only a small portion of overall mortality. As such, the results per se may not be so useful for policy purposes. It makes little sense, for example, to argue that a certain instrument should be of particular interest to policy-makers because it works well in the postneonatal period. Especially, if the impact is limited to the postneonatal deaths and the implementation of a policy involves a large cost, it is an absurd recommendation. Therefore, policy suggestions should be something which accounts for the determinants of neonatal mortality as well. But this position is not without difficulty because the results of this study do not coincide with those of Grossman et al. (1988) in many respects. We cannot reject, for example, their suggestion regarding the abortion availability because this variable is not related with postneonatal mortality. Thus, in a nutshell, what we can say about the policy implication is very circumscribed. Nevertheless, with this limitation in mind, some comments are in order in what follows along with some suggestions for further research.

Grossman and his associates suggest policies that reduce the inequality in health resources such as neonatal intensive care hospitals across states based on the results that although their impacts on the neonatal mortality rate are substantial, they are characterized by a nonuniform and uneven distribution in terms of the coefficient of variation (Grossman et al. 1988). Their suggestion as to the neonatal intensive care hospital is still relevant in the postneonatal period in light of their effects on the postneonatal mortality rate.

The estimation results of this study also suggest that some more weight should be put on the MIC projects in the formation of policy to reduce the infant mortality rate. Some studies, for example, Mundinger (1985) ascribes recent slowdowns in the rate of decline in infant mortality to cutbacks in maternal and infant care projects. Although a bit exaggerated in light of the result of Grossman et al. (1988) which finds no impact of MIC projects on neonatal mortality, his view can be supported in part by the finding of this study. Also, that the impacts of Medicaid and WIC programs on postneonatal mortality are estimated to be negligible seems to mean that the rationale of policy emphasis on these programs should be reestablished by future research. Although there is a lot of evidence regarding the positive relationship between the survival probability and these programs, more studies should explore how robust are these findings, hopefully using more refined measures of public programs.

A finding of this study as to the strong negative effect of AFDC support for teenagers on the infant's survival probability also needs more study to corroborate whether it really results from disincentives induced by welfare. Of course, it is a highly controversial area which is beyond our intended scope. But, as indicated earlier, there is a reliable piece of evidence that out of wedlock birth affects infant mortality. Given that, the problem is whether welfare does influence fertility among youths. If this linkage turns out to be plausible, the finding of strong positive impacts of AFDC on mortality is more than a statistical artifact, and the result can be regarded as a piece of evidence which supports the view of the critics on the current welfare system.

In the preceding chapter, we have answered many of the questions raised in the introduction and raised some new questions. However, the findings of this study do not necessarily remain invariant to the estimation technique employed and thus may be misleading. Moreover, we simply overlooked some important issues on the nature of the survival probability production process. For example, substitution and complementarities which are particularly interesting from the viewpoint of policy formation was not examined because we employed a linear functional form for production function as a matter of convenience. Needless to say, therefore, a piece of research is not enough, and further studies on the subject are required to pin down the more precise relationship considered in this study.

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