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THE IMPACT OF SOCIOECONOMIC VARIABLES ON NEONATAL MORTALITY: THE IMPORTANCE OF DISTINGUISHING EARLY AND LATE NEONATAL PERIODS

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THE IMPACT OF SOCIO-ECONOMIC VARIABLES ON NEONATAL MORTALITY: THE IMPORTANCE OF DISTINGUISHING EARLY AND LATE NEONATAL PERIODS

By

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ABSTRACT

by

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The literature on the relationship between socio-economic variables and infant mortality is reviewed. It is argued that the inconsistencies found in previous research can be clarified by the distinction of four different sequential environments (or periods) in which different variables impinge upon infant mortality. Two micro-level models, one for early neonatal mortality and another for late and post-neonatal mortality are proposed. Vital statistics, census and other data are employed to test two macro-level structural equation models. Path analysis and tobit techniques are used for the statistical analysis. The findings show that, in fact, different variables affect early and late neonatal mortality; also that the same variables have different effects in those periods. The implications of the findings are discussed.

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INTRODUCTION

It is conventional to subdivide infant mortality (from birth to one year) into the neonatal period (from birth to 28 days) and the post-neonatal period (the remainder of the first year of life). Mortality rates for both periods tend to decline systematically with development (Preston, 1976). This decline is mostly due to a decrease in post-neonatal mortality. However, post-neonatal mortality in the U.S. since 1970 has remained relatively stable, while neonatal mortality has decreased.

The usual explanation for the earlier decrease in post-neonatal mortality is related to the role of the child's environment in this period. The main causes of death in this period are exogenous (parasitic diseases, respiratory and gastrointestinal infections, and accidents, for example), all of which are related to the social environment (Bouvier and Vander Tak, 1976; Pharoah and Morris, 1979). The main causes of death in the neonatal period are, instead, endogenous (immaturity, birth injuries, post natal asphyxia and congenital malformations, for example) (Bouvier and Vander Tak, 1976).

Consequently, one would expect that socioeconomic differences would have a greater impact on post-neonatal mortality than on neonatal mortality. Indeed, differentials in mortality among socioeconomic groups are greater in the post-neonatal period (MacMahon, et al., 1970; Antonovosky and Bernstein, 1977). However, while the smallest differentials among social classes occur soon after birth (Antonovsky and Bernstein, 1977; Morris, 1979; Hemminki, et al., 1980) neonatal mortality also seems to be affected by socioeconomic differences (Adamchack, 1979; Antonovsky and Bernstein, 1977; Brooks, 1980; Markides and McFarland, 1970; Stockwell, et al., 1978, Institute of Medicine, 1973).

The lack of a clear relationship of the impact of socioeconomic variables on neonatal and post-neonatal mortality is reflected in most studies on this topic. Many studies using an ecological framework have found an inverse relationship between income and infant mortality (Yankauer, 1950, 1958; Anderson, 1958; Willie and Rothney, 1962; NCHS, 1972) while others have failed to find a clear relationship (Willie, 1959; Stockwell, 1962; Donabedian, et al., 1965). The latter studies argue that the significant decline in post-neonatal mortality accounts for the decline in the relationship between SES and infant mortality. Recent studies have questioned these conclusions, finding once again an inverse relationship (Brooks,1975, 1980; Gortmaker, 1979a, Markides and Barnes, 1977).

There is also lack of agreement on the details of the relationships. Most researchers agree that there is a critical level, namely that of poverty, but an inverse linear relationship with SES has been questioned (Antonovsky and Bernstein, 1977; Adamchack, 1979). When introducing the variable education, the studies cited as well as those of Shah and Abbey (1971), Benjamin (1965) and MacMahon et al. (1970), found that education of the father and the mother have an independent effect on infant mortality, even on the exogenous causes of death (NCHS,1972). However, when looking at differences between neonatal and post-neonatal mortality, Adamchack and Stockwell (1978) found that education was positively related with the former and negatively related with the latter. Bross and Shapiro (1982) found that mother's education did not have a direct effect on infant mortality and Gortmaker (1979a) found that while post-neonatal mortality was directly influenced by income level of the family (poverty-no poverty), and by mother's and father's education, neonatal mortality was affected only by income level. In general, it can be said that the relationship is fairly clear in terms of post-neonatal mortality (Hunt, 1967; Brooks, 1980), but that there are inconsistent findings for neonatal mortality.

The present paper addresses these inconsistencies and attempts to clarify the relationships among intervening variables as has been done by Struening et al. (1973) and more recently by Brooks (1980). Two individual level models are introduced and it is argued that the distinction between neonatal and post-neonatal mortality is analytically misleading from a sociological viewpoint. In order to understand the relationship between socioeconomic variables and infant mortality, a sequence of four environments must be distinguished:

mother's previous socioeconomic environment, the intrauterine, the hospital and the home. In Part II, two macro level models for the white population living in Michigan cities with a population between 10,000 and 50,000 are tested. Different variables and different processes are shown to affect early and late neonatal mortality. Results are discussed and conclusions are drawn.

PART I. A FORMAL STATEMENT OF RELATIONSHIPS

The Sequence of Environments

In order to account for the differentials between social classes some "environmental" explanations have to be developed. An important basic distinction is that between the intrauterine environment and the child's post-delivery environment (Gortmaker, 1979). The former is created by the mother and therefore we must consider her environment. The child's post-delivery environment may be divided into two parts. In the U.S., as in most developed societies, 99.4% of all babies are born in a hospital (NCHS, 1975). Therefore, the first environment the child encounters is the hospital. The standard period that a newborn baby at no risk stays in a hospital, is three days. However, we know that the highest risk in the neonatal period is the first six days.[1] Hence, a period of six days seems to be a reasonable temporal limit for the newborn's first environment. Then, the surviving infant will encounter a second environment, namely the home. Table 1-1 shows the relationship between time period, environment, mortality period, and SES differentials in mortality.

Two Micro-Level Models

Given the several environments shown in Table 1-1 different variables are relevant in each mortality period and

TABLE 1-1 THE SEQUENCE OF INFANT ENVIRONMENTS

TIME PERIOD	Before Pregnancy	20 weeks to Birth	Birth (Day 0)	Days 1 to 6	Days 7 to 27	Months 1 to 12
ENVIRONMENT	Mother's	Intrauterine	Hospital		Home	
MORTALITY PERIOD		Fetal deaths	Early Neonatal deaths		Late Neonatal	Post-Neonatal
SES DIFFERENTIAL		1:2.13	1:1.36	1:1.42	1:3.11	1:3.19

SOURCE: Antonovsky and Bernstein (1977); MacMahon et al., (1970). The ratio is: mortality in the highest SES group = 1 and mortality in the lowest SES group.

and their effects will be different for each period. A micro level model, that is, a model that states the relationships at an individual level for early neonatal mortality is shown in Figure 1. The model shows an "ideal typical" mother whose pregnancy will probably end in the death of the newborn. Since a detailed representation of the interrelationships of variables would be confusing, different "blocks" have been distinguished. Age of mother, young or old, constitutes block I. It has direct effects on: a) income, education and work (block II); b) illegitimacy and family size (block III); c) history of pregnancy loss (block IV); d) congenital diseases (block V); and e) prenatal care, nutrition, smoking, etc. (block VI). The logic, based upon the literature, is detailed in the following paragraphs.

Young families tend to be poorer than older families (Lampman, 1971). This relationship is independent of family size as will be discussed later. Instead, it may well be an effect of the career to adulthood. The relationship between age and education is curvilinear, that is, positive up to the ages of 25-30 and then downward in slope (U.S.D.C.,1970). Also curvilinear is the relationship between work and age (for women). It is positive until age 25, then negative through ages 30-34, and then positive again up to ages 50-54. (U.S.D.L., 1980)

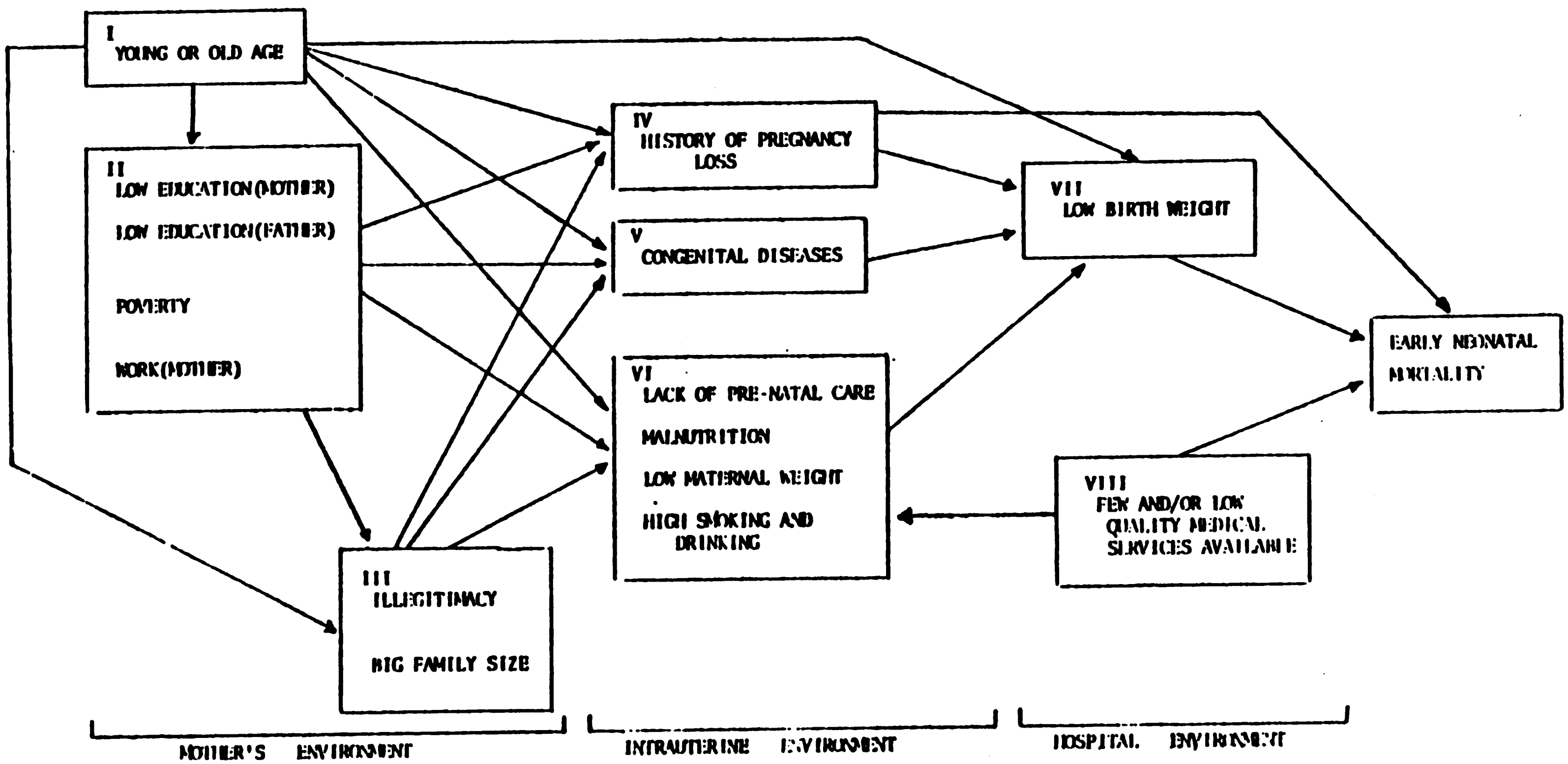
Young age of the mother is associated with illegitimacy (NCHS, 1975). In fact, "two out of every five births to teenagers are illegitimate and an even larger proportion are conceived out of wedlock" (O'Connel, 1980: 13). Furthermore, for the white population with which we are concerned, the fraction of births premaritally conceived has more than doubled since 1970 (O'Connel and Moore, 1980; Alan Guttmacher Institute, 1976; Sklar and Berkov, 1974).[2]

In terms of household size, the relationship with age in the U.S. is J-shaped, that is, older persons (above 55 years) show the highest percentage living in smaller houses, with the youngest persons (below 25 years) next highest (Kuznets, 1978). Since we are concerned with infant mortality, the most relevant effect is the direct association between young age and small family size. However, many indirect effects intervene. Lower status groups as well as lesser educated ones tend to marry earlier (even to have births before marriage). Therefore, the effect of family size is increased at younger ages for lower status groups (Bumpass, 1969).

Age of mother seems to have an independent effect on congenital diseases (Apgar, 1970) and on the history of pregnancy loss (Institute of Medicine, 1973; Andrews and Roberts, 1982). As Leridon puts it:

> "The risk does not seem to be constant at different periods of a woman's reproductive life...the aging of the ova, the exhaustion of the organism, the cumulative risk of various ailments...all these may aggravate the risk of miscarriage as a direct function of mother's age." (1976:319)

FIGURE 1-1. MICRO-MODEL FOR EARLY NEONATAL MORTALITY (0 to 6 days)



There seems to be no doubt then as to the effects of older age on the history of pregnancy loss, but those affected seem to be a very small genetically disadvantaged population (Institute of Medicine, 1973). There is some doubt, however, as to the finding that younger mothers also have a higher risk (NCHS, 1973). Resseguie (1977) argues that this effect is an artifact and that higher internal differences are found in each age group when controlled for education. Hence, other interrelationships probably should be taken into account.

Prenatal care seems to be related to age. As with most age relationships, it seems to be a J-shaped curve, in this case inverted. Very young and older women in France and England seem to have a lower level of prenatal care. (Blondel, et al., 1980; Butler and Bonham, 1963). For the U.S., the relationship found by Gortmaker (1979a,b)only associates young age with lower prenatal care. Statistics of NCHS (1978b) however, tend to show the same inverted J-relationship for the U.S. Younger women tend to have a lower weight during pregnancy than older women. (In relationship to birth weight, see O'Sullivan et al., 1975; Rush, et al., 1972; Bradfield, et al., 1975; Baizerman, 1977). Age is also related to smoking behavior, with younger women smoking the most (U.S. DHEW, 1973; Saugstad, 1981).

Income, mother's and father's education, and mother's work compose block II. They have direct effects on: a) illegitimacy and family size; b) congenital diseases; and c) prenatal care, nutrition, smoking, etc. Low income and less educated groups tend to have higher illegitimacy (NCHS,1975), a relationship which has been consistently shown. Although the relationship between income and fertility (family size, i.e., completed fertility) is an "elusive one" (Freedman and Thornton, 1982), the complexities appear only when tastes and norms are controlled and when income is differentiated beyond a mere poverty vs. nonpoverty dichotomy. The association between low socioeconomic status and high fertility, and the subsequent aggravating effect of family size on poverty, have been consistently documented (Orshansky, 1968; Rindfuss and Sweet, 1977). It is true that there is some circularity in the argument that "poverty breeds big families" but recent data does tend to show that contrary to common sense expectations, the birth of a new child stimulates income-producing activities and family assets (Cramer, 1980; Smith and Ward, 1980). Teenagers, too, tend to get a stable job after they have a child (Fustenberg, 1976).

The effect of education on family size also has a tradition of research that establishes a negative relationship. The actual relationship between mothers' education and family size is, apparently, more complicated. As Janowitz (1976) shows, the nature of the relationship is affected by whether or not the mother holds a job. The findings tend to show that education of the mother is relevant for family size at lower levels of education and the importance of the job is more significant at higher levels of education. Besides, it has been shown that the effects of mother's education vary systematically with her husband's education, when her education is low. It is for this reason that we include the father's education in the mother's environment. This variable has already been shown to be relevant for infant mortality (Gortmaker, 1979a).

A word of caution is in order at this point. The model does not include father's occupation. This indicator is normally (and reasonably) used as an indicator of SES. Most studies, particularly the British ones, have found a negative association with infant and fetal mortality. We would argue, however, that father's occupation per se is not relevant in the mother's environment. Its measurement has been accomplished through income. Even more, when looking at its effect on birth weight, Neligan et al., (1976) found that it disappeared, once the variables relating to the mother were included. Similar findings were reported by Gortmaker (1979a).

The incidence of congenital malformations is certainly linked to the environment through mutagens and teratogens (Leck, 1977; Persaud, 1977; Brent and Harris, 1976). There is a well-documented negative relationship between SES and congenital malformations found in studies in different countries (Leck, 1977; Falkner, 1977; Hemminki et al., 1981). For the U.S., using race as a proxy this relationship has been shown by Chase and Nelson (1973) and NCHS (1978a). For effects of poverty status (Puerto Ricans as opposed to other whites) and of education, see Chase and Nelson (1973). Work is particularly important factor in this respect, with a higher number of delivery abnormalities among those women who work (Gofin, 1979; Hemminki et al., 1980).

The effects of income and education on prenatal care are evident because of differential access and use of health services that characterize lower classes (Richardson, 1970; Hoschstim, 1970; Taffel, et al., 1978; NCHS, 1980b), and even because of desires and attitudes surrounding use of health services (Lefcowitz, 1973).

Lower income and less educated groups have a higher incidence of symptoms of malnutrition and of lower maternal weight at younger ages (Winnick, 1976; Habitch, et al., 1974). Moreover, lower income groups smoke more cigarettes and tend to consume more alcohol (U.S. DHEW, 1973; Khosla, 1972; Syme and Berkman, 1976; Morris, 1979; Preston, 1969).

Illegitimacy and family size constitute block III. These variables have relationships with: a) prenatal care, nutrition, maternal weight, etc.: and b) history of pregnancy loss and congenital diseases. Illegitimacy *per se* does not seem to have any effect, except where we consider it an indicator of an unwanted pregnancy. Then it would impinge upon prenatal care, nutrition, and weight of the mother.

The higher the parity, the lower the probability of getting medical assistance. This surprising finding has been shown with consistency (Yankauer et al.,1953; Collver et al., 1967; Butler and Bonham, 1963 for England; and Blondel, et.al., 1980 for France). There is substantial evidence (Leridon, 1976; James, 1970, 1976) that parity has an effect on congenital diseases and on spontaneous abortion and stillbirth (history of pregnancy loss). The higher the parity, the higher the risk of subsequent abortion, independently of mother's age (Leridon, 1976).

Birth weight (block VII) is the best single indicator of infant mortality (among others, recently, Gortmaker, 1979a; Brooks, 1980; Bross and Shapiro, 1982; Stewart, et.al., 1981). In the U.S., the difference between the poor and the wealthy is almost identical with that of birth weight (Rush,et.al., 1974; Brooks, 1980). Furthermore, the difference in prenatal mortality between developed and developing countries can be partially explained on the basis of low birth weight (Rush, et.al., 1974; Puffer and Serrano, 1975).

Until the sixties, immaturity was considered to be the main cause of low birth weight and prematurity was the main concern (Baird, 1964). Recently, however, small babies have been separated into two categories: the true premature baby who is the right size for fetal age but is born too soon, and the so-called "small-for-dates" infant who is born on time but has not grown properly in utero and is therefore small at delivery (Winnick, 1976).

The importance of low birth weight was shown by Brimblecombe, et.al., (1968) and its greater importance over prematurity as a predictor of mortality was argued in a study by Neligan et al. (1976) that had the suggestive title: "Born too soon or born too small"? The relationship between low birth weight and mortality for Great Britain is shown in Pharoah and Alberman (1981) and for the U.S. in Lee et al. (1976). In Gortmaker's (1979a) sample, 70% of the deaths occurred among low birth weight children.

Given this fact, one would expect that the decline in neonatal mortality indicated in the introduction would be due to a decrease in the proportion having low birth weight. However, this is not the case. Instead, the reason seems to be the lowering of relative risk in each birth weight group due to prenatal care, as Lee et al. (1980) suggest; as Gortmaker (1979a) has shown for New York City and Quick et al. (1981) for Portland; as Pharoah and Alberman (1981) demonstrate for Great Britain; and as Usher (1977) shows for Canada. After birth, the improvement seems to be particularly relevant in the first 24 hours (Wallace, 1978). This, then, shows the relevance of the inclusion of the variable expressing the availability of high quality medical services since special units are not available in every hospital.

With respect to the availability of physicians and hospital beds, however, the evidence is contradictory. While it is usually assumed that they are important factors, among other things, because of their potential effect on prenatal care , Miller and Stokes (1978) found that physicians and hospital beds were related positively (not negatively) with infant mortality. This was corroborated by Miller and Smith (1980). Brooks (1978) found that the number of physicians and hospital beds were unrelated to infant mortality. Similar findings are those of Grossman and Jacobwitz (1981) for neonatal mortality.[3]

The model shows that those variables having an independent effect on birth weight are: a) age; b) history of pregnancy loss and congenital disease; c) prenatal care, nutrition, etc.; d) illegitimacy and family size.

Some doubts have been raised as to the effects of age in that they are actually indirect ones in which prenatal care and maternal weight are the intervening variables (Gortmaker, 1979a,b; O'Sullivan,1975; Rush et al., 1972). Traditionally, however, it had been argued that age has an effect on birth weight (Meuken, 1972; 1980; NCHS, 1973). Recently, Bross and Shapiro (1982) and Biraben and Darnaud (1978) found that the relationship is important (an inverse J-curve found in France and Germany). The latter authors do control for parity but do not control for prenatal care and weight. None of these controls are made by Bross and Shapiro. Therefore, one tends to accept the explanation of weight and prenatal care. Nonetheless, an arrow is shown in the model.

History of pregnancy loss and congenital diseases (blocks IV and V) are closely related (Leridon,1971). Their impact on low birth weight has been shown by many researchers (Shapiro et al., 1968; Institute of Medicine, 1973; Spiers et al., 1976).[4] More important probably is the differential survival found for infants with Down's Syndrome (a form of mongolism) with low and high birth weight (statistically significant at .001 level) (Chen et al., 1969). They also have a direct impact on mortality due to the genetic disadvantage that they imply.

Prenatal care has also been considered of utmost importance in infant mortality rates. Its importance on the survivorship of small-for-date infants has been already stated. (Gortmaker, 1979a; Mednick et al., 1979).

Malnutrition of the mother is the main cause of abnormalities in the fetal environment that lead to symmetrical growth failure. Such is the condition that accounts for the vast majority of small-for-date infants in less developed countries (Winnick, 1976). This relationship has been shown in many studies (Antonov, 1967; Smith, 1947; UN, 1954; and recently by Habitch et al., 1974; and Baizerman, 1977; Klein et al., 1977).

Low maternal weight and stature are also related to low birth weight (O'Sullivan et al., 1965; Rush et al., 1972; Bradfield et al., 1975; Baizerman, 1977). As already mentioned, both prenatal care and low maternal weight and stature have been considered so important that they have been offered as the variables explaining higher risk among infants of young and older women.

Although it has been a debatable point, the evidence that smoking and alcohol have the effect of lowering birth weight seems to be definitive (Meyer, 1976, 1977; Goldstein, 1977; Lowe, 1977; Munro, 1977; Simpson, 1977; Fielding and Yankauer, 1978a,b; Ounsted and Scott, 1982; Fortney et al., 1982).

If illegitimacy is looked upon as an indicator of an unwanted pregnancy[5] and if it is related to prenatal care and maternal nutrition, one can understand the repeated evidence that legitimacy has an effect on birth weight (NCHS, 1972, 1973,1975; Chase and Nelson, 1973; Struening et al., 1973; Gee et al., 1979; Brooks, 1980).

In more general terms, legitimacy could be a parsimonious indicator of lifestyle patterns that impinge upon the birth weight of the infant. However, the recent trend of increasing illegitimacy among whites accompanied by diminishing stigma attached to it, could change the relevance of this relationship.

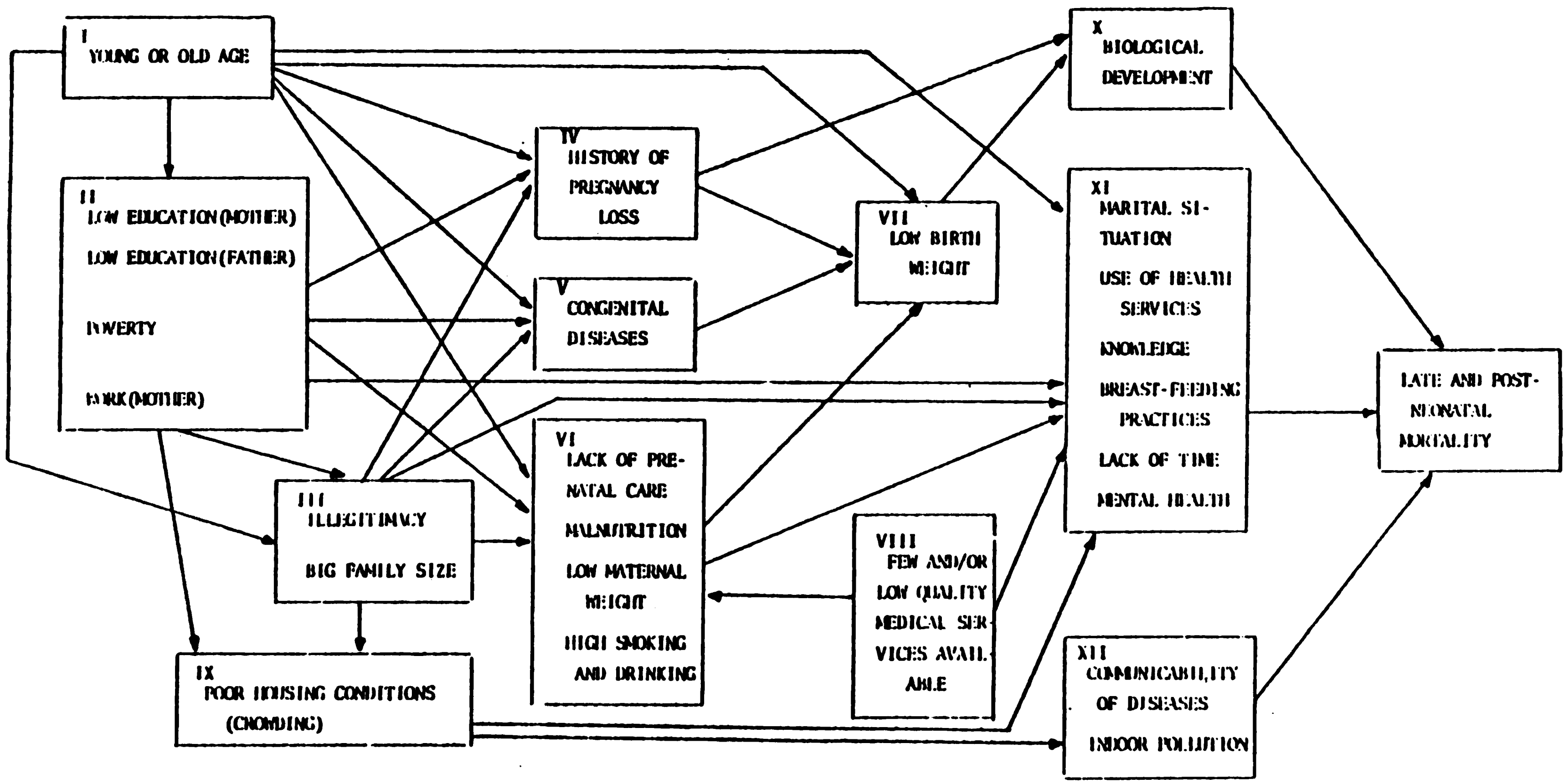
The effect of age and parity on neonatal mortality (Shapiro et al., 1968; Phillipe, 1978) could also be mediated by birth weight, as NCHS (1980) suggest and as Biraben and Darnaud (1978) show. Saugstad (1981) demonstrates that the higher the parity the higher the birth weight (meaningless for the U.S. but not for many other countries). This relationship that would decrease the risk for higher parities could certainly be outweighed by the increase in risk due to the relationship between parity and prenatal care.

Our analysis attempts to show that there is no direct effect of income and education on birth weight, and hence any model certainly will explain Gortmaker's (1979a) findings. It also explains the contradictory evidence in terms of the relationship between income as well as education and infant mortality.

The central issue is that the scholars cited in the introduction have never seen the relationship in a sequential way, and they have not considered the potential of birth weight as an intermediate variable. The few exceptions, such as the studies by Gortmaker (1979a), by Brooks (1980), and by Bross and Shapiro (1982), have shown its importance. However, these studies do not distinguish between early neonatal and late neonatal, as suggested in this thesis, and hence their results could be confusing. It is only through the mediation of the variables that are relevant in the intrauterine environment that the relationship between immaturity, prematurity, birth weight and social class is a valid one (Baird, 1964; Chase and Nelson, 1973). Given these relationships, it can be easily seen why we include birth weight, history of pregnancy loss, and medical services as the only causes of early neonatal mortality - that which takes place in the hospital environment.

When the home environment is included, the situation is much more complex. A micro-model for late and post-neonatal mortality is shown in Figure 1-2. Three new blecks are identified as being applicable to the home environment. These are: first, the variables related to the biological development of the newborn; second, the variables related to parent's behavior;

FIGURE I - 2. MICRO-MODEL FOR LATE AND POST NEONATAL MORTALITIES (7 days to 1 year)



and third, the variables that include ecological aspects, such as communicability of diseases, indoor pollution, etc.

Low birth weight is the only direct cause in the biological development of the child. Research tends to show that low birth weight children are handicapped in their physical and mental development (Babson, 1970; Cruise, 1973; Zachau-Christiansen and Ross, 1975; Neligan et al., 1976).

There are two main independent effects of age on mother's behavior. First, age is related to breast feeding practices. Until the 1970's the trend has been that the younger the cohort the lower the incidence of breast feeding (Hirschman and Butler, 1981). The second effect of age is the one related to career trajectory. Teenage mothers are much more likely than older mothers to drop out of school, to have frustrated future work plans, to have a lower income and to have a restricted "marriage market" when unwed (Trussell, 1976; Fustenberg, 1976; Presser, 1977; Card and Wise, 1978; Haggstrom and Morrison, 1979). Certainly these factors will have, in turn, an effect on the child's environment. Although their attitude toward child rearing may be a positive one (Fustenberg, 1976), there is no doubt that a teenager's child will be at risk (Sugar, 1976).

Income as well as mother's and father's education interact in terms of the care of the child. The level of use of medical services and the extent of knowledge about childhood diseases and nutrition, help to determine whether or not the child suffers involuntary neglect.

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Finally, one important aspect is the relationship between education and breast feeding. Hirschman and Butler (1981) found that the relationship is J-curved, with the most highly educated mothers practicing breast feeding the most. As in other cases, the husband's education intervenes. Further, mothers that work more often breast feed their first-born infants than those who do not work (Hirschman and Butler, 1981). Never married women are less likely than married women to breast feed their first infant (Hirschman and Butler, 1981). Whether or not the mother works clearly affects the pattern of infant care. For example, a teenage mother who does not work may become the principal caretaker of the baby. That mother must arrange care by others if she does work (Fustenberg, 1976).

Legitimacy seems to be related to lifestyle and the child's environment, generating a somewhat handicapped condition in later stages of life (Berkov and Sklar, 1975; Card, 1981). Gee et al., (1979) and Brooks (1980) show its impact on post-neonatal mortality.

As far as family size is concerned, Philippe (1978) shows that infant mortality risk grows in a parabolic way, according to the birth order, while Shapiro et al., (1968) found no clear relationship between birth order and neonatal death.

In order to rationalize the fact that risk increases with increased parity, but that neonatal mortality does not vary while the general infant mortality does, we can consider that family size impinges upon the child's home environment. It would seem clear that the larger the family size, the less time and care the mother will be able to give to the child. This explanation is consistent with Benjamin's (1965) study and with Scrimshaw's (1978) findings for less developed countries. It may well be that mortality is a "response" to high fertility (see Olsen, 1980), with an increasing risk to the child. Besides, as indicated earlier, the higher the parity, the smaller the proportion of mothers that breast feed their children (Hirschman and Butler, 1981).

Inadequate nutrition, smoking and drinking also have direct effects, decreasing the quality and quantity of breast feeding, (Winnick, 1976; Population Information Program, 1979) and increasing the negative immunological effects on the child (Hirschman and Butler, 1981).

Finally, a new block of variables related to housing conditions has been included in the first environment (block IX). A number of different forces can be distinguished here. The existence of poor sanitary facilities, lack of electricity, types of water sources, etc. have been shown to have an impact on infant mortality (Bouvier and Van Der Tak, 1976; Johnson and Nelson, 1982). In highly developed countries, however, the most relevant variable seems to be overcrowding in the living quarters. Crowding increases the possibility that a child will contract an infectious disease (Vaughan and McKay, 1975). The effects of crowding were found to be significant by Struening et al. (1973), by DHSS (1970), and by

Brennan and Lancashire (1978). Crowding also increases the probability of indoor pollution, which in turn has an effect on infant mortality (Van Der Leude, 1980). In this sense housing is related to family size.

Another effect of crowding is the problem of lack of intimacy (Gove et al., 1979). The effects of crowding in producing ill health and ineffectual care of the offspring were suggested by Galle and Gove (1978). Apparently parents tend to feel harassed by their children (Gove et al., 1979) and tend to develop poor relationships with their children under conditions of crowding. This situation is especially intense for the mother (Baldassare, 1981).

In addition, mental health is negatively associated with household density, a condition related to family size (Gove et al., 1979). That mental health is relevant to the frequency of prenatal mortality seems to be recognized by Bompiani et al., 1980 and by Meyerwitz and Lipkin, 1976.

Finally, income has an effect on housing. Low income families tend to have poor housing conditions (Downs, 1977; Foley, 1980).

In summary, then, late and post-neonatal mortality will be increased because:

a) Low birth weight will result in a handicapped biological development.

b) Age, family size, housing and legitimacy will impinge upon the child's affective development and stability.

c) Income and education of the parents will have an effect on their use of health services, on their knowledge and sensitivity in detecting a sick child, and in their nutrition patterns.

d) Mother's work and family size will have an effect on the time the mother can dedicate to the care of the infant and on breast feeding practices.

e) Housing and family size will have independent effects, increasing the communicability of infectious diseases and indoor pollution, and eventually adversely affecting the mental health of the mother.

f) Malnutrition, tobacco and alcohol consumption will have an effect on breast feeding.

PART II. AN EMPIRICAL TEST

The micro level models introduced in the previous section are complete in the sense that they show all the stated relationships among the variables (Namboodiri et al., 1975; Blalock, 1982). They were introduced because, as Blalock states, it is

> "recommended that one always construct models involving all variables believed to be of theoretical interest, even where some of these may have to be omitted from the empirical study. One may then trace out the implications of these models before deciding whether or not it is safe to neglect the intervening variables. Such a procedure provides justifications that are based on theoretical rather than pragmatic considerations, and it therefore provides the reader with an opportunity to evaluate the adequacy of the entire structural model before being presented with the data and interpretations" (1982:166)

In this section two macro level models, in their "reduced forms" (i.e. intermediate variables are omitted) are tested to show the validity of the subdistinctions in the neonatal period. The variables omitted are those corresponding to the intrauterine and to the home environments. Only history of pregnancy loss, from the intrauterine environment, is included to account for what seems to be a genetic disadvantage of a limited group (Institute of Medicine, 1973). Since the models are recursive, no bias or misleading conclusions are generated with this step, and there is only a loss in the "richness of the explanation" (Blalock, 1982:155).

Consequently, the causal variables included are: old age of women, poverty level, education of women, women's work, family size, illegitimacy, crowding in the home, history of pregnancy loss, low birth weight and availability and quality of medical services.[6]

The empirical test is done using the white population residing in the 60 cities of Michigan that had between 10,000 and 50,000 inhabitants in the 1970 Census of Population.[7] Therefore the test is done at the aggregate level and some paths are altered in the macro level models in order to avoid, insofar as possible, a specification error, which would increase the effects of a potential ecological fallacy (Irwin and Lichtman, 1978).

MODELS AND HYPOTHESES

The primary research question posed is the following: Are different variables and different processes involved in early neonatal mortality (0 to 6 days) as compared with late neonatal mortality (7 to 28 days), given that they take place in different environments?

To the extent that in both mortality periods the mother's prior socioeconomic as well as intrauterine environments are equally relevant, the same relationships are hypothesized, as shown in the brief summary of the discussion in Part I.

Young age of the mother is associated with illegitimacy (Sklar and Berkov, 1974; NCHS, 1975; Alan Guttmacher

Institute, 1976; O'Connel, 1980; O'Connel and Moore, 1980). Therefore it is hypothesized that:

H_1: Old age will have a negative direct effect on illegitimacy.

Given simple facts of reproductive life, and to the extent that younger people (below 25) and older people (above 55) tend to live in smaller households (Kuznets, 1978) it is hypothesized for potential mothers (aged 15 to 44) that:

H_2: Old age will have a positive direct effect on family size.

Old age of the mother seems to have an independent effect on history of pregnancy loss (Institute of Medicine, 1973; Leridon, 1976; Andrews, 1976; Andrews and Roberts, 1982). Hence:

H_3: Old age will have a positive direct effect on history of pregnancy loss.

Traditionally, it had been argued that old age has a negative effect on birth weight (NCHS, 1973; Biraben and Darnaud, 1978; Menken, 1972, 1980; Bross and Shapiro, 1982). Recent studies, however, have indicated that the effects of age are mediated through use of prenatal care, weight, and smoking behavior (Ruch et al., 1972; Bradfield et al., 1975; O'Sullivan, 1975; Gortmaker, 1979a). There is contradictory evidence as to the effects of age on prenatal care. In general, very young and older women have a lower level of prenatal care (Butler and Bonham, 1963; NCHS, 1978b; Blondel et al., 1980). Gortmaker (1979a,b), however, found that it makes a difference only for young age mothers. Young age is also associated with lower weight and with smoking and alcohol consumption which, in turn, have a negative effect on birth weight (Rush et al., 1972; Bradfield et al., 1975; O'Sullivan, 1975; Baizerman, 1977; Goldstein, 1977; Lowe, 1977; Meyer, 1976, 1977; Munro, 1977; Simpson, 1977; Usher, 1977; Fielding and Yankauer, 1978a,b; Omsted and Scott, 1982). Therefore, it is hypothesized that:

H_4: Old age will have a negative direct effect on low birth weight. (This hypothesis is offered with great reservations.)

Negative relationships between income as well as education and illegitimacy are well known and have been repeatedly and consistently shown (e.g. NCHS, 1975). Therefore it is hypothesized that:

H_5: Poverty level will have a positive direct effect on illegitimacy.

H_6: Women's education will have a negative direct effect on illegitimacy.

A tradition of research has shown the association between low socioeconomic status, in particular poverty, and high fertility (e.g. Orshansky, 1968; Rindfuss and Sweet, 1977). While the effects of mother's education and work are somewhat more complex (Janowitz, 1976) the relationship should hold true at the aggregate level. Hence:

H_7: Poverty level will have a positive direct effect on family size.

H_8: Women's education will have a negative direct effect on family size.

H_9: Women's work will have a negative direct effect on family size.

The relationship between SES and congenital malformations has been shown for different countries (Leck, 1977; Falkner, 1977; Hemminki et al. 1981; for the U. S. see differences between Puerto Ricans and other whites in Chase and Nelson, 1973). Women's work is particularly relevant too. (Gofin, 1979; Hemminki et al., 1980). To the extent that history of pregnancy loss and congenital diseases are highly interrelated (Leridon, 1971), it is hypothesized that:

H_{10}: Poverty level will have a positive direct effect on history of pregnancy loss.

H_{11}: Women's education will have a negative direct effect on history of pregnancy loss.

H_{12}: Women's work will have a positive direct effect on history of pregnancy loss.

Lower income and less educated groups have reduced access to health services and prenatal care (Richardson, 1970; Hoschstim, 1970; Gortmaker, 1977; Taffel et al., 1978; NCHS, 1980b), have more symptoms of malnutrition and lower maternal weight (Winnick, 1976; Habitch et al., 1974) and tend to smoke more and consume more alcohol (U.S. DHEW, 1973; Khosla 1972; Syme and Berkman, 1976; Morris, 1979; Preston, 1969). These in turn, have a negative effect on low birth weight. Consequently, in the reduced form of the model, it is hypothesized that:

H_{13}: Poverty level will have a positive direct effect on low birth weight.

H_{14}: Women's education will have a negative direct effect on low birth weight.

H_{15}: Women's work will have a positive direct effect on low birth weight.

At the aggregate level, medical services are not distributed equally and therefore it is hypothesized that:

H_{16}: Poverty level will have a negative direct effect on the number of medical doctors available.

H_{17}: Women's education will have a positive direct effect on the number of medical doctors available.

H_{18}: Poverty level will have a negative direct effect on the number of bassinets available.

H_{19}: Women's education will have a positive direct effect on the number of bassinets available.

H_{20}: Poverty level will have a negative direct effect on the quality of medical services available.

H_{21}: Women's education will have a positive direct effect on the quality of medical services available.

Illegitimacy _per se_ would not seem to have any theoretical relevance to low birth weight, unless it is considered an indicator of an unwanted pregnancy. If this is the case it would affect prenatal care, nutrition and weight of the mother. This seems to be an appropriate way of rationalizing the repeated evidence of its negative effects on birth weight

(NCHS, 1972, 1973, 1975; Chase and Nelson, 1973; Struening et al., 1973; Gee et al., 1979; Brooks, 1980). Hence, it is hypothesized that:

H_{22}: Illegitimacy will have a positive direct effect on low birth weight.

The higher the parity the lower the probability of getting medical assistance (Yankauer et al., 1953; Colver et al., 1967; Butler and Bonham, 1963; Blondel et al., 1980). Therefore, given the effects of prenatal care, it is expected that:

H_{23}: Family size will have a positive direct effect on low birth weight.

The higher the parity the higher the risk of spontaneous abortions and stillbirths (James, 1970; 1976; Leridon, 1976), independently of mother's age. Therefore, it is proposed that:

H_{24}: Family size will have a positive direct effect on history of pregnancy loss.

To the extent that history of pregnancy loss reduces the birth weight of the new born and thereby increases the risk of mortality (Shapiro et al., 1968; Institute of Medicine, 1973;Spiers et al., 1976), it is hypothesized that:

H_{25}: History of pregnancy loss will have a positive direct effect on low birth weight.

Birth weight is the best single indicator of infant mortality (among others, recently, Gortmaker, 1979a; Brooks, 1980; Bross and Shapiro, 1982; Stewart et al., 1981). Since availability of medical services and prenatal care would have an effect on the nutrition of the mother and consequently on the fetus, it is hypothesized that:

H_{26}: Availability of medical doctors will have a direct negative effect on low birth weight.

Up to this point, the relationships were common for the early and the late neonatal mortality models. Given their temporal locations and contexts in which they take place, however, different variables act (or the same impinge) differently upon these two types of mortality. Birth weight seems to be particularly relevant in determining the survivorship of the infant in the hospital environment and only somewhat relevant for the subsequent biological development of the infant at later stages (Babson, 1970; Cruise, 1973; Zachau-Christiansen and Ross, 1975; Neligan et al., 1976). Hence, it is proposed that:

H_{27}: Low birth weight will have a stronger positive direct effect on early neonatal mortality as compared with late neonatal mortality.

The availability of medical services, as measured by the number of medical doctors and the number of bassinets available, are relevant in both the hospital and the home environments, assuming the parental demand for these services. Since the evidence that these medical services have a positive effect on infant mortality is contradictory (Usher, 1977; Wallace, 1978; Miller and Stokes, 1978; Gortmaker, 1979a;

Lee et al., 1980; Miller and Smith, 1980; Brooks, 1978; Grossman and Jacobwitz, 1981; Pharoah and Alberman, 1981; Quick et al., 1981), it is cautiously hypothesized that:

H_{28}: Number of medical doctors available will have a direct negative effect on both early and late neonatal mortality.

H_{29}: Number of bassinets available will have a direct negative effect on both early and late neonatal mortality, with a possible stronger effect on the former.

Quality of medical services available, however, seems to be particularly relevant in the first 24 hours (Wallace, 1978). Therefore, it is hypothesized that:

H_{30}: Quality of medical services will have a stronger negative direct effect on early neonatal mortality than on late neonatal mortality.

When introducing the home environment, the direct effects of old age on early and late neonatal mortality are different. As discussed earlier, the effects on early neonatal mortality, if any, are positive. As to late neonatal mortality, in which the home environment is assumed to be important, it is generally agreed that children born to teenagers have high risks (Trussel, 1976; Fustenberg, 1976; Presser, 1977; Card and Wise, 1978; Haggstrom and Morrison, 1979; Menken, 1972, 1980; Sugar, 1976). Hence, it is expected that:

H_{31}: Old age will have a positive direct effect on early neonatal mortality and a negative direct effect on late neonatal mortality.

History of pregnancy loss and congenital diseases are particularly relevant for the survivability of the new born in the hospital environment; but there are also negative effects in later stages. Therefore, it is proposed that:

H_{32}: History of pregnancy loss will have a stronger direct positive effect on early neonatal mortality than on late neonatal mortality.

The hypothesized direct causes of early neonatal mortality then, are low birth weight, availability of medical services, and history of pregnancy loss. The resulting model is shown in Figure 2-1 by the solid and dashed lines.

Given that late, in contrast to early neonatal mortality, takes place mostly when the infant is already living at home and that income and education have a negative relationship with use of medical services and, most importantly with breast feeding practices that enhance the infant's health (Hirschman and Butler, 1981), it is hypothesized that:

H_{33}: Poverty level will have a positive direct effect on late neonatal mortality.

H_{34}: Women's education will have a negative direct effect on late neonatal mortality.

Since the fact that the mother works implies that she has less available time to care for an infant, it is hypothesized that:

H_{35}: Women's work will have a positive direct effect on late neonatal mortality.

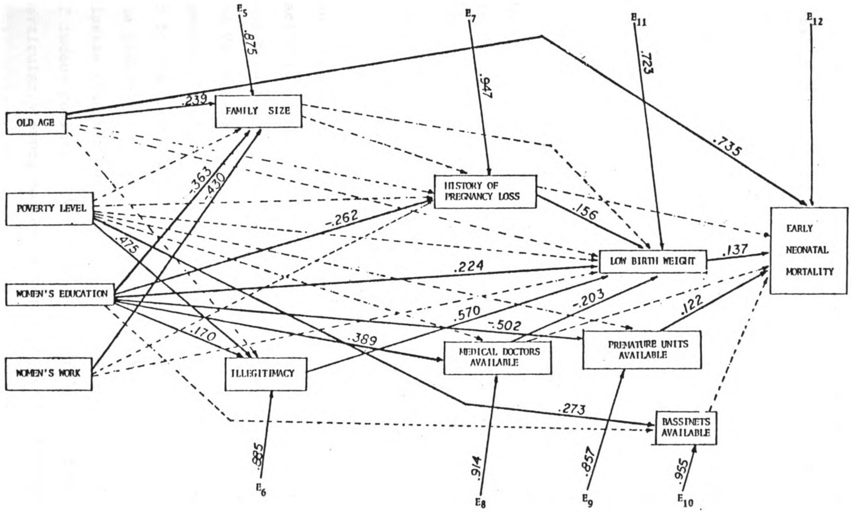


FIGURE 2.1. MACRO-MODEL FOR EARLY NEONATAL MORTALITY (0 to 6 days).

Philippe (1978) has shown that the risk of infant mortality grows with parity and Benjamin (1965) obtained findings that can be interpreted the same way. In addition, Hirschman and Butler (1981) show that the higher the parity the smaller the proportion of mothers that breast feed their children. Therefore, it is proposed that:

H_{36}: Family size will have a positive direct effect on late neonatal mortality.

Illegitimacy is related to lifestyle and the child's home environment which tends to generate somewhat handicapped conditions at later stages of life (Berkov and Sklar, 1975; Card, 1981) and increases post-neonatal mortality (Gee et al., 1979; Brooks, 1980). Hence, it is hypothesized that:

H_{37}: Illegitimacy will have a positive direct effect on late neonatal mortality.

Finally, there is a new set of variables in the home environment that is particularly relevant. Poor sanitary facilities, lack of electricity, type of water resources, etc. have been shown to have an impact on infant mortality (Bouvier and Van Der Tak, 1976; Johnson and Nelson, 1982).In highly developed countries, however, the most relevant variable seems to be overcrowding in the living quarters. Crowding increases the possibility that a child will contract an infectious disease (Vaughan and McKay, 1975), increases the probability of indoor pollution (Van der Leude, 1980), and promotes, particularly among mothers,a tendency to feel harassed by their children (Gove et al., 1979 ; Baldassare, 1981)with the negative consequences for mental health, which in turn has effects on infant mortality (Bompiani et al., 1981; Meyerwitz and Lipkin, 1976). Hence, it is proposed that:

H_{38}: Crowding will have a positive direct effect on late neonatal mortality.

In turn, since housing conditions are negatively related to socioeconomic status (Downs, 1977; Foley, 1980), it is hypothesized that:

H_{39}: Poverty will have a positive direct effect on crowding.

The relationship between crowding and family size, is not at all clear. It might be expected that the larger the family size, the greater the probability of crowding. However, this is not necessarily the case. Notice, for example, that a couple with one child, living in one room, will live in a more crowded environment than a couple with three children living in two rooms. Therefore, the sign in this relationship cannot be determined. Hence it is only hypothesized that:

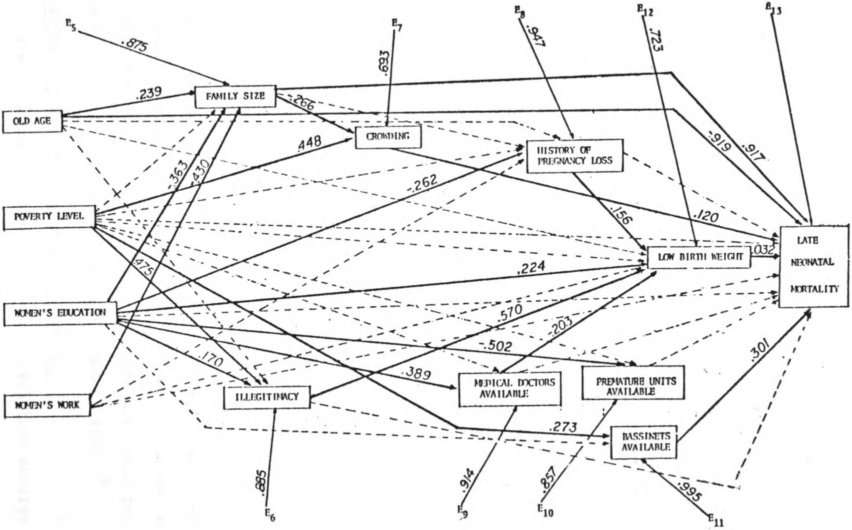
H_{40}: Family size will have a direct effect on crowding.

The resulting model is shown in Figure 2-2 by the solid and dashed lines.

METHODS

Two structural equation models are developed, the logic used being that of path analysis (Simon, 1954; Blalock, 1964; Duncan, 1966, 1975; Land, 1969). All the equations, with the exception of the last one, are estimated by ordinary least squares regression.

FIGURE 2-2. MACRO-MODEL FOR LATE NEONATAL MORTALITY.



Most of the dependent variables in the present study are rates and/or proportions. A rate, by definition, is a "limited dependent variable", namely, its range is limited by the permissible bottom and top values ($0<y<$ top value; where top values are normally 1,100,1000). This issue becomes critical, as in the present study, when the limited dependent variable presents many limit values (in this case zeroes) because of some underlying stochastic mechanism.[8] In the present study, early neonatal and particularly late neonatal rates, present many limit zero values. Clearly, the number of these limit values is known, and hence we are in presence of a censored variable. For this reason a tobit analysis (Tobin, 1958; Schmidt, 1980, 1981; Maddala, 1983) is performed. The tobit or censored regression model is a maximum likelihood estimation procedure that makes use of the properties of the truncated normal distribution and implies a model in which the $Y_i<0$ are observed as $Y_i=0$ and in which if $Z_i=X_i\beta+\varepsilon_i$ and the Z_i are i.i.d. as $N(0_1\sigma^2)$, we observe Z_i if $Z_i>0$

$$Y_i = 0 \text{ if } Z_i \leq 0$$

where β is a vector of unknown parameters; X_i is a vector of explanatory variables, εi are residuals that are independently and normally distributed with mean 0 and common variance, and observations on the explanatory variables are available for all values of Y, whether $Y_i=0$ or $Y_i>0$ (see Schmidt, 1980).[9]

OPERATIONALIZATION OF THE VARIABLES AND DATA

The empirical models at the city level then consist of those variables related to the mother's prior socioeconomic environment that later will presumably have direct effect in the home environment, such as age, poverty level, education, work, family size, illegitimacy and crowding; of the only measured variable from the intrauterine environment, history of pregnancy loss; those variables that are relevant in the hospital environment, such as low birth weight and availability and quality of medical services, and of the dependent variables, early and late neonatal mortality. These variables, as operationalized in this study follow.

Age of the women is operationalized at the city level as a "dummy" variable, so as to avoid the problem of non-linearity (Suits, 1957; Miller and Erickson, 1974). On the basis of whether or not the proportion of women aged 15 to 44 that are in the age group 15 to 25 is higher for a given city than the average percentage for all cities, that city is classified as young or old.[10]

Poverty level is measured by the proportion of all families in the city that are below poverty level according to the Census definition.

Education of mother is measured as the proportion of all women in the reproductive ages (15 to 44) that have completed college.

Women's work is measured as the proportion of women in the reproductive ages who work.

Family size is operationalized as the average number of children per women aged 15 to 44.

Crowding is measured as the proportion of all houses that have more than two persons per room. All data for the preceding variables for the white population were obtained from the 1970 Census of Population and Housing (fourth count).

Illegitimacy is measured as the proportion of all births in the city that are illegitimate.

History of pregnancy loss is measured as 1 minus the proportion of women having three or more previous deliveries without any stillbirth.

Low birth weight is measured as the proportion of all births in the city that weighed less than 2,500 grams. All data for the above three variables were obtained from birth certificates. This data were directly purchased from the Office of Vital Statistics, Michigan Department of Public Health.

Availability of medical services is measured as the number of bassinets per women aged 15 to 44 available in hospitals located in the city and its surrounding area. This area is defined as being within 30 minutes commuting time. Thus, all hospitals that could be reached in 30 minutes via automobile were included. These areas were delimited on the basis of data provided by the statewide hospital proximity analysis (MDSHT, 1966; 1980; MDPH, 1980). The number of bassinets used was obtained from the hospital directory for 1973 (AHA, 1973).

As another dimension of the medical availability variable, the number of medical doctors per number of women in reproductive ages was obtained for each city. This data is available from the Directory of Physicians (AMA, 1973).

The quality of medical services is measured by the number of hospitals in the defined zones that had a premature nursery in 1970 per women aged 15 to 44. Functionally, premature nurseries may be regarded as similar to a neonatal intensive care unit as of 1970.

Early and late neonatal mortality rates are measured by the number of deaths (at mother's place of residence) in days 0 to 6 and 7 to 28, divided by the number of births. These data were obtained from death certificates, obtained from tapes released by the Office of Vital Statistics, Michigan Department of Public Health.[10]

With the exception of Census data and the availability and quality of medical services, the data used in the analysis are averages for the three year period 1970-1972.[11] Since the number of cases is, in many instances, very small, this average provides some stability to the measures.

A general computer file for the 60 cities was created with these variables: transformations, calculations of proportions and OLS regressions were done with the Statistical Package for the Social Sciences (SPSS); and the tobit analysis was performed with a version of LIMDEP, a program from the Rand Corporation.

FINDINGS AND DISCUSSION

The results for the common equations for early and late neonatal mortality are shown in Table 2-1 (reproduced over the solid lines in Figures 2-1 and 2-2).

An initial regression was calculated with all the variables in the model. Those that were not significant according to their t ratios were dropped and the regression was recalculated with the remaining variables.[12] The table shows the number of the hypothesis and its sign at the upper left corner of the cell. The β coefficient, if the relationship is statistically significant, is shown at the lower right corner of the cell.[13]

As can be seen hypotheses 2,5,8,9,11,17,22,25,26 and 39 are confirmed. While an effect was predicted in hypotheses 1,3,7,10,12,13,15,16,19,20,23 and 24, no statistically significant effect was found. In the original regression with the totality of the variables most of the signs were in agreement with the expected directions, but given the small sample size of 60, they were not significant.

That old age would have a direct effect on low birth weight (Hypothesis 4) was posed with some doubts and indeed was not confirmed. Poverty level also does not show a direct effect

TABLE 2-1 HYPOTHESIZED COMMON RELATIONSHIP, THEIR SIGNS AND THEIR β COEFFICIENTS FOR EARLY AND LATE NEONATAL MODELS

Independent Variable	DEPENDENT VARIABLE: Illegitimacy	Family Size	History of pregnancy loss	Medical doctors available	Bassinets available	Premature units available	Low birth weight	Crowding
Old Age	H1(-) -	H2(+) .239	H3(+) -				H4(-or?)	
Poverty Level	H5(+) .475	H7(+) -	H10(+) -	H16(-) -	H18(-) .273	H20(-) -	H13(+) -	H34(+) .448
Women's Education	H6(-) .170	H8(-) .363	H11(-) -.262	H17(+) .389	H19 .	H21(+) -.502	H14(-) .224	
Women's Work		H9(-) -.430	H12(+) -				H15(+) -	
Illegitimacy							H22(+) .570	
Family Size			H24(+) -				H23(+) -	H40(?) -.266
History of Pregnancy Loss							H25(+) .156*	
Medical Doctors Available							H26(-) -.203	

*Significant at p = .1

on low birth weight, although a direct effect was expected. The relationship between family size and crowding, a hypothesis stated without any prediction as to sign, has a negative relationship.

More relevant are the findings in which the expected sign is reversed. The predicted negative relationship between women's education and illegitimacy shows, instead, a positive direction. While not expected, it could be a consequence of the trend among young white educated women to have illegitimate births, as was mentioned in Part I.

The direction of the empirical relationships between number of premature nurseries available and women's education and that of bassinets available and poverty level are both opposite that expected. Many ad hoc explanations could be developed to account for this fact. However, a reasonable explanation in light of the fact that some previous research has found a positive relationship between availability of medical services and infant mortality (Miller and Stokes, 1978) is that sign of the causal direction is the reverse of that as stated. Health planners certainly take into account those poverty areas that have high infant mortality in the allocation of their resources and this fact could explain the relationship found. In fact, the aforementioned study using time series data showed that counties that initially were relatively poor, with higher mortality and with fewer facilities were the ones that experienced the greatest change in terms of health resources

(Miller and Stokes, 1978:271). No cross sectional study like the present one could possibly explore this functional relationship. Clearly an alternative and simpler explanation is that the relevant variable for low birth weight and infant mortality is not availability of medical services, but rather access to them. The former interpretation, however, is reinforced by the fact that medical doctors available shows the expected relationship. Despite some health planners, the distribution of medical doctors in the U.S. is still a choice of individuals and not of planners.

The specific results of the tobit analysis for early neonatal mortality are shown in Table 2-2. A likelihood ratio procedure[14] was performed to test the hypothesis of no effect and to test that the coefficients dropped were in fact not significantly different from zero. Both instances showed the expected results, (chi squares of 65.418 and 12.04, with 5 and 9 degrees of freedom respectively.)

The coefficients shown in Table 2-2 are the standardized (β) and non-standardized ones (b). It should be kept in mind that the size of the coefficients is not the size of the effect of the independent variable on early neonatal mortality. The actual effect is given by the coefficient multiplied by the probability that the dependent variable has a non-zero value (Schmidt, 1981). For early neonatal mortality this probability is .95. An equivalent to R^2 is also calculated and shown.[15]

TABLE 2-2 TOBIT REGRESSION COEFFICIENTS FOR EARLY NEONATAL MORTALITY

Independent variable	Unstandardized coef. (b)*	Standardized coef. (β)*	Eq. to R^2**
Old Age	17.980	.735	
Premature Nurseries Available	2.979	.122	.534
Low birth weight	3.359	.137	

* P (y>o) = .95. See text.
**See note 15.

While it was hypothesized that history of pregnancy loss would have a direct effect on early neonatal mortality (H32), the empirical relationship is not statistically significant. Old age, instead has a significant direct effect on early neonatal mortality (H31). Whether this is due to the fact that old age accounts for those aspects of reproductive life that were not measured in history of pregnancy loss[16] or because of its effects on the actual use of medical services, the present findings provide support to those that argue that age has direct effects on neonatal mortality.

Low birth weight has the expected positive effect on early neonatal mortality (H27). An unexpected finding is the direction of the relationship between premature nurseries available and the dependent variable. These findings, however, are consistent with the ones reported on Table 2-1 and other research, as already mentioned. The results of the tobit analysis for late neonatal mortality are reported in Table 2-3, and the same remarks stated for early neonatal mortality are applicable. The chi-square values in this case were 20.76 and 5.494 with 3 and 7 degrees of freedom, respectively. For late neonatal mortality the proportion of non-zero values is .633.

TABLE 2-3 TOBIT REGRESSION COEFFICIENTS FOR LATE NEONATAL MORTALITY

Independent Variable	Unstandardized Coef.(b)*	Standardized Coef.(β)*	Eq. to R^2**
Old Age	-4.623***	-.919***	
Family Size	4.613	.917	
Crowding	.006	.120	.274
Bassinets Available	.151	.301	
Low birth weight	.162	.032	

* $P(y>0) = .633$. See text for Table 2-2.
** See note 15.
*** Significant at p = .1

As can be seen from the comparison of Tables 2-2 and 2-3, different variables impinge differently upon early and late neonatal mortality. A test to see if the coefficients and specifications of both models were equal was performed and it was rejected.[17]

The table shows that the only variables having significant direct effects on late neonatal mortality are old age, family size, crowding, bassinets available, and low birth weight. All of these variables were expected to have direct effects. The direction of the relationship between bassinets available and the dependent variable is, once again, the opposite of that expected. Many other direct effects had been hypothesized but their effects do not appear to be statistically significant. Total, direct and indirect effects[18] on early and late neonatal mortality are shown in Table 2-4. Low birth weight has indeed the predicted (H27) stronger positive direct effect on early neonatal as compared to late neonatal mortality. Availability of medical doctors does not show an empirical direct effect as expected (H28), but the sign of its indirect effect is negative as hypothesized.

Number of bassinets available not only does not show the expected differential impact (H29), but its sign is positive when it has a direct effect on late neonatal mortality, as already mentioned. Again, many explanations could be proposed for the unexpected differential impact. Bassinets available could be simply an indicator of availability of medical services or they could indicate the availability of technological support facilities which keep marginal infants alive during the early neonatal period that then are more prone to die during the late neonatal period. The same is the case for premature nurseries available, a proxy for quality of medical services. While the differential impact is the expected one, with a significant impact on early neonatal mortality and no impact on late, the sign is the opposite of the one hypothesized. It seems evident that higher quality services cannot lead to a higher early neonatal mortality and hence it is reasonable to speculate that the relationship may be a non recursive one. In spite of the unexpected sign, however, the

TABLE 2-4. DIRECT, INDIRECT AND TOTAL EFFECTS ON EARLY AND LATE NEONATAL MORTALITY

Variable	EARLY NEONATAL			LATE NEONATAL		
	Direct Effect	Indirect Effect	Total	Direct Effect	Indirect Effect	Total
Low birth wt.	.137*	-	.137	.032**	-	.032
Medical doctors available	-	-.028	-.028	-	-.006	-.006
Bassinets available	-	-	-	.301**	-	.301
Premature nurseries available	.122*	-	.122	-	-	-
Old Age	.735*	-	.735	-.919**	.212	-.707
History of pregnancy loss	-	.021	.021	-	.005	.005
Poverty level	-	.037	.037	-	.144	.144
Women's Education	-	-.047	-.047	-	-.314	-.314
Women's work	-	-	-	-	-.380	-.380
Family size	-	-	-	.917**	-.032	.885
Illegitimacy	-	.078	.078	-	.018	.018
Crowding	-	-	-	-	.120	.120

* P (y>o) = .95. See text for Table 2-2.
**P (y>o) = .633. See text for Table 2-2.

present findings do not provide support to that research that argues that availability of medical services is not related to infant mortality, which presumably might decline with a time lag.

Old age has the expected positive and negative effects on early and late neonatal mortality, respectively (H31). This variable is a good example of the misleading conclusions that would emerge from the use of total neonatal mortality. Notice that if both periods - early and late - were added, the different signs of age would cancel out, leaving the researcher with "no effect". Instead when the models are correctly specified, old age is one of the most important variables.

History of pregnancy loss has no direct effect on either type of mortality but its indirect effects show the expected differential impact on them (H32).

In contrast to previous research (e.g. Gortmaker, 1979) and the predicted relationship (H33), poverty level has no direct effect on late neonatal mortality. It only has an indirect positive effect, in agreement with what was expected. The same is the case of women's education that has no direct effect on late neonatal mortality. However, its indirect effects, as expected are stronger for late neonatal mortality, with the hypothesized negative sign (H34). Women's work does not have a direct effect either, and its indirect negative effects on late neonatal mortality are contrary to the expected ones (H35), being mediated by its impact on family size. This finding contradicts previous findings at the aggregate level that did not control for the effects of family size and did not use a path analytic approach (Adamchak, 1979).

Family size presents the hypothesized positive direct effect on late neonatal mortality (H36), although its indirect effects are negative due to its relationship with crowding. Illegitimacy does not have a significant direct effect, as predicted (H37). Nonetheless, its indirect effects are in the expected direction. It is worth remarking that all the indirect effects of illegitimacy are mediated through low birth weight. Given the stronger effect of the latter on early neonatal mortality, illegitimacy has a stronger indirect effect in this period. Besides, illegitimacy is the exclusive mediator of the effects of poverty on early neonatal mortality and one of the two mediators in the late neonatal period. These findings, then, support and refine previous evidence on the importance of illegitimacy (Brooks, 1980).

Finally, crowding, as predicted (H38), has a significant positive direct effect on late neonatal mortality. This finding, together with that on the impact of women's work are particularly relevant due to the fact that these variables have not been systematically included in multivariate models.[19] Further, the present findings, in general, by distinguishing direct and indirect effects, throw important light on the processes involved as well as under what conditions the relationships may change.

SUMMARY AND CONCLUSIONS

In Part I two individual level models that formalize the relationships between socioeconomic variables, intervening variables, and early and late neonatal mortality were introduced. It was argued that the standard treatment of the neonatal period as a total unit is analytically misleading.

In Part II, forty hypotheses were derived, summarizing Part I, and two macro level models were tested to answer the research question of whether there are different variables and different processes taking place in early versus late neonatal mortality.

Old age of the mother, quality of medical services, and low birth weight were shown to be the only variables that have a direct effect on early neonatal mortality. Old age, family size, crowding, bassinets available and low birth weight were the ones that had a direct effect on late neonatal mortality. Consequently, the findings corroborate the assertion that the treatment of neonatal mortality as a total unit can be conceptually misleading.[20] Further, given that the effects of the coefficients could cancel out depending on the relative frequencies of early and late neonatal, with respect to total neonatal mortality, it can be the cause of some of the contradictory evidence mentioned in the Introduction.

In this respect, the findings refine the interrelationships between socioeconomic variables and intervening ones, including apparently biological variables. In doing so, this shows that the mechanisms that operate in early and late neonatal mortality are quite different. Moreover, it demonstrates that socioeconomic differences, while having indirect effects, are also relevant in the first six days of life of the newborn. Although some multicollinearity exists among the variables, the comparison is not vitiated. Only the stability of the coefficients across samples can be questioned, given their large standard errors.

While the generalizability of the findings is somewhat questionable given the sample used, no ecological fallacy seems to be evident given the overall agreement of the results for the macromodels and the expected relationships deduced from the micro models. It is in this sense that the findings throw some doubt with respect to the actual sign of the causal direction of the relationship between neonatal mortality and availability and quality of medical services at a given point in time. At the same time, the findings enhance the importance of some variables that are normally omitted in the literature, such as mother's work and crowding in the home.

The problems of generalizability and stability of the coefficients preclude any firm policy recommendation. However, the findings on the importance of illegitimacy as a mediating variable for old age and crowding are suggestive of the actual relevance of social programs that provide family planning services and counseling, that provide a targeting of older mothers, and that seek the improvement of housing conditions, in order to reduce neonatal mortality.

The processes by which these variables impinge upon infant mortality, however, are not at all clear. Why is age curvilinearly related to the use of prenatal care? What, if any, are the attitudinal components of this relationship? What aspects of women's work are the ones that reduce late neonatal mortality? Is this effect also present at the individual level? These are a few among many of the questions for which current research does not provide a satisfactory answer. The latter aspects suggest the need for further comprehensive research using models that examine the effects of variables such as those proposed here, and eventually improved with the addition of new variables, better measurements and micro-level data. In particular, more sophisticated multivariate longitudinal research is needed, not only for pure methodological reasons, but also to investigate the possibility of non-recursive relationships and to evaluate the differential effects of the relevant variables under changing conditions.

NOTES

[1]The 6th day may seem arbitrary and clearly an actual empirical breakdown would have to be determined on the basis of some form of discriminant or cluster analysis with data not available for the present study. Nonetheless the 6th day is a reasonable critical point, given that it is the end of the period of perinatal mortality, established on biological grounds, and it has been shown to be critical in terms of socioeconomic differences (Antonovsky and Bernstein, 1977). The period has been named "sema-natal" mortality (Gandotra et al., 1980). The name used here is early neonatal mortality.

[2]There is a psychosocial variable that has not been included in the model. Teenagers have a higher number of unwanted pregnancies (Fustenberg, 1976). We have not considered this as an independent effect of age for two reasons:
a) The relationship between age and illegitimacy as stated in the text, and b) the noted decline of unwanted pregnancies in the U.S. is certainly related to SES (Westoff, 1981). Thus, we consider that the possible effects of an unwanted pregnancy and the lack of care during the pregnancy period will be accounted for, in our model, by the effects of age on prenatal care and by the effects of education, income and marital status (legitimacy). The importance of this variable, however, cannot be underestimated in light of the finding that an increase in the legal abortion rate is the single most important factor in the reduction of white neonatal mortality rates and that it has been shown that there was a lagged decline of neonatal mortality following the rapid increase in the use of the pill and IUD (Grossman and Jacobwitz, 1981).

[3]It should be emphasized that "availability" is not the same as "access".

[4]One could argue that the relevant variable in both cases, as well as in others, is maternal morbidity (Zachau-Christiansen and Ross, 1975).

[5]See Note 2.

[6]Father's education, included in the micro level models of Part I, was dropped from the model due to its almost perfect multicollinearity (at the aggregate level) with mother's

education. There is some multicollinearity among the remaining variables. Multicollinearity, however, is a problem that affects the matrix of independent variables. Since this variance-covariance matrix is the same for the two models compared, the comparison is absolutely valid. The coefficients, however, will still be unstable for the purposes of generalization across samples, given their large standard errors. Hence, no claim is made in the present study that the findings as to the absolute effects of the variables are "definitive".

[7]These selections were done as a way of controlling for the possible impact of size of place (Yerushalamy and Silverman, 1945) and rural and urban differentials (Dow,1932; Shapiro et al., 1968; Kitagawa and Hauser, 1973; Gortmaker, 1979a), to control for the impact of different legal regulations on abortion, family planning and medicaid which have ben shown to be relevant (Grossman and Jacobwitz,1981), and to minimize the heterogeneity of the population and consequently, the ecological fallacy. Given, however, that the aggregation is not done on the basis of size of the city and the small range of variation of this variable, the variable is not included in the model. Cities smaller than 10000 were not included because the necessary Census data is not available at that level; bigger cities were too few so as to compensate for the risks of ecological fallacies given the heterogenecy composition with respect to medical resources and other variables.

[8]Notice that if the problem were exclusively that infant mortality assumes only positive values one could postulate a log-normal distribution rather than a normal one. Instead, in the present study, there is also an underlying stochastic process going on. Assume that two cities have identical probabilities of death (in the "true" world) and of births, e.g. an actual late neonatal mortality rate of 4/1000 and a birth rate of 24/1000, but that one city, called A, has 10,000 inhabitants and consequently only 240 births and the other, called B, has 50,000 inhabitants and hence 1,200 births. In the simple world of this example, city A would show a mortality rate of 0, because there were not enough births for one death to occur. City B will show a rate of at least 4. Clearly we "know" that their probabilities were the same, and we are actually interested in that true probability, but, as in the more standard economic cases in which tobit analysis is used, there is a "threshold" to be attained (for the economic cases see Tobin, 1958; Maddala, 1983).

[9]If F_i and f_i are the distribution and density functions of the standard normal, evaluated at $\beta'X_i/\sigma$, which are vectors of unknown parameters (β) and known constants (X_i), by the symmetric distribution of the error(s) (normally distributed), we know that when $Y_i=0$, Prob $(Y_i=0)$ = Prob $(\varepsilon_i<\beta'X_i)$ = $(1-F_i)$ and that when $Y_i>0$, Prob $(Y_i>0)$, $f(Y_i/Y_i>0) = F_i \dfrac{f(Y_i-\beta'X_i,\sigma^2.}{F_i}$

From there, the likelihood function can be derived. For a more detailed discussion see Schmidt (1980); Maddala (1983:151ff).

[10]Notice that the measure is based on all women aged 15 to 44 and not exclusively on actual mothers.

[11]Birth data for 1969 were not available.

[12]An F test for the composite hypothesis of $H_o:\beta_1=\beta_2=\beta_3 \ldots\beta = 0$ was also performed and rejected.

[13]The significance level used throughout the present work is of p=.001. Otherwise it is specifically mentioned in footnotes.

[14]A likelihood ratio test is the equivalent of an F test in OLS. To test a composite hypothesis that $H_o:\theta E H_o$ against $\theta E H - H_o$, the test statistic λ is used; where $\lambda = \frac{L(X1\theta_o)}{L(X1\hat{\theta}}$, and where $\hat{\theta}_o$ and $\hat{\theta}$ represent the MLE of the unspecified parameters in H_o and H_1 respectively. Under appropriate conditions the random variable $-2 \log \lambda$ will possess an asymptotic χ^2 distribution with degrees of freedom equal to thedifference between the number of independent parameters in H and H_o (the number of restrictions) (See Hoel, Port and Stone, 1971:80ff).

[15]The tobit model, being a MLE, does not provide an R^2. For the sake of tradition, however, an R^2 is derived here. If Z_1 and Z_2 are independently distributed chi-square variables, with r_1 and r_2 degrees of freedom, respectively, the variable $F = \frac{Z_1/r_1}{Z_2/r_2}$ has an F distribution with r_1 and r_2 d.f. (See for example, Gujaranti, 1978:399). In a standard F test of H_o: $\beta_1=\beta_2=\ldots\beta_k=0$, the quotient, in general, takes the form of $\frac{RSS/(K-1)}{ESS/(N-K)}$, where RSS is the variation due to regression, ESS is the variation due to residual and K is the number of independent parameters estimated. But $ESS/(N-K) = \hat{\sigma}^2$ and, for large sample size, $\hat{\sigma}^2 \longrightarrow \sigma^2$, becoming a constant (Schmidt, 1976:9). Since RSS/σ has a chi-square distribution with K-1 d.f. (Schmidt, 1976: 28-9) and $\sigma^2/\sigma^2 = 1$, $F = Z_1/(K-1)$. Since, it is also the case that $F = \frac{R^2/(K-1)}{(1-R^2)/(N-K)}$ (see derivation in Gujarati, 1978:131),

it follows that $Z_1/(K-1) = \frac{R^2/(K-1)}{(1-R^2)/(N-K)}$

and $\frac{R^2}{1-R^2} = \frac{Z_1/(K-1) \quad (K-1)}{(N-K)} = \frac{Z_1}{(N-K)} = A$

Therefore $R^2 = \frac{A}{1+A}$, where $A = \frac{Z_1}{(N-K)}$ and Z_1 is a variable distributed as chi-square with K-1 d.f.

Clearly the actual result, given the asymptotic theory employed is going to be only an approximation.

[16]Given the measurement of pregnancy loss, the genetic disadvantage that it implies is only captured if there has been a previous pregnancy. Besides since only stillbirths enter in the measurement, miscarriages are not taken into account (or at least undercounted).

[17]Notice that no further test was done because total neonatal mortality is equal to the sum of early plus late neonatal mortality. Hence (in matrix notation) =

$Y_N = (\beta_E+\beta_L)\ X_i + \varepsilon$, where N is neonatal, E is early neonatal and L is late neonatal. The tests of $H_1: \beta_E \neq 0 \leftrightarrow \beta_L \neq \beta_N$ and $H_1: \beta_L \neq 0 \leftrightarrow \beta_E \neq \beta_N$. These tests have already been reported in the text. The only remaining test is $H_o: \beta_E = \beta_L$; which was the one performed. The value of the resulting test statistic was 268.638, with 8 degrees of freedom.

[18]It is worthwhile to remind the reader that direct and indirect effects, and their sum total effect, are certainly different from the total association (correlation) between the variables (Alwin and Hauser, 1975).

[19]Adamchak (1979) is one exception. Adamchak 's study, however, does not examine the different processes by which these variables impinge upon infant mortality.

[20]Notice that even for those interested in neonatal mortality, a more efficient estimation could be attained by the separate estimation of early and late. To the extent that each model will have an error term and they could be correlated, the single model for total neonatal will imply a less efficient estimation (in the statistical sense).

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JOURNAL ABBREVIATIONS

ADC	Archives on Disease in Childhood
AHG	Annual Human Genetics
AJE	American Journal of Epidemiology
AJOC	American Journal of Obstetrics and Gynecology
AJPH	American Journal of Public Health
AJS	American Journal of Sociology
AOGS	Acta Obstetrica et Gynecologia Scandinava
ARS	Annual Review of Sociology
ART	American Review of Tuberculosis
ASR	American Sociological Review
BJPSM	British Journal of Preventive Social Medicine
CJPH	Canadian Journal of Public Health
CP	Clinical Perinathology
D	Demography
E	Econometrica
FPP	Family Planning Perspectives
G	Genus
HSR	Health Services Research
I	Inquiry
IJE	International Journal of Epidemiology
JASA	Journal of the American Statistical Association

JBS	Journal of Biosocial Science
JECH	Journal of Epidemiology and Community Health
JHSB	Journal of Health and Social Behavior
JP	Journal of Pediatrics
JPSM	Journal of Preventive Social Medicine
JYA	Journal of Youth and Adolescence
L	The Lancet
MC	Medical Care
MMFQ	Milbank Memorial Fund Quarterly
P	Population
PB	Population Bulletin
PDR	Population and Development Review
PED	Pediatrics
PHR	Public Health Reports
PR	Population Reports
PS	Population Studies
RS	Rural Sociology
SB	Social Biology
SF	Social Forces
SM	Sociological Methodology
SMR	Sociological Methods and Research
SSB	Social Security Bulletin
SSM	Social Science and Medicine
WR	Welfare in Review