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PRENATAL MEDICAL CARE AND INFANT MORTALITY*

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INTRODUCTION

In 1969, forty three percent of expectant black mothers and seventy two percent of expectant white mothers began prenatal medical care during their first trimester of pregnancy. By 1977, fifty nine percent of black mothers and seventy seven percent of white mothers had begun prenatal care during their first trimester. Only three percent of expectant black mothers and one percent of expectant white mothers currently receive no medical attention before the onset of labor (U.S. National Center for Health Statistics 1978, Table A; 1980a, Table 20).

The main purpose of this paper is to inquire: How can we determine whether prenatal medical care has favorably influenced the outcome of pregnancy?

The role of prenatal care has been the subject of serious dispute in the obstetric and public health literature for nearly four decades. This dispute has been fomented in great part by the non-experimental nature of the evidence. Virtually all of the studies of prenatal care analyze cross-section data on the uncontrolled experience of thousands of women and their pregnancies. The subjects under study are therefore self-selected. There are no randomized treatments. Possible confounding variables cannot be eliminated. The data do not reveal how the subjects actually made use of the medical services. This paper investigates in detail what inferences can and cannot be legitimately drawn from this type of evidence.

Four main conceptual issues, I argue, underly the controversy about prenatal care and pregnancy outcome. First, the relationship between the timing of prenatal visits and the duration of pregnancy has been poorly

characterized. Mothers with little or no prenatal care, it has been repeatedly observed, have a higher proportion of pre-term babies. This fact suggests that prenatal care prevents premature labor. But early termination of pregnancy from any cause necessarily interrupts the course of prenatal care. It is unclear how these two confounding explanations can be dissected from the data.

Second, the phenomena of spontaneous and induced fetal loss exert a powerful selective effect on maternal and fetal characteristics. As a result of this selective effect, those mothers who initiate care late in pregnancy will have infants with characteristics quite different from those who initiate care earlier during gestation. Because the underlying health characteristics subject to selection may be subtle and difficult to measure, it is unclear how the mere accumulation of more explanatory data on weight gain, rubella history, fetal presentation, etc. can obviate the potential errors of inference produced by this selection effect.

Third, the frequently observed correlation between the quantity of prenatal care and birth weight lacks a convincing biological or behavioral explanation. Prenatal surveillance, to be sure, might indirectly improve birth weight by preventing early termination of pregnancy. But a mechanism for a direct effect of prenatal care on the rate of intra-uterine growth (that is, on birth weight for a given gestational age) is more elusive. That prenatal medical advice in fact alters maternal nutrition, smoking, or alcohol intake has not been verified. It is unclear how the retrospective analysis of large cross-section data bases is likely to resolve this difficulty.

Fourth, past analyses of prenatal care have not squarely confronted a critical fact about the recent decline in U.S. neonatal and infant mortality. That is, the decline in mortality primarily reflects a striking improvement in the survival rates of low birth weight infants. By contrast, there has been comparatively little change in the proportion of low birth weight infants or the fraction of pre-term deliveries. If the recent growth in prenatal care had a significant impact on infant survival, then we should expect to observe a relation between prenatal care and birth weight-specific mortality in cross-section data. Yet most studies do not observe such a relation. If prenatal care also prevents early termination of pregnancy and enhances intrauterine growth rates, it is unclear why concomitant changes in the proportion of premature infants were not observed.

I delineate these issues with data on over 140,000 pregnancies of at least 20 weeks' duration in Massachusetts during 1975-76. Specific attention is directed at the subpopulation of approximately 6,800 black women's pregnancies. Although the experience of Massachusetts may not be representative of the United States, the characteristics of the data base are typical of previous cross-section studies of prenatal care and pregnancy outcome.

In this investigation, I define prenatal care as medical attention received from the time of conception up to, but not including, labor and delivery. The analysis of perinatal medical care, received during labor and delivery and in the neonatal period, is another matter. In fact, as I speculate in the end, an important benefit of prenatal care would be to guarantee access to perinatal care.

This paper does not attempt to produce a definitive benefit-cost analysis of prenatal care. My goal here is to raise new questions and to suggest some future lines of investigation. These will be pursued in a later article.

PRENATAL CARE AND INFANT MORTALITY: AN INITIAL EXAMINATION

During 1975-76, there were 138,943 recorded live births in Massachusetts. Among live births, 1,229 infants (8.8 per 1000) died within 28 days of age. Also reported were 1,335 fetal deaths. In Massachusetts, reporting of fetal deaths beyond 20 weeks' gestation is legally required.

The analysis below is based upon information encoded in the individual birth certificates and, where applicable, matched death certificates of these cases. Infant deaths beyond the neonatal period (28 days of age) were not analyzed. Similar cross-section data bases on linked birth and death records were studied by Chase (1974,1977), Chase et al. (1973), Cunningham et al. (1976), Gortmaker (1979), Kane (1964), Kessner et al. (1973), Kleinman et al. (1978), Lewit (1977), Mellin (1972), Morris et al. (1975), Niswander and Gordon (1972), Pakter and Nelson (1974), Russell and Burke (1975), Shah and Abbey (1971), Shwartz (1962), Shwartz and Vinyard (1965), Slesinger and Travis (1975), Susser et al. (1972), Taylor (1970), Terris and Glasser (1974), Terris and Gold (1969), Williams (1975), and others.

Figure 1 depicts the crude relation between the total number of prenatal visits reported during pregnancy and the probability of neonatal death among all live births. Intervals of one standard error are shown around each point estimate of the neonatal mortality rate. The point at the extreme right of the Figure, corresponding to "?" on the abscissa, represents the neonatal death rate among women with an unknown number of prenatal visits. Although fetal deaths were excluded from the results shown in Figure 1, their inclusion does not alter the qualitative relationship indicated in the Figure.

On its face, Figure 1 suggests that the quantity of prenatal care-- as measured by the reported number of prenatal visits-- has a substantial effect on pregnancy outcome. Beyond an apparent minimum of three prenatal visits, the neonatal mortality rate rapidly declines. After approximately ten visits, however, there are apparent diminishing returns. Although the neonatal mortality rates beyond twenty visits are not very precise, the data suggest absolute decreasing returns to prenatal care. In fact, not one of these conclusions is justified by these data.

Fig. 1 →

To see this, we must ask why some women report three prenatal visits, while others report ten visits, and still others report twenty five visits.

It is established obstetric practice in Massachusetts, and throughout the United States, for expectant mothers to follow a recommended schedule of visits-- every four weeks for the first 28 weeks of pregnancy, every two weeks thereafter until the 36th week, then weekly until full term, and perhaps twice weekly if the baby is past due (U.S. National Center for Health Statistics, 1978; American College of Obstetricians and Gynecologists, 1974). The typical woman who recognizes her pregnancy at 6-8 weeks' gestation, follows the visit schedule recommended by her doctor, and delivers at 38-42 weeks will report about 10 to 15 visits. In fact, over two thirds of the women in the sample reported a quantity of care in this range.

Those women reporting a quantity of prenatal care outside this range, however, constitute a much less homogeneous group. One important subpopulation of pregnant women, apparently concentrated among lower income and poorly educated groups, and among unmarried mothers and those

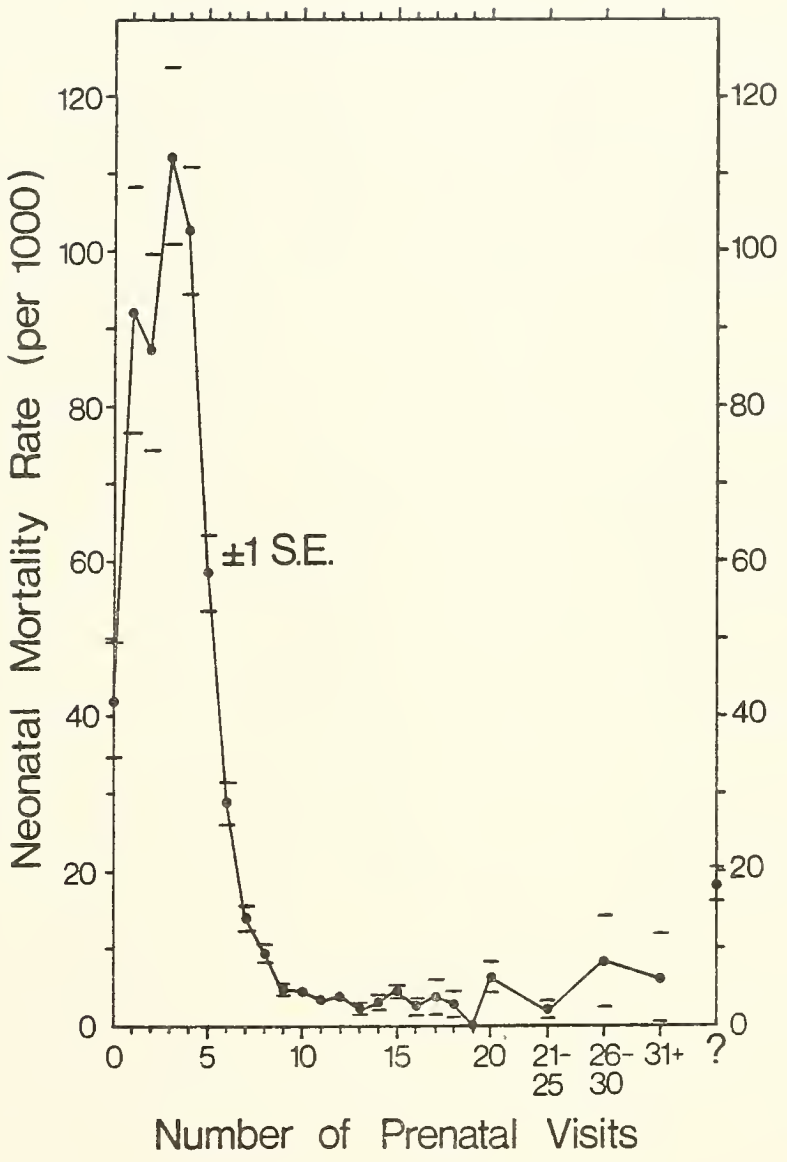

Figure 1. Relation Between Number of Prenatal Visits and Neonatal Mortality Rate per 1000 Live Births. Massachusetts, All Races, 1975-76.

of high parity, do not adhere to standard prenatal medical practice (Chase et al. 1973; Gortmaker 1979; Lewit 1977; U.S. National Center for Health Statistics 1978). These women may seek medical care only if they perceive some complication late in the course of pregnancy. With respect to such complications, those mothers with no prenatal care may therefore represent a population very different from those women with even one or two visits. In the range below ten prenatal visits, there is still another subpopulation of women who did follow the standard prenatal care schedule. As a result of placental insufficiency, infection, congenital anomalies, or other causes, their pregnancies-- and therefore the course of prenatal care-- were terminated prematurely. Yet another group of women with previously established high risks (e.g., diabetes, rheumatic heart disease) or with increased risk detected during pregnancy (e.g., preeclampsia, intrapartum bleeding) seek care earlier and make more frequent prenatal visits. Among women with a large number of prenatal visits, however, there is also a group who were frequently monitored solely because they remained pregnant beyond their expected date of delivery.

Finally, 5.5 percent of live births of black women and 1.7 percent of live births of white women were recorded to have an unknown number of prenatal visits. Among those records with missing data on prenatal care, but with completed information on other characteristics, there was a disproportionate fraction of out-of-wedlock, higher order births, and teenage pregnancies. Since prenatal care information on live birth certificates is typically completed by hospital staff personnel and not by the mother, missing data are more likely to occur when the patient

has no prior hospital record of the pregnancy (U.S. National Center for Health Statistics 1978, Technical Appendix). The unknown prenatal care category is therefore very likely to contain a disproportionate fraction of women with no prenatal care.

These facts seriously complicate the interpretation of Figure 1. Since early termination of pregnancy interrupts the normal course of prenatal care, the marked decline in neonatal mortality in the range of 3 to 10 visits could mean merely that the extent of care is a proxy variable for fetal maturity. If the group with an unknown number of visits is composed primarily of women with no care, then the observed neonatal mortality for women who reported no care may be substantially overstated. Aside from this possible bias, the elevated neonatal mortality rate of the no care group could reflect poor socioeconomic status, illegitimacy, or other covariates of the demand for care. The increasing mortality rate in the range from 0 to 3 visits, moreover, could reflect the higher complication rate among mothers who seek care only late in gestation. The possibility of increased mortality in the range beyond 15 visits could merely reflect the higher medical risks of some mothers in this group.

PRENATAL CARE AND THE DURATION OF PREGNANCY

The first necessary step in unravelling these difficulties is to examine in detail the relation between prenatal care and gestational age.

Many investigators (e.g., Eastman 1947; Oppenheimer 1961; Pakter et al. 1961) have noted that mothers with little or no prenatal care have substantially higher rates of pre-term delivery. It has not gone unnoticed, however, that shortened gestation may interrupt the standard prenatal care schedule, and therefore induce a spurious correlation between prematurity and the total number of visits (Drillien 1957; Hellman 1953; Kane 1964; Shwartz 1962; Shwartz and Vinyard 1965; Terris and Glasser 1974; Terris and Gold 1969). Terris and Glasser (1974) recognized that this spurious correlation also applied to the time of initiation of care, since the interval to the first prenatal visit might just as well be truncated by early termination of pregnancy. Statistically adjusted measures of prenatal care, such as the average number of visits per week of gestation, were similarly inappropriate because the frequency of visits on the standard schedule increased later in pregnancy.

Despite its repeated recognition, this paradox remains unresolved. Studies of the effect of prenatal care on other dimensions of pregnancy outcome (such as birth weight and mortality) have merely capitulated that the quantity of prenatal care and the duration of pregnancy were confounded variables. Hence, measurement of prenatal care was somehow to be adjusted for gestational age. Kane (1964), for example, excluded cases delivered prior to 38 weeks, while Chase et al. (1973, Table 3.9) excluded cases delivered prior to 36 weeks. Lewit (1977), and Russell and Burke (1975), included gestational age as an additional

explanatory variable in ordinary least squares regressions of prenatal care on birth weight and infant death. (The fact that their linear specifications failed to correct for the nonlinearly increasing frequency of visits at the end of pregnancy was overlooked.) Wells et al. (1958) similarly adjusted for length of gestational age in an analysis of covariance of prenatal care and perinatal death. The frequently cited Institute of Medicine study of New York City births in 1968 (Kessner et al. 1973, p. 59) constructed an a priori index of prenatal care "adequacy", determined by the number of prenatal visits adjusted for gestational age. A given schedule was deemed "adequate" in this study only if the mother had private obstetrical care. The same adequacy index, exclusive of the private obstetrical care requirement, was subsequently used by Gortmaker(1979) in a multiple contingency table analysis. As in the Institute of Medicine study, this author assigned all observations with unknown care to the "inadequate" category (Gortmaker 1979, Appendix A).

None of these studies has had any bearing on the causal relation between prenatal care and the duration of pregnancy. The possibilities that prenatal attention could suppress early labor, or identify overdue mothers requiring induced labor, or screen out fetuses that are subsequently ill-fated, remain untested.

Prenatal Care and Premature Delivery as Competing Risks: At any time during gestation, a woman is subject to some instantaneous risk of termination of pregnancy. This risk of termination will depend upon the duration of pregnancy thus far, as well as other maternal and infant characteristics. It could depend upon the presence of prenatal medical

attention. The timing of prenatal care also represents a type of risk. That is, at any time during gestation, there is some instantaneous probability that a woman will make a prenatal visit. In particular, there is some instantaneous probability that a woman thus far without care will initiate prenatal care. This risk of visiting the doctor will depend in turn upon various maternal and infant characteristics.

Our problem is that the risk of visiting the doctor and the risk of termination of pregnancy are in competition. Among women who received no prenatal care, the termination of pregnancy occurred, in effect, before the initial visit could take place. Among those who did receive care, the initial visit occurred before the termination of pregnancy. In this context, we may inquire whether the initiation of prenatal care (when it does occur prior to termination of pregnancy) modifies the subsequent risk of pregnancy termination.

Let $\lambda_V(v)$ and $\lambda_T(t)$, respectively, be the instantaneous risks (or "hazard rates") for making an initial visit and for termination of pregnancy. The rate $\lambda_V(v)$ is the probability that prenatal care is initiated in the short interval $(v,v+dv)$, given that no care has been received prior to time v. The rate $\lambda_T(t)$ is the probability that pregancy will terminate in the short interval $(t,t+dt)$, given that gestation has lasted until time t. The concept underlying the hazard $\lambda_V(v)$ has been mentioned only once in the literature (Terris and Glasser 1974). The hazard $\lambda_T(t)$ is the more familiar gestational age-specific force of exit in a fetal life table (Bakketeig et al. 1978; Mellin 1962; Taylor 1970).

Consider the event that pregnancy terminates without prenatal care at time t. (Time is measured from the point of conception.) Provided that

the risks of initiation of care and termination of pregnancy are initially independent, the probability of this event is

$$\lambda_T(t)\exp[-\int_0^t \lambda_T(s)ds] \times \exp[-\int_0^t \lambda_V(s)ds] \qquad (1)$$

The first expression in (1) is the probability that pregnancy terminated at time t. The second expression is the probability that prenatal care was not sought in the interval [0,t]. (See David and Moeschberger 1978; Lancaster 1979).

Let $\lambda_{TV}(t|v)$ be the risk of termination of pregnancy at time t, given that prenatal care was initiated at time $v \leq t$. The interdependence of hazard rates captured by this notation is a special case of the more general hypothesis that the number and timing of each prenatal visit affects the risk of termination of pregnancy. Now consider the event that care is initiated at time v and pregnancy subsequently terminates at time t. The probability of this event is

$$\lambda_V(v)\exp[-\int_0^v \lambda_V(s)ds]\exp[-\int_0^v \lambda_T(s)ds] \times \lambda_{TV}(t|v)\exp[-\int_v^t \lambda_{TV}(s|v)ds] \qquad (2)$$

The first expression in (2) is the probability that prenatal care is initiated at time v and pregnancy did not terminate in the interval [0,v]. The second expression is the probability of termination of pregnancy at time t given that prenatal care was initiated at time v and that the pregnancy was intact at time v.

The hypothesis that the presence of care affects the subsequent rate of termination of pregnancy means that $\lambda_{TV}(t|v) \neq \lambda_T(t)$. When $\lambda_{TV} < \lambda_T$, prenatal care slows down the rate of termination of pregnancy, that is, it prevents prematurity. When $\lambda_{TV} > \lambda_V$, prenatal care accelerates

the termination of pregnancy.

An Illustrative Test: Figure 2 depicts the frequency distribution of length of gestation among mothers with and without prenatal care in Massachusetts in 1975-76. The results in Figure 2 confirm the association between prenatal care and full term gestation. Twenty nine percent of mothers with no prenatal care, as opposed to five percent of mothers with some prenatal care, had gestations less than 36 weeks' duration. (Although Massachusetts requires reporting only of pregnancies of 20 weeks' duration, a small fraction of the sample included pregnancies of shorter duration.)

Fig. 2 →

To construct a statistical test of the hypothesis that prenatal care affects the duration of gestation, we must impose some additional restrictions on the data and our model. First, I exclude cases with unknown prenatal care and unknown gestational age. (These are omitted in Figure 2 and constitute 4 percent of the entire sample.) In this illustration, the problem of nonrandom missing observations is therefore not addressed. Second, I consider both live births and fetal deaths. Inclusion of fetal deaths admits the possibility that prenatal care prevents spontaneous abortion or other causes of premature delivery resulting in death during labor. Third, I examine only a subsample of 6,736 black women's pregnancies. The alternative of analyzing the pregnancies of women of all races, with ad hoc indicator variables for each race, does not appear warranted at this stage. The effects of prenatal care among black mothers may differ considerably from the corresponding effects among other races.

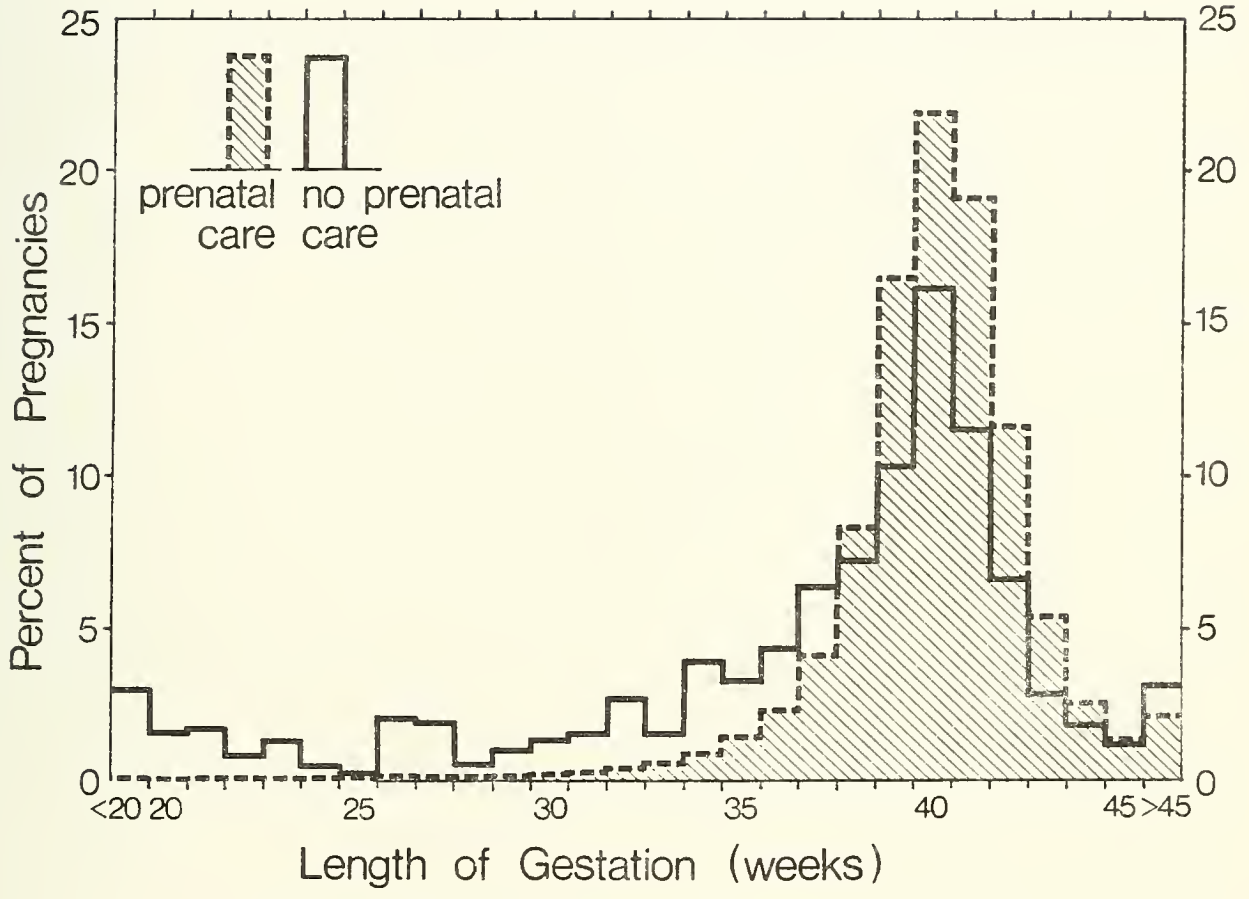

Figure 2. Frequency Distribution of Length of Gestation Among Women With and Without Prenatal Care. Live Births and Fetal Deaths, Massachusetts, All Races, 1975-76.

I further restrict the model to the proportional hazards form

$$\lambda_{TV}(t|v) = (1+\alpha)\lambda_T(t) \qquad (3)$$

where $\alpha > -1$ is a constant, independent of t and v. Although the instantaneous effect of prenatal care on the rate of termination of pregnancy is assumed to be time-independent, the total effect of prenatal care on the duration of pregnancy will nevertheless depend upon the time of initiation of care.

To complete the statistical model, we must specify how the hazards λ_T and λ_V depend upon time and other observed characteristics. Let $\underset{\sim}{X} = (X_1,\ldots,X_K)$ be a vector of explanatory variables. I assume that λ_T and λ_V depend upon t and $\underset{\sim}{X}$ in the following way

$$\begin{aligned}\lambda_T(t|\underset{\sim}{X}) &= (\rho_T\omega_T)(\rho_T t)^{\omega_T-1}\prod_{k=1}^{K}(1+\theta_{Tk}X_k)\\ \lambda_V(v|\underset{\sim}{X}) &= (\rho_V\omega_V)(\rho_V v)^{\omega_V-1}\prod_{k=1}^{K}(1+\theta_{Vk}X_k)\end{aligned} \qquad (4)$$

The expressions $(\rho_T\omega_T)(\rho_T t)^{\omega_T-1}$ and $(\rho_V\omega_V)(\rho_V v)^{\omega_V-1}$ are Weibull hazard functions. The parameters ω_T and ω_V, in particular, incorporate the possibility that the rates of termination of pregnancy and initiation of prenatal care are time dependent. The hazard rate increases monotonically for $\omega > 1$, decreases monotonically for $\omega < 1$, and remains constant for $\omega = 1$. In the expressions $\Pi_k(1+\theta_{Tk}X_k)$ and $\Pi_k(1+\theta_{Vk}X_k)$, each parameter θ corresponds to the incremental effect of a given explanatory variable on one of the hazard rates. Each multiplicand $(1+\theta X)$ represents the contribution of a specific explanatory variable to the proportional risks of termination of pregnancy and initiation of care. Under the restrictions (4), these proportional risks are assumed to be independent of gestational

age. Similarly, under the restriction (3), the expression $(1+\alpha)$ represents the contribution of prenatal care to the relative risk of termination of pregnancy.

Suppose that we have independent observations $\{t_i, \underset{\sim}{X}_i : i=1,\ldots,N\}$ on the durations of pregnancy and other explanatory variables for mothers with no prenatal care, and independent observations $\{t_j, v_j, \underset{\sim}{X}_j : j=1,\ldots,M\}$ on the durations of pregnancy, times of initiation of care and other explanatory variables among mothers with prenatal care. If the data $\{t_i, t_j, v_j\}$ are observed in continuous time, then the joint likelihood of these N+M observations is

$$L^I = \prod_{i=1}^{N} \lambda_T(t_i|\underset{\sim}{X}_i)\exp[-\Lambda_T(t_i|\underset{\sim}{X}_i)]\exp[-\Lambda_V(t_i|\underset{\sim}{X}_i)]$$

$$\times \prod_{j=1}^{M} \lambda_V(v_j|\underset{\sim}{X}_j)\exp[-\Lambda_V(v_j|\underset{\sim}{X}_j)](1+\alpha)\lambda_T(t_j|\underset{\sim}{X}_j)\exp[-\Lambda_{TV}(t_j,v_j|\underset{\sim}{X}_j)] \quad (5)$$

where $\lambda_T(t|\underset{\sim}{X})$ and $\lambda_V(v|\underset{\sim}{X})$ are defined in (4), and where $\Lambda_T(t|\underset{\sim}{X}) = (\rho_T\omega_T)^{\omega_T} \prod_k (1+\theta_{Tk}X_k)$, and $\Lambda_V(v|\underset{\sim}{X}) = (\rho_V\omega_V)^{\omega_V} \prod_k (1+\theta_{Vk}X_k)$, and $\Lambda_{TV}(t,v|\underset{\sim}{X}) = (1+\alpha)\Lambda_T(t|\underset{\sim}{X}) - \alpha\Lambda_T(v|\underset{\sim}{X})$. This likelihood function, which I have superscripted with the numeral "I" to distinguish it from others used below, can be rewritten in the form

$$L^I = \prod_{i=1}^{N} \lambda_T(t_i|\underset{\sim}{X}_i)\exp[-\Lambda_T(t_i|\underset{\sim}{X}_i)]\cdot(1+\alpha)^M \cdot \prod_{j=1}^{M} \lambda_T(t_j|\underset{\sim}{X}_j)\exp[-\Lambda_{TV}(t_j,v_j|\underset{\sim}{X}_j)]$$

$$\times \prod_{i=1}^{N} \exp[-\Lambda_V(t_i|\underset{\sim}{X}_i)] \cdot \prod_{j=1}^{M} \lambda_V(v_j|\underset{\sim}{X}_j)\exp[-\Lambda_V(v_j|\underset{\sim}{X}_j)]$$

$$= L_T^I \times L_V^I \quad (6)$$

where L_T^I and L_V^I are multiplicatively separable in the parameters $\{\alpha, \omega_T, \rho_T, \theta_{Tk}\}$

and $\{\omega_V, \rho_V, \theta_{Vk}\}$ respectively. Hence, the maximum likelihood estimates of these two sets of parameters can be obtained separately without bias.

Table 1 displays the main characteristics of the subsample of black women's pregnancies. There were 82 neonatal deaths (12.3 per 1000 live births) and 43 fetal deaths. Among observations excluded from this sample because of missing information on birth weight, gestational age, initiation of prenatal care, or other explanatory variables, there were 29 additional neonatal deaths and 38 additional fetal deaths.

Table 1 →

Table 2 displays the maximum likelihood estimates of the parameters of L_T^I and L_V^I in (6). The estimate of the parameter α is -0.293. That is, prenatal care reduces the hazard rate for termination of pregnancy by 29.3 percent. The estimate of ω_T far exceeds 1. That is, the instantaneous risk of termination of pregnancy rises very rapidly with increasing gestational age. For the Weibull hazard function (4), the ratio of the mean gestational age of black mothers without prenatal care to the mean gestational age of black mothers with care throughout pregnancy is $(1+\hat{\alpha})^{1/\hat{\omega}_T} = 0.978$ (approximate standard error 0.007). That is, for a 40 week pregnancy, the absence of care reduces the mean gestational age by about 0.88 weeks (approximate standard error 0.28).

Table 2 also reveals statistically significant effects of attained education and prior fetal loss on the hazard of pregnancy termination. For a black women with 16 years of education, the risk of pregnancy termination is reduced by an estimated 12 percent relative to a black women with 8 years of education (i.e., $(1+16\hat{\theta}_{T1}) \div (1+8\hat{\theta}_{T1}) = 0.88$, standard error 0.04). The interpretation of the statistically significant effect of illegitimacy (X_4) is more complicated. Explanatory variables for such

potentially confounding factors as teenage pregnancy (X_3), first pregnancy (X_6), and reduced education (X_1) are held constant. Therefore, the estimated reduced risk of early termination of pregnancy for illegitimate births may reflect the experience of relatively older black women of higher parity. I shall return to this puzzling observation below.

Table 2 →

Table 2 shows that the hazard rate for the initiation of prenatal care also increases with gestational age (i.e. $\hat{\omega}_V = 1.63 > 1.$) The rate of initiation of care for a black women with 16 years of attained education is 41 percent greater than the rate of initiation of care for a black women with 8 years of attained education (i.e., $(1+16\hat{\theta}_{V1})\div(1+8\hat{\theta}_{V1}) = 1.41$, standard error 0.06). This estimate corresponds to a 19 percent reduction in the mean time to initiate care (standard error 2.1%). For an expectant black mother who seeks care at 12 weeks' gestation, this represents an average reduction of 2.3 weeks in the mean time to the first visit (standard error 0.26). The combined effect of illegitimacy, advanced maternal age, and previous pregnancies is substantial. A 35 year old multiparous woman delivering an illegitimate child has a rate of initiation of care one tenth that of a primagravida in her 20s delivering a legitimate child (i.e., $(1+5\hat{\theta}_{V2})\cdot(1+\hat{\theta}_{V4})\div(1+\hat{\theta}_{V6}) = 0.1$). This corresponds to an estimated 4-fold increase in the mean time to initiation of care (standard error 0.23).

These estimates were derived from a selected data base, with specifically chosen explanatory variables, and in the context of a specific parametric formulation. The conclusion that prenatal care reduces pre-term delivery may not withstand alternative data bases, explanatory

TABLE 1 -- SAMPLE CHARACTERISTICS OF 6,736 BLACK WOMEN'S PREGNANCIES. MASSACHUSETTS, 1975-76.

Number of Neonatal Deaths	82	
Number of Fetal Deaths	43	
Percent Initiated Care in First Trimester	74.5 %	
Percent Initiated Care in Second Trimester	20.9 %	
Percent Initiated Care in Third Trimester	3.6 %	
Percent with Prior Perinatal Loss[a]	17.3 %	
Percent Primagravida	39.7 %	
Percent Recorded Illegitimate	48.8 %	
Percent Aged Over 30 Years	12.6 %	
Percent Aged Under 20 Years	25.3 %	
Mean Gestational Age (weeks)	39.2	(s.d. 3.12)
Mean Duration of Prenatal Care (weeks)[b]	28.5	(s.d. 7.90)
Mean Birth Weight (grams)	3123.	(s.d. 619.)
Mean Attained Education (years)	11.6	(s.d. 0.22)
Mean Annual Volume of Deliveries at Hospital of Birth (thousands)	3.1	(s.d. 2.09)

[a] Includes prior neonatal death or prior fetal death of at least 20 weeks' duration.

[b] Data on initiation of prenatal care was recorded by month of pregnancy. Calculation of weeks of care assumed that prenatal care was initiated at the midpoint of the recorded month of pregnancy.

(s.d. = standard deviation)

TABLE 2 -- MAXIMUM LIKELIHOOD ESTIMATES OF THE EFFECT OF PRENATAL CARE ON THE RATE OF TERMINATION OF PREGNANCY. LIKELIHOOD MODEL I. 6,736 BLACK WOMEN, MASSACHUSETTS, 1975-76.

		Parameter Estimates	
		L_T^I	L_V^I
Effect of Prenatal Care	α	-0.293 (0.075)	-
Weibull Hazard Parameters	ω	15.631 (0.114)	1.626 (0.053)
	ρ	0.026 (0.0001)	0.060 (0.004)
Parameters of Explanatory Variables			
Years of Education	θ_1	-0.013 (0.004)	0.086 (0.020)
Years of Age over 30	θ_2	-0.003 (0.006)	-0.166 (0.006)
Years of Age under 20	θ_3	0.005 (0.010)	-0.076 (0.008)
Illegitimacy	θ_4	-0.071 (0.022)	-0.260 (0.017)
Prior Perinatal Loss	θ_5	0.091 (0.031)	-0.037 (0.029)
Primagravida	θ_6	-0.001 (0.028)	0.254 (0.032)
Log Likelihood		-16734.3	-21371.9

Standard errors in parentheses.

variables, or a formulation other than the proportional hazards model of (3) and (4). It is noteworthy, however, that the above estimate and standard error of the parameter α changed only minimally with inclusion or exclusion or other explanatory variables, such as type of care (private versus ward), the percentage of rental housing or the median income in the cnesus tract corresponding to the mother's residence, or alternative specifications of the effect of maternal education and age. The results did not change substantially when fetal deaths were excluded from the sample. Although I assumed that the week of initiation of care corresponded to the midpoint of the reported month of initiation of care, the use of a more complicated likelihood function that incorporated the interval characteristics of these data also did not substantially alter the results. Finally, when I included observations with unknown care in the analysis, assuming that these women in fact received no care, the estimate of α was reduced in absolute value to -0.20.

UNOBSERVED CHARACTERISTICS AND FETAL SELECTION

This analysis of the relation between prenatal care and the duration of pregnancy has so far overlooked one serious problem of interpretation. This difficulty is motivated by Figure 3, which depicts the relation between the month of initiation of care and the proportion of births of less than 36 weeks' gestation for white and black mothers. Intervals of one standard error are shown for blacks. The corresponding standard errors for whites were considerably smaller, and are omitted for clarity. Figure 3 shows that the increasing relation between late care and prematurity is interrupted during the third trimester. Since the thirty sixth week of gestation occurs during the ninth calendar month, this finding is not simply an artifact of the 36 week cutoff used in the Figure.

In any cohort of pregnant women, the initial fetal population is likely to be extremely heterogeneous in its health characteristics. If this heterogeneity is reflected in the hazard rates for termination of pregnancy -- with the least fit infants having the highest hazard rates -- then the phenomenon of fetal loss can play a powerful selective role. In comparison to the fetal population at the time of conception, those infants that have remained in utero up to the third trimester will necessarily contain a smaller fraction of ill-fated fetuses. One distinctive characteristic of mothers who initiate care in the third trimester is that their infants have remained in utero just that long. Hence, for no reason other than natural selection, late initiators of care may have infants with lower rates of pregnancy termination than earlier initiators of care. But this selection effect need not apply to mothers without care, whose infants may have been delivered at any time

during gestation. These phenomena are exactly reflected in Figure 3.

Fig. 3 →

If we could ascertain all the relevant determinants of variation in the hazard rates for termination of pregnancy, then in principle we could take full account of this selection phenomenon. The difficulty with this solution is not merely its cost. Even if we could assemble detailed data on fetal ultrasound measurements, urinary estriol levels, maternal weight gain, etc. on a large cross-section of women, there may still be substantial unobserved variation in fetal robustness. These unobserved characteristics will then be subject to selection. The inverse relation between late care and the duration of pregnancy might not be eliminated by conditioning on the observable characteristics.

Moreover, if the phenomenon of fetal loss selects out the least fit infants, then any factor that slows the rate of termination of pregnancy will also retard this selective process. If prenatal care, in particular, reduces the hazard rate for termination of pregnancy, then at any given week of gestation, those mothers who had early care will tend to have a higher proportion of less fit infants. This possibility is also consistent with the data in Figure 3.

The problem is further complicated if the mothers under study could ascertain those health characteristics of their infants that are not revealed to the analyst in the data. Mothers who perceive their babies to be less fit, or potentially less fit, may initiate care earlier, while those with uneventful pregnancies may delay care. This hypothesis would account not only for the lower proportion of pre-term deliveries among late initiators of care, but also for the higher proportion of preterm deliveries among mothers who initiated care in the first month (see Figure 3).

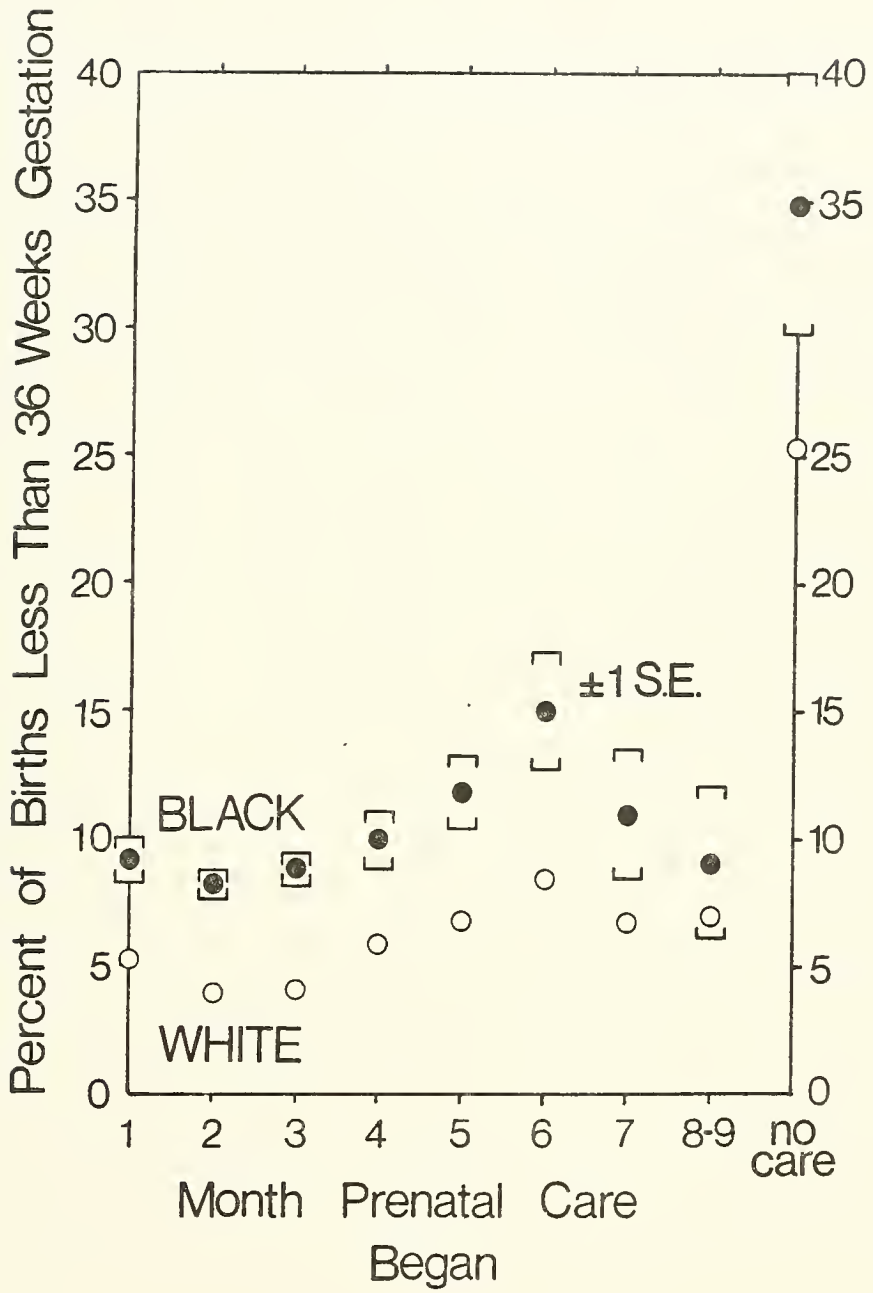

Figure 3. Relation Between Month of Initiation of Prenatal Care and Percent of Births Less Than 36 Weeks Gestation among White and Black Women. Live Births and Fetal Deaths, Massachusetts, 1975-76.

Our observations almost exclusively cover pregnancies of at least 20 weeks' duration. Hence, the cohort actually observed is likely to be more homogeneous than the original fetal cohort at the time of conception. The selective effect of fetal loss may therefore be less significant. Data from more complete fetal life table analyses (Bakketeig et al. 1978; Mellin 1962; Taylor 1970), extending back to the weeks immediately after conception, reveal an initial period of relatively high hazard rates of pregnancy termination. As the ill-fated fetuses are progressively eliminated from the cohort, the overall hazard rate gradually falls. After approximately 20 weeks, the hazard rate then begins to rise. This increase in the hazard rate, however, does not imply that only robust infants remain in utero beyond 20 weeks. There is still likely to be substantial remaining variation in fetal characteristics up to and including the fortieth week of pregnancy. Nevertheless, the censoring of early fetal deaths in our sample could bias estimates of the effect of prenatal care and other explanatory characteristics. If prenatal care prevented early fetal loss, for example, then it could extend an otherwise short pregnancy beyond the 20 week cutoff. On the other hand, the early medical attention could permit some women and their physicians to screen out and terminate an eventually ill-fated pregnancy before 20 weeks.

It is not at all clear how these complicated structural relationships can be identified with the available cross section data. One possible strategy is to specify a model of the fetal selection process, and then to investigate how that model affects our inferences about the effects of prenatal care and other explanatory variables. Such a model of fetal

selection will now be considered.

Let ε be a scalar index of fetal "defectiveness", whose values are restricted to the positive real numbers. Infants with low values of ε are more robust than infants with high values of ε. Although fetal defectiveness cannot be directly observed, it is assumed to affect the hazard rate for pregnancy termination. I shall denote this dependence by $\lambda_T(t|\underset{\sim}{X},\varepsilon)$, retaining the specification that $\lambda_{TV} = (1+\alpha)\lambda_T$. For a given cohort of pregnant women, ε initially has probability density $f(\varepsilon)$. Now let $G_{TV}(t|v,\underset{\sim}{X},\varepsilon)$ be the probability that a pregnancy of defectiveness ε, with observed characteristics $\underset{\sim}{X}$ and time of initiation of care v, survives at least to gestational age t. Then, by Bayes Rule, the probability density of ε among those infants with characteristics $(v,\underset{\sim}{X})$ who remain in utero at least to age t is

$$f(\varepsilon|t,v,\underset{\sim}{X}) = \frac{G_{TV}(t|v,\underset{\sim}{X},\varepsilon)f(\varepsilon)}{\int_0^\infty G_{TV}(t|v,\underset{\sim}{X},\zeta)f(\zeta)d\zeta} \qquad (7)$$

A similar formula applies to mothers with no prenatal care, where G_{TV} is replaced by G_T, the corresponding probability of survival.

If λ_T is an increasing function of ε, then for a given $(v,\underset{\sim}{X})$, both the mean and variance of $f(\varepsilon|t,v,\underset{\sim}{X})$ decline with increasing t. That is, as a result of fetal selection, those infants remaining in utero are on average less defective and more homogeneous as gestation advances.

In keeping with the proportional hazards specification, I now let $\lambda_T(t|\underset{\sim}{X},\varepsilon) = \lambda_T(t|\underset{\sim}{X})\varepsilon$. Moreover, I let ε have a gamma density with mean 1 and variance 1/h. That is,

$$f(\varepsilon) \propto \exp[-h\varepsilon]\varepsilon^{h-1} \tag{8}$$

Then for pregnancies with defectiveness ε and characteristics $(v,\underset{\sim}{X})$,

$$G_{TV}(t|v,\underset{\sim}{X},\varepsilon) = \exp[-\Lambda_{TV}(t,v|\underset{\sim}{X})\varepsilon] \tag{9}$$

where, in the case of mothers with no care, Λ_{TV} is replaced by Λ_T. From (7), (8), and (9), we obtain

$$f(\varepsilon|t,v,\underset{\sim}{X}) \propto \exp[-(h+\Lambda_{TV}(t,v|\underset{\sim}{X}))\varepsilon]\varepsilon^{h-1} \tag{10}$$

where, again, in the case of mothers with no care, Λ_{TV} is replaced by Λ_T. The conditional density of ε is therefore also gamma, but with mean $h/(h+\Lambda_{TV}) < 1$ and variance $h/(h+\Lambda_{TV})^2 < 1/h$. Since Λ_{TV} is an increasing function of t, the mean and variance of ε decline with gestational age. Moreover, if $\alpha < 0$, then Λ_{TV} is an increasing function of v. That is, late care accelerates the process of selecting the least defective infants.

From (9), the probability that a woman will still be pregnant at t, given characteristics $\underset{\sim}{X}$ and initiation of care at v, is

$$G_{TV}(t|v,\underset{\sim}{X}) = \int_0^\infty G_{TV}(t|v,\underset{\sim}{X},\varepsilon)f(\varepsilon)d\varepsilon = \left[\frac{h}{h+\Lambda_{TV}(t,v|\underset{\sim}{X})}\right]^h \tag{11}$$

The probability that a woman will deliver at t, given initiation of care at v and characteristics $\underset{\sim}{X}$, is therefore

$$\left[-\frac{\partial}{\partial t} G_{TV}(t|v,\underset{\sim}{X}) \right]\cdot G_{TV}(t|v,\underset{\sim}{X}) = (1+\alpha)\lambda_T(t|\underset{\sim}{X})\left[\frac{h}{h+\Lambda_{TV}(t,v|\underset{\sim}{X})}\right]^{h+1} \tag{12}$$

Now suppose that we have independent observations $\{t_i,\underset{\sim}{X}_i: i=1,\ldots,N\}$

on the durations of pregnancy and characteristics of women with no care, and independent observations $\{t_j, v_j, X_j : j=1,\ldots,M\}$ on the durations of pregnancy, the times of initiation of care, and the characteristics of women with prenatal care. If there are no unobserved determinants of the hazard rate λ_V for initiation of care, then the joint likelihood of these observations is $L_V^I \times L_T^{II}$, where

$$L_T^{II} = \prod_{i=1}^{N} \lambda_T(t_i|X_i)\left[\frac{h}{h+\Lambda_T(t_i|X_i)}\right]^{h+1} \cdot (1+\alpha)^M \prod_{j=1}^{M} \lambda_T(t_j|X_j)\left[\frac{h}{h+\Lambda_{TV}(t_j,v_j|X_j)}\right]^{h+1}$$

...(13)

Maximum likelihood estimates of the parameters $\{\alpha, \rho_T, \omega_T, \theta_{Tk}, 1/h\}$ in L_T^{II} are presented in Table 3. The estimate of the variance of ε is significantly different from zero. The maximum value of $\log L_T^{II}$ is substantially greater than the corresponding maximum value of $\log L_T^I$ in Table 2. Strictly speaking, model I is the limiting case of model II for $1/h \downarrow 0$. Therefore, its parameters are not properly in the interior of the parameter space of Model II. But it is sufficient to note that a null hypothesis of any arbitrarily small value of 1/h will be rejected in favor of Model II, and that L_T^{II} is right hand continuous at 1/h = 0. Hence, Model II represents a substantially better fit than model I.

Table 3 →

The maximum likelihood estimate of α in Table 3 is -0.351, as compared to -0.293 in Table 2. That is, our previous failure to account for fetal selection in Model I resulted in a biased estimate of the effect of prenatal care. The magnitude of this bias, however, is not too large. For example, the ratio of the mean gestational age of black mothers without prenatal care to the mean gestational age of black mothers with care throughout pregnancy is $(1+\hat{\alpha})^{1/\hat{\omega}_T} = 0.977$ (standard error 0.008). (Under

TABLE 3 -- MAXIMUM LIKELIHOOD ESTIMATES OF THE EFFECT OF PRENATAL CARE ON THE RATE OF TERMINATION OF PREGNANCY. MODEL II, INCORPORATING UNOBSERVED REGRESSORS. 6,736 BLACK WOMEN, MASSACHUSETTS, 1975-76.

		Parameter Estimates	
		L_T^{II}	L_V^{II}
Effect of Prenatal Care	α	-0.351 (0.084)	-
Weibull Hazard Parameters	ω	18.217 (0.198)	2.094 (0.039)
	ρ	0.026 (0.0002)	0.072 (0.006)
Parameters of Explanatory Variables			
Years of Education	θ_1	-0.012 (0.006)	0.115 (0.037)
Years of Age over 30	θ_2	-0.004 (0.008)	-0.019 (0.008)
Years of Age under 20	θ_3	0.012 (0.014)	-0.104 (0.008)
Illegitimacy	θ_4	-0.044 (0.032)	-0.305 (0.025)
Prior Perinatal Loss	θ_5	0.134 (0.048)	-0.027 (0.046)
Primagravida	θ_6	-0.028 (0.036)	0.313 (0.053)
Variance of Unobserved Regressor	1/h	0.207 (0.018)	0.427 (0.034)
Log Likelihood		-16652.9	-21214.7

Standard errors in parentheses.

our proportional hazards specification, this ratio is independent of ε.) That is, for a 40 week pregnancy, the absence of care reduces the mean gestational age by about 0.94 weeks (standard error 0.32). For the parameter estimates in Model I, the corresponding reduction was 0.88 weeks (standard error 0.28).

The maximum likelihood estimate of ω_T in Table 3 is significantly greater than that in Table 2. When we ignore fetal selection, the hazard rate appears to rise more slowly because the high-ε (less robust) fetuses are being progressively eliminated from the cohort (See also Lancaster 1979). Similarly, the estimate of θ_{T5} in Table 3 exceeds the corresponding estimate in Table 2. That is, fetal selection operates more effectively on mothers with a prior history of fetal loss, and therefore failure to account for fetal selection leads to underestimates of the impact of this risk factor.

The model of equations (7) through (13) applies to the omitted regressor ε in the determination of the hazard rate λ_T. But a completely analogous argument could be applied to the determination of the hazard rate λ_V. If we assume that $\lambda_V(v|\underset{\sim}{X},\delta) = \lambda_V(v|\underset{\sim}{X})\delta$, where δ is the unobserved characteristic, and if δ similarly has a gamma density at the onset of pregnancy, and if δ is distributed independently of ε , then we can derive a likelihood L_V^{II} in a manner analogous to that for L_T^{II}.

Maximum likelihood estimates of the analogous parameters for L_V^{II} are presented in the right hand column of Table 3. Again, the log likelihood substantially exceeds that in Table 2, and the estimate of the variance of the unobserved regressor has a small standard error. The estimate of ω_V is similarly increased. Moreover, many of the

estimates of θ_{Vk} in Table 3 differ significantly from those in Table 2. For example, since improved education accelerates the rate of initiation of care, it will tend to eliminate high-δ mothers from the cohort, and thus reduce the average hazard rate for initiation of care. Hence, the estimates of the effect of attained education in Table 2 will be biased downward. This is confirmed in Table 3.

The most important limitation of this analysis is the assumption that the unobserved regressors ε and δ are independently distributed. This restrictive assumption does not admit the possibility that fetal and maternal health characteristics are correlated with prenatal care demand factors and in particular, that mothers' perception of the health of their pregnancy affects the rate of initiation of care. For example, the statistically significant negative estimate in Table 2 of the effect of illegitimacy status on the hazard rate of pregnancy termination is pulled toward zero but remains negative in Table 3. Among black women delivering children out of wedlock, especially those of high parity, some mothers may have very low risk pregnancies. Those who anticipate an uneventful pregnancy may also have much lower rates of initiation of care. If we do not take account of fetal selection, illegitimacy status appears to deter pre-term delivery. The introduction of two independent sources of variation in the hazards λ_T and λ_V apparently eliminates some of this bias. But it does not fully incorporate the possibility that the underlying health of the pregnancy affects the demand for care.

One possible solution to this difficulty is to allow for interdependence of the omitted regressors ε and δ. In the instant case, this suggestion

would require a joint distribution whose marginal densities are gamma. Although there is a class of such bivariate gamma distributions (Johnson and Kotz 1972), they do not appear to admit a correlation coefficient that can assume both positive and negative values. More important, my preliminary experiments with such bivariate densities suggested that the correlation coefficient between ε and δ and the parameter α could not be simultaneously identified. For the present competing risk model, it appears difficult if not impossible to ascertain both the effect of prenatal care on the subsequent risk of preterm delivery and the possible feedback effect of the underlying health of the pregnancy on the demand for care.

PRENATAL CARE AND THE RATE OF INTRAUTERINE GROWTH

I now focus on the relation between prenatal care and birth weight. Since the duration of pregnancy indirectly affects weight at birth, I study the effect of prenatal care on birth weight conditional upon gestational age.

Figure 4 shows the relation between gestational age and mean birth weight according to the trimester of initiation of care, among mothers of all races in Massachusetts during 1975-76. Both live births and fetal deaths are included. These data correspond to the empirical intrauterine growth curves of the obstetrical literature (Gruenwald 1966, 1974; Lubchenco 1975; Williams 1975).

The evidence in Figure 4 appears to confirm the commonplace finding that prenatal care improves birth weight, conditional upon gestational age (Chase et al. 1973; Gortmaker 1979; Kessner et al. 1973; Lewit 1977; Russell and Burke 1975; Shah and Abbey 1971; U.S. National Center for Health Statistics 1978). In the range from 39 to 42 weeks' gestation, mothers who initiated care in the first trimester have infants with mean birth weights 200 to 300 grams greater than mothers who received no care. The relation between the timing of care and birth weight follows a dose-response pattern.

Fig. 4

The results in Figure 4 could merely reflect the confounding influence of such factors as education, socioeconomic status, and race, which could affect both the timing of care and birth weight. To eliminate this possibility, we must specify a model of the effect of care on birth weight, conditional upon these potentially confounding variables as well as gestational age. As in the previous sections, it is more appropriate to confine the analysis to a single race, rather than to employ an ad hoc

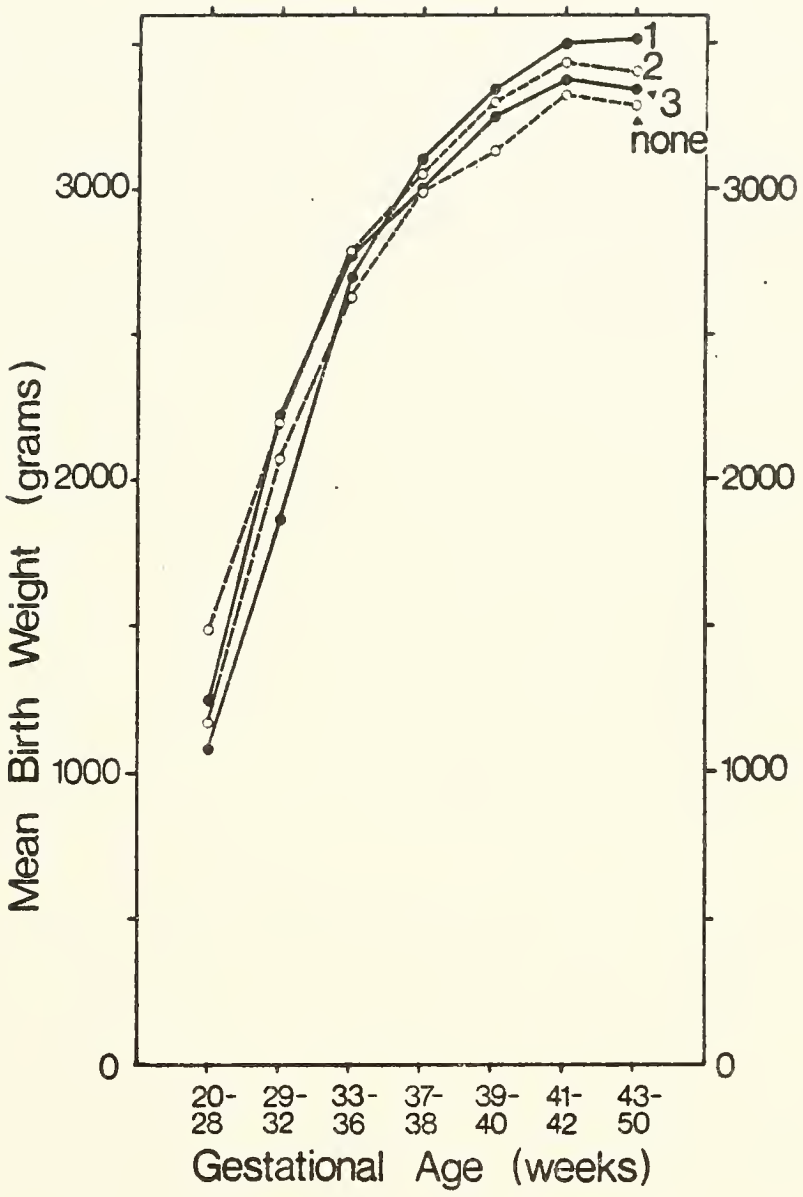

Figure 4. Relation Between Gestational Age and Mean Birth Weight According to Trimester of Initiation of Prenatal Care. Live Births and Fetal Deaths, Massachusetts, All Races, 1975-76.

(Note the gestational age intervals are of unequal duration.)

indicator variable for race in a study of the entire sample. Beyond that, however, the choice of an appropriate model is not clear.

One complicating factor is that the data of Figure 4 represent weight at birth among a cross section of infants of different gestational ages, and not the intrauterine growth curve of any one infant during the course of pregnancy. If there is a systematic relation between the duration of gestation and the rate of intrauterine growth across infants, then the slopes of the empirical curves in Figure 4 are biased measures of the rate of intrauterine growth. Since the determinants of these variations in the risk of pregnancy termination or the rate of intrauterine growth may be difficult to observe, we must again confront the problem of fetal selection. This means that prenatal care and other explanatory factors will affect not only the intrauterine growth rate of a given infant, but also the distribution of these unobserved factors across infants. Unless we are prepared to make strong parametric assumptions, the net effect of these complicated interactions is not obvious.

In order to compare the effects of prenatal care on intrauterine growth rates with the previously discussed effects on the duration of pregnancy, I shall specify a relatively simple model. Let the rate of growth of fetal weight be a function of gestational age and other explanatory factors, including the extent of prenatal care. I shall assume that this function takes the form

$$\frac{dw}{dt} = \Phi(t,\underset{\sim}{X})\Psi(v) + \nu \qquad (14)$$

where dw/dt is the growth rate of weight, t is gestational age, $\underset{\sim}{X}$ is

a vector of explanatory variables, $\Psi(v)$ measures the proportional effect of prenatal care, and ν is a stochastic error term. I further approximate $\Phi(t,X)$ by the polynomial

$$\Phi(t,\underset{\sim}{X}) = \beta_1 + 2\beta_2 t + 3\beta_3 t^2 + \sum_{k=1}^{K} \eta_k X_k \qquad (15)$$

In accord with the presentation of the data in Figure 4, I let

$$\Psi(v) = \prod_{i=1}^{3} (1+\gamma_i Y_i) \qquad (16)$$

where $Y_i = 1$ if initiation of care occurs in trimester i, and zero otherwise. From (14), (15), and (16), and the initial condition $w(0) = 0$,

$$w = (\beta_1 t + \beta_2 t^2 + \beta_3 t^3 + \sum_{k=1}^{K} \eta_k X_k t) \prod_{i=1}^{3} (1+\gamma_i Y_i) + \nu t \qquad (17)$$

In this parameterization, the parameters η_k measure the absolute contribution of each explanatory variable X_k to the rate of intrauterine growth (in grams per week), while the parameters γ_i measure the proportional effect of prenatal care. Moreover, the variance of the stochastic error νt increases with gestational age. A simple regression model of absolute birth weight with homoskedastic errors would therefore attach too much statistical weight to the high gestational age infants.

Table 4 presents maximum likelihood estimates of the parameters of (17) under the assumption that the error component νt is normally distributed with mean zero and variance $\sigma^2 t^2$. The estimated effects of maternal age, legitimacy status, prior perinatal loss, and parity are statistically significant. Prenatal care appears to increase the rate of intrauterine growth by about 2 percent in comparison to no care. But the null hypothesis of zero effect cannot be rejected at the 5 percent significance level. Moreover, there is no clear dose-response relation

between the onset of care and the rate of intrauterine growth.

Table 4 → Although the specification (17) is hardly general, the finding of a weak effect of prenatal care on birth weight was reproduced when I specified an additive effect for care, rather than the multiplicative form (14), or when I employed alternative measures of the quantity of care, or when I allowed for different multiplicative interactions between prenatal care and other explanatory variables.

Consider a multiparous, married black mother in her 20s, with 12 years of education, with no prior history of perinatal loss. If she received no prenatal care and delivered at 38 weeks, then from Table 4 her infant's birth weight is expected to be 3063 grams. If we hold constant the duration of pregnancy, the effect of prenatal care initiated in the first trimester is to add an expected 61 grams to birth weight. However, if we calculate the total effect of initiation of care in the first trimester, inclusive of its effect on gestational age (about 1 week), then prenatal care adds an expected 169 grams to birth weight. With respect to the determination of birth weight, the contributing effect of prenatal care on the rate of intrauterine growth is about 60 percent of the contributing effect of prenatal care on gestational age.

The finding of a relatively weak effect of prenatal care on intrauterine growth rates among black infants is not so surprising. Although retarded fetal growth (in particular, placental insufficiency) can be detected during pregnancy, there is little in the way of treatment (Shearman et al. 1974). Although maternal cigarette smoking substantially retards intrauterine growth (Hasselmeyer et al. 1980), there is little

TABLE 4 -- ESTIMATES OF THE EFFECT OF TRIMESTER OF INITIATION OF PRENATAL CARE ON THE RATE OF INTRAUTERINE GROWTH. 6,736 BLACK WOMEN, MASSACHUSETTS, 1975-76.

		Parameter Estimates
Constant Term	β_1	-183.946 (7.775)
Gastational Age (weeks)	β_2	13.612 (0.479)
Gestational Age Squared (weeks^2)	β_3	-0.175 (0.007)
Years of Education	η_1	0.042 (0.080)
Years of Age over 30	η_2	0.533 (0.093)
Years of Age under 20	η_3	-0.197 (0.147)
Illegitimacy	η_4	-2.293 (0.377)
Prior Perinatal Loss	η_5	-1.265 (0.463)
Primagravida	η_6	-1.208 (0.405)
Initiated Care 1st Trimester	γ_1	0.020 (0.020)
Initiated Care 2nd Trimester	γ_2	0.024 (0.020)
Initiated Care 3rd Trimester	γ_3	0.027 (0.024)
Variance of Error Term	σ^2	187.622 (2.180)

Standard errors in parentheses.

evidence that the advice of medical practitioners has affected this practice. Only approximately 30 percent of current female smokers of all races quit smoking during pregnancy. Among women of all races who were last pregnant during the period 1965-75, only 35 percent of cigarette smokers reported receiving any physician advice (J. Harris, unpublished). Nor can I find any evidence that prental care has induced mothers to forego alcohol abuse. Despite all the recent advances in understanding nutrition and maternal weight gain (Niswander, Gordon et al. 1972; Habicht et al. 1974), a recent controlled trial of nutritional supplementation among black women in New York City yielded negative results (Rush et al. 1980). This study has the striking interpretation that caloric supplementation for pregnant mothers merely ends up distributed to the remaining family members (Jacobson 1980).

PRENATAL CARE AND INFANT MORTALITY: A REPEAT EXAMINATION

So far, I have investigated the relation between prenatal care and the duration of pregnancy, as well as the relation between prenatal care and the rate of intrauterine growth. I now return to the relation between prenatal care and infant mortality.

Birth weight has been found repeatedly to be a critical determinant of perinatal survival (Cunningham et al. 1976; Niswander, Gordon et al. 1972; Shah and Abbey 1971; Shapiro, Schlesinger and Nesbitt 1968; U.S. National Center for Health Statistics 1965, 1972). At any given birth weight, neonates of preterm gestational age are at greater risk than full term infants (Susser, Marolla, and Fleiss 1972). The consensus of the literature, however, is that prenatal care exerts an influence on mortality solely through its effect on birth weight. The Institute of Medicine study, for example, noted that in a linear regression with infant death as a dependent variable, the addition of a medical care "adequacy" index plus six other independent variables had no explanatory power beyond that of birth weight alone (Kessner et al. 1973, p. 63). In Gortmaker's (1979) multiple contingency table analysis, prenatal care had no consistent effect on neonatal mortality among white mothers when birth weight was included as a predetermined variable. Among black mothers, prenatal care of "intermediate" adequacy (as opposed to "adequate" or "inadequate" care) was found to have a significant effect. Shah and Abbey (1972) similarly found birth weight to be the critical intervening variable in the determination of neonatal and post-neonatal survival. Neonatal mortality, adjusted for birth weight, they found, was lower among women who initiated care in the third trimester .

The problem with these conclusions is that they do not square with a critical fact about the recent, renewed decline in infant mortality in the United States.

During 1965 to 1970, the U.S. infant mortality rate declined from 24.7 to 20.0 deaths per 1000 live births, an absolute decrease in the mortality rate equal to that for the entire period 1950 to 1965. By 1978, the U.S. infant mortality rate had reached an estimated 13.6 per 1000 (U.S. National Center for Health Statistics 1977, 1979, 1980a). In contrast to the pattern of mortality decline during the first half of this century, most of the recent absolute decline in infant mortality represented an improvement in neonatal survival. At least beyond 20 weeks' gestation, a substantial decline in fetal death rates was also observed. These improvements in infant survival applied to all races.

Figure 5 depicts the relation between birth weight and neonatal mortality, determined from matched birth and death records, for the U.S. in early 1950 and 1960 (U.S. National Center for Health Statistics 1972, Table D), and for Massachusetts during 1969 to 1978 (Massachusetts Department of Public Health, unpublished). From 1950 to 1960, the largest proportional decline in mortality occurred among infants weighing over 2500 grams. This category comprised only about one quarter of all neonatal deaths in 1960. During 1969 to 1978, by contrast, there was a substantial decrease in mortality for infants weighing between 1000 and 2500 grams.

Fig. 5 →

The contributions of these changes in birth weight-specific mortality to the total absolute decline in neonatal mortality in Massachusetts is calculated in Figure 6. The height of each open bar in this Figure represents the observed neonatal mortality rate among all races in

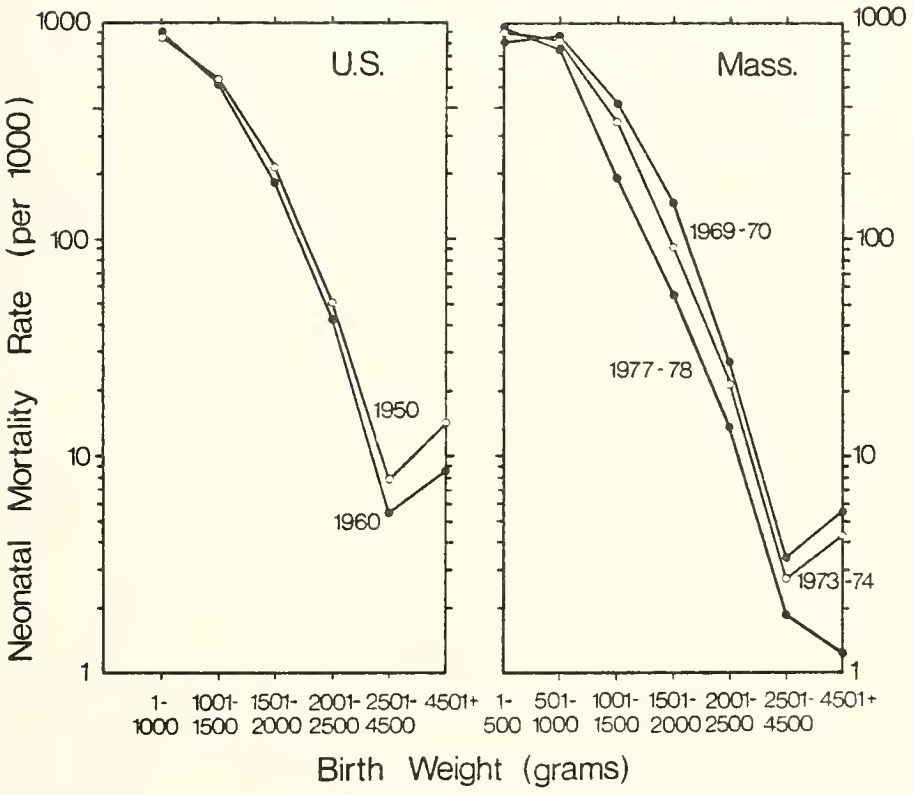

Figure 5. Relation Between Birth Weight and Neonatal Mortality Rate per 1000 Live Births. United States, 1950 to 1960. Massachusetts, 1969 to 1978.

Massachusetts for each year during 1969 to 1978. The height of the combined open and cross-hatched areas for the years 1970 to 1978 represents the birth weight-adjusted neonatal mortality rate. This rate is obtained by applying the birth weight-specific mortality rates for each year to the distribution of birth weights prevailing in 1969. Over 90 percent of the absolute decline in neonatal mortality in Massachusetts, Figure 6 shows, represents an improvement in birth weight-specific mortality.

Fig. 6 →

There is considerable indirect evidence that the trends in Figure 5 are representative of the entire U.S. experience (Pakter and Nelson 1974, p. 859; Kleinman et al. 1978; Chase 1977). The percentage of low birth weight and very low birth weight infants in the U.S. has declined somewhat during the past 15 years. But this change is a fraction of the amount required to explain the decline in mortality if birth weight-specific mortality had remained unchanged (Lee et al. 1980).

A small fraction of the observed improvement in birth weight-specific morality may represent favorable shifts in maternal age and parity (Morris et al. 1975). Although the measurement convention for gestational age was made more precise only after 1968, there is little evidence that the joint distribution of birth weight and gestational age has changed significantly.

There are two types of explanations for this trend. First, we have better medical care during the perinatal period, including labor, delivery, and early neonatal life. This improved perinatal care included advances in neonatal intensive care, transport of high risk mothers to regional centers, treatment of Rh-incompatibility and neonatal jaundice, and improved understanding of neonatal respiratory distress syndrome

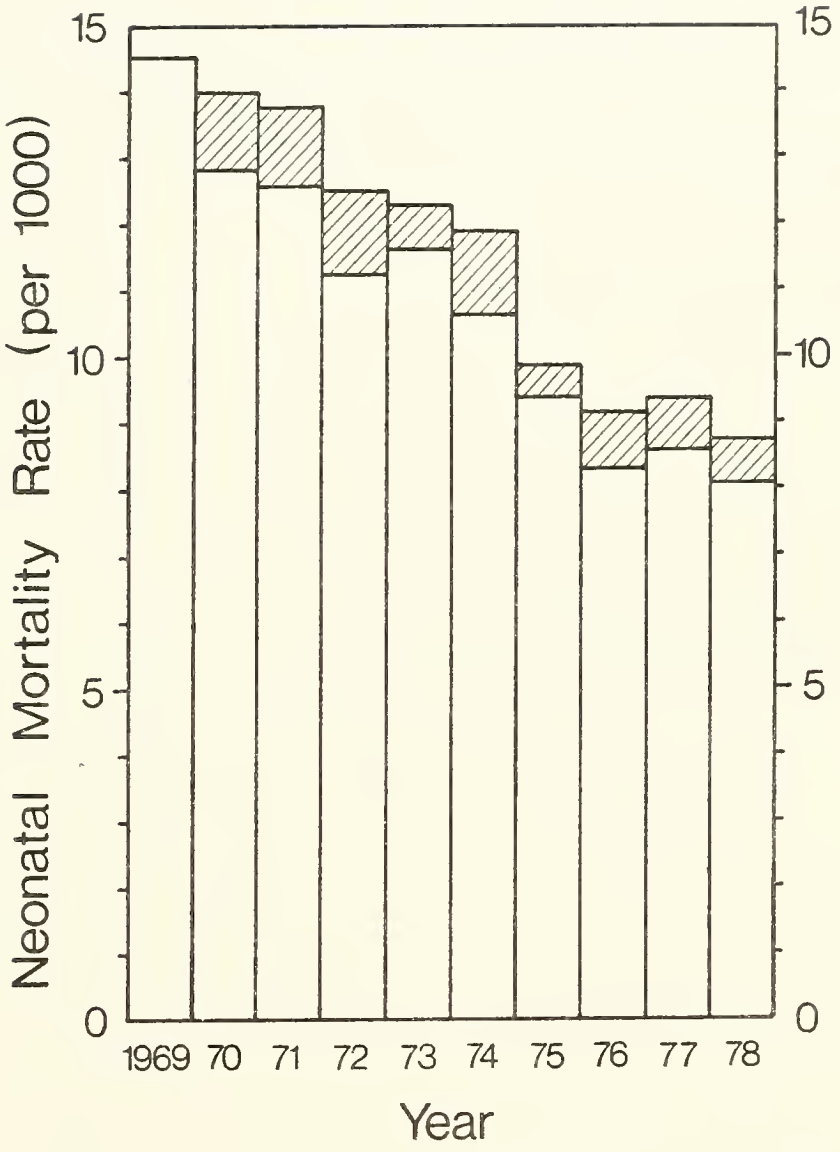

Figure 6. Observed and Birth Weight-Adjusted Neonatal Mortality Rate per 1000 Live Births, Massachusetts, All Races, 1969 to 1978.

(Neonatal mortality rates correspond to open bars. Birth weight-adjusted neonatal mortality rates correspond to summation of open and cross-hatched bars. See text.)

(Borkowf et al. 1979; Kitchen and Campbell 1971; T.R. Harris 1978; Stern 1976; Usher 1977).

Second, infants of a given birth weight have become better specimens in some critical but ill-defined respect. This explanation frequently invokes family planning, contraception, elective abortion, genetic screening, and the elusive fact that babies are now more wanted (U.S. National Center for Health Statistics 1980b; Jacobowitz and Grossman 1980).

It is hardly clear what role, if any, prenatal care has played in this scenario. If the continued growth in the demand for prenatal care had a significant impact on infant survival, then we should expect to observe a relation between prenatal care and birth weight-specific mortality in cross section data. Moreover, if prenatal care in fact prevents early termination of pregnancy or enhances intrauterine growth rates, it is unclear why concomitant changes in the proportion of premature infants were not observed.

Table 5 shows the relation between birth weight and neonatal mortality according to the month of initiation of prenatal care for all races. Except for the Unknown Care category, the neonatal mortality rates for mature infants (over 2500 grams) are indistinguishable. In the low birth weight category, those mothers who initiated care in the first month are at somewhat greater risk. The neonatal mortality rate then increases as care is delayed to the sixth month. But among those initiating care in the third trimester, the mortality rate for low birth weight infants is substantially lower.

We now see the pitfalls of a priori classifications of the amount of care

based upon clinical standards (Kessner et al. 1973; Gortmaker 1979; U.S. National Center for Health Statistics 1978). Aggregation of mothers with no care and third trimester care into a single "inadequate" care category would produce a contradictory relationship between the adequacy of care the birth weight specific mortality.

Table 5 →

The results in Table 5 again confront us with the problem of fetal selection. I have already suggested that the fetal population varies substantially in the rate of pregnancy termination. The sources of this variation, I further suggested, are largely unobserved. Similarly, infants of the same birth weight may vary considerably in their survival characteristics. The sources of this heterogeneity are also largely unobserved. The results in Table 5 suggest that those latent characteristics determining the pregnancy termination rate are correlated with those latent characteristics that determine birth weight-specific mortality.

This explanation is certainly plausible. Congenital anomalies, infection, maternal smoking, or placental insufficiency may shorten gestation and affect survival characteristics. The task of devising a structural model to test this hypothesis, however, is plagued by problems of identification.

Let μ be a latent characteristic that affects the probability of survival. An infant survives, I assume, if $\mu \leq \bar{\mu}$, where $\bar{\mu}$ may depend on various explanatory variables $\underset{\sim}{X}$, including birth weight, as well as gestational age and the amount or timing of prenatal care. For a given cohort of pregnant women, if μ were distributed independently of ε, then the probability of death, conditional upon $(t,v,\underset{\sim}{X})$ is

$$\int_{\bar{\mu}(t,v,\underset{\sim}{X})}^{\infty} f(\mu)d\mu \qquad (18)$$

TABLE 5 -- NEONATAL MORTALITY ACCORDING TO BIRTH WEIGHT AND MONTH OF INITIATION OF PRENATAL CARE. ALL RACES, MASSACHUSETTS, 1975-76.

Month of Initiation of Care	Birth Weight ≤ 2500gm	Birth Weight > 2500gm
1st	115.4 (8.7)	1.8 (0.3)
2nd	99.0 (4.9)	1.9 (0.2)
3rd	90.8 (6.1)	2.0 (0.3)
4th	88.6 (10.4)	1.3 (0.4)
5th	94.0 (14.3)	1.2 (0.5)
6th	113.3 (22.2)	2.0 (1.0)
7th	30.5 (15.0)	2.2 (1.3)
8th or 9th	16.1 (16.0)	3.0 (2.1)
No Care	173.3 (30.9)	1.9 (1.9)
Unknown	143.3 (19.8)	5.3 (1.4)

All rates per 1000 live births. Standard errors in parentheses.

where $f(\mu)$ is the marginal density of μ. When μ has a logistic distribution, for example, equation (18) is the logistic model (Lewit 1977). When μ is normally distributed, (18) is a probit equation.

Suppose, however, that ε and μ were not independent across pregnant women. Then the distribution of μ, like ε, will change during the course of pregnancy. If ε and μ are positively correlated, then as gestation progresses, the proportion of low-ε types, and therefore the proportion of low-μ types, will increase. If $f(\mu|\varepsilon)$ if the conditional density of μ given ε, then the probability of death, conditional upon $(t,v,\underset{\sim}{X})$ is now

$$\int_0^{\infty}\int_{\bar{\mu}(t,v,\underset{\sim}{X})}^{\infty} f(\mu|\varepsilon)f(\varepsilon|t,v,\underset{\sim}{X})d\mu d\varepsilon \qquad (19)$$

where $f(\varepsilon|t,v,\underset{\sim}{X})$ is defined in (7). Hence, if μ and ε are correlated, the single equation probit or logistic model (18) will lead to biased estimates of the effect of prenatal care and other explanatory variables. The structural parameters of $\bar{\mu}(t,v,\underset{\sim}{X})$ cannot be estimated separately from those determining the hazard rate for pregnancy termination and therefore the density $f(\varepsilon|t,v,\underset{\sim}{X})$.

The main problem in applying (19) to our data is that we must impose some restriction on the density $f(\mu|\varepsilon)$ in order to identify these structural parameters. That is, we must decide in advance how the selective process of eliminating high-ε infants affects the distribution of μ. Unfortunately, our inferences about the structural parameters are likely to be very sensitive to the type of restriction imposed.

The results of one such restriction are illustrated in Table 6. Both columns represent estimates of the parameters of $\bar{\mu}(t,v,\underset{\sim}{X})$, which is assumed to be a linear function of gestational age, the duration of care, and other

explanatory variables, including birth weight. Both neonatal and fetal deaths are included.

The left hand column of Table 6 (denoted model III) corresponds to the case where μ and ε are assumed to be independently distributed (equation 19). Specifically, I assume μ has gamma distribution with mean 1 and variance 1/h. If we have independent observations $\{t_p, v_p, \underset{\sim}{X}_p : p = 1,\ldots,P\}$ on surviving infants and $\{t_q, v_q, \underset{\sim}{X}_q : q = 1,\ldots,Q\}$ on perinatal deaths, then the likelihood of these observations, conditional upon the time of initiation of care and the explanatory variables, is $L_T^{II} \times L^{III}$, where

$$L^{III} = \prod_{p=1}^{P} J(\bar{\mu}(t_p, v_p, \underset{\sim}{X}_p)h;h)$$

$$\times \prod_{q=1}^{Q} (1 - J(\bar{\mu}(t_q, v_q, \underset{\sim}{X}_q)h;h) \qquad (20)$$

where $J(x;y) = [\Gamma(y)]^{-1} \int_0^x e^{-z} z^{y-1} dz$ is the incomplete gamma function. Since L^{III} does not involve any of the parameters of L_T^{II}, the Table displays only the parameters of L^{III}. The log likelihood at the bottom of this column is the maximized value of $\log L^{III}$.

The right hand column of Table 6 (denoted Model IV) corresponds to a special case of interdependence between ε and μ (equation 20). Specifically, I assume that ε and μ have a degenerate one-dimensional distribution, that is, they have an identical gamma density with mean 1 and variance 1/h. Again consider the likelihood of the observations $\{t_p, v_p, \underset{\sim}{X}_p : p = 1,\ldots,P\}$ on surviving infants and $\{t_q, v_q, \underset{\sim}{X}_q : q=1,\ldots,Q\}$ on perinatal deaths. In each subset, some mothers will report

TABLE 6 -- MAXIMUM LIKELIHOOD ESTIMATES OF THE EFFECT OF PRENATAL CARE ON THE PROBABILITY OF PERINATAL SURVIVAL. 6,736 BLACK WOMEN, MASSACHUSETTS, 1975-76.

	Model III Parameter Estimates	Model IV Parameter Estimates
Constant Term	-0.254 (0.403)	0.379 (0.229)
Gestational Age (weeks)	0.095 (0.013)	0.001 (0.007)
Duration of Prenatal Care (weeks)	-0.010 (0.009)	0.002 (0.002)
Birth Weight (kilograms)	1.212 (0.295)	0.646 (0.032)
Annual Volume of Deliveries (thousands)	0.066 (0.032)	0.045 (0.018)
Years of Education	-0.014 (0.023)	-0.005 (0.012)
Years of Age over 30	-0.034 (0.027)	-0.013 (0.016)
Years of Age under 20	-0.033 (0.034)	-0.024 (0.023)
Illegitimacy	-0.035 (0.097)	0.062 (0.064)
Prior Perinatal Loss	-0.224 (0.133)	-0.156 (0.077)
Primagravida	0.048 (0.151)	-0.029 (0.083)
Variance of Omitted Regressor μ	0.413 (0.143)	0.176 (0.015)
Log Likelihood	-314.09	-17013.75

Standard errors in parentheses. Estimates of the parameters $\{\alpha, \rho_T, \omega_T, \theta_{Tk}\}$ for Model IV are not displayed.

prenatal care, while others will not. The likelihood of these observations, conditional upon the time of initiation of care and the explanatory variables, is $L_T^{II} \times L^{IV}$, where

$$L^{IV} = \prod_{p=1}^{P} J(\bar{\mu}(t_p, v_p, \underset{\sim}{X}_p)(h+\Lambda_{TV}(t_p, v_p|\underset{\sim}{X}_p));h)$$

$$\times \prod_{q=1}^{Q} [1 - J(\bar{\mu}(t_q, v_q, \underset{\sim}{X}_q)(h+\Lambda_{TV}(t_q, v_q|\underset{\sim}{X}_q));h)] \qquad (21)$$

where Λ_{TV} is replaced by Λ_T in cases where no care was obtained. The partial likelihood L^{IV} involves not only the paremters of $\bar{\mu}$, but also $\{\alpha, \rho_T, \omega_T, \theta_{TK}\}$, which appear in L_T^{II}. Unlike Model III, the parameters of L_T^{II} and L^{IV} must be estimated jointly. Since the estimates of $\{\alpha, \rho_T, \omega_T, \theta_{TK}\}$ were very close to those in Table 3, they are not shown in Table 6. The log likelihood at the bottom of the right hand column is the maximized value of $\log(L_T^{II} \times L_T^{IV})$.

For Model III, with μ independent of ε and therefore no fetal selection, birth weight and gestational age significantly affect the probability of survival. The duration of care, on the other hand, has a paradoxical negative effect. For Model IV, which incorporates fetal selection, the effect of prenatal care is weakly positive, whereas the influence of gestational age appears to be reduced. The latter parameter, however, captures only the direct effect of gestational age on $\bar{\mu}$, that is, the effect of increased duration of pregnancy on the survival rate of a given infant. There is also an indirect effect on Λ_{TV}, that is, the effect of increased duration of pregnancy on the distribution of latent characteristics.

For the parameter estimates in Table 6, we can calculate the elasticity of the perinatal mortality rate with respect to each continuous explanatory variable. For a married, multiparous black mother in her 20s, with 12 years of attained education and no prior perinatal loss, who delivers a 3100 gram infant at 38 weeks in a hospital with 3100 deliveries annually, I obtain the following elasticities:

	Model III		Model IV
Birth Weight	-7.79		-8.72
Gestational Age	-0.75	direct	-0.25
		indirect	-0.32
Duration of Care	0.52		-0.23
Annual Volume of Deliveries	-0.42		-0.61

In both models, birth weight has the dominant effect. Comparison of the direct and indirect elasticities for gestational age suggests that a substantial fraction of the observed effect of duration of pregnancy on mortality represents fetal selection over time. Although prenatal care has a favorable direct effect on mortality in Model IV, its elasticity is small. (The indirect effect of retarding fetal selection was negligible in this example.) Therefore, the main effect of prenatal care on perinatal mortality will still be its influence on birth weight. In this example, the complete absence of care would increase the perinatal mortality by about 20 percent, conditional upon birth weight and gestational age. Using the estimates derived in the previous section, I calculate that the

absence of care, through its effect on the intrauterine growth rate and therefore birth weight, would increase mortality by about 17 percent. Similarly, the absence of care, through its effect on gestational age and therefore on birth weight, would increase mortality by about 32 percent.

Finally, it is noteworthy that the annual volume of deliveries in the hospital of birth has a significant effect on suvival. This finding supports the hypothesis that perinatal medical care, as opposed to prenatal care, has an important influence on birth weight-specific mortality.

SUMMARY AND CONCLUSIONS

This paper has revealed several critical limitations of the use of non-experimental, cross-section data to evaluate the effect of prenatal care on pregnancy outcome. These limitations were illustrated with data on over 140,000 pregnancies in Massachusetts during 1975-76, with specific attention to a sample of approximately 6,800 black women's pregnancies.

In order to study the relation between the timing of prenatal care and the duration of pregnancy, I devised a continuous time stochastic model in which the initiation of care and premature delivery were competing risks. For a specific parametric version of this model, I found that prenatal care prevented pre-term delivery among black pregnant women. The magnitude of this effect was equivalent to an approximate 1 week increase in mean gestational age.

I then introduced an important complication into this model. Unborn infants, I noted, vary considerably in their risks of early termination of pregnancy. These variations in risk set up a powerful selection mechanism in which the least robust infants are progressively eliminated from a cohort of pregnant women as gestation continues. It is not clear how to make valid inferences from the data in the face of this selection phenomenon.

One strategy, which I pursued here, is to specify a particular parametric model of fetal selection, and then to investigate its consequences for the analysis of prenatal care. For the model investigated in this

paper, I showed that failure to take account of fetal selection can lead to biased inferences about the effects of prenatal care and other risk factors. For example, any intervention that retards early termination of pregnancy will necessarily retard the fetal selection mechanism. Unless we incorporate the effect of this intervention on the distribution of fetal characteristics across pregnant women, its influence will appear to be reduced.

I further complicated the analysis by introducing the possibility that mothers could ascertain those fetal characteristics that are not revealed to the investigator. Moreover, these latent characteristics could in turn affect the demand for medical care. I concluded the model under consideration could not be used to make inferences about both the effect of prenatal care on the risk of pre-term delivery and the feedback effect of these risks on the demand for care.

I then considered the relation between prenatal care and birth weight. Previous studies of this empirical relation have not been sensitive to the possible biological or behavioral mechanisms underlying this relationship. To remedy this difficulty, I specified a model in which the timing of care affected the rate of intrauterine growth. For a specific parametric version of this model, I found that the influence of care on birth weight among black women was only weakly positive. For a specific example, the effect of prenatal care on intrauterine growth would result in an average increase in birth weight of about 60 grams. By contrast, the estimated effect of prenatal care on the duration of gestation would

result in an average increase in birth weight of about 110 grams.

The finding of a weak relation between prenatal care and intrauterine growth among black women is consistent with current understanding of the determinants of birth weight. In contrast to premature labor, which can be treated if not detected in advance (Chard 1974), there is no available treatment for placental insufficiency or other forms of intrauterine growth retardation (Spearman et al 1975). Physician advice does not clearly alter material smoking, alcohol use, or nutrition. It is unclear how we can make any stronger inferences about the effect of prenatal care from these non-experimental data when the underlying mechanisms of the effect remains in doubt.

I then turned to the relation between prenatal care and infant death. Previous investigators have found that prenatal care exerts its primary influence on survival through its effect on birth weight. This conclusion, I showed, does not square with an important fact about the recent, renewed decline in infant mortality in the United States. That is, the marked improvement in survival primarily reflects the reduced mortality of low birth weight in infants. Yet there has been relatively little change in the distribution of birth weight or gestational age. If the continued growth in prenatal care had contributed to this trend, then we should expect to observe a relation between prenatal care and birth weight-specific mortality. Moreover, if prenatal care in fact affects birth weight, it is unclear why more marked changes in the rate of prematurity were not observed.

I examined the relation between birth weight-specific mortality and the timing of prenatal care for Massachusetts women during 1975-76. I confirmed the frequently cited, contradictory observation that mothers who initiate care late during pregnancy have infants with considerably lower birth weight-specific mortality. This finding, I suggested, was consistent with the effects of fetal selection. If the fetal population varies in its risks of early termination of pregnancy, then among those mothers remaining pregnant into the third trimester, there will be a smaller proportion of high risk infants. If the risk of premature delivery is correlated with fetal survival characteristics, then the fetal selection mechanism will also affect the distribution of these survival characteristics.

I formulated a specific, restricted model of the relation between fetal selection and fetal survival characteristics. Applying this model to the subsample of black women's pregnancies, I found that prenatal care had a weakly positive effect on birth weight-specific perinatal mortality.

The main difficulty with this model and others described in this paper is that the estimated effects are necessarily contingent on the mechanism of sorting unobserved characteristics. In the case where these latent regressors affect demand or other fetal health characteristics, strong restrictions are required to identify the model. This means that we cannot jointly determine both the effect of medical care on fetal health, the distribution of fetal health characteristics, and the feedback affect of these health characteristics on the demand for care. Although this difficulty applies to a class of hazard models in which the underlying

stochastic processes are not gaussian, a similar predicament has been noted for analogous normal models with discrete endogenous variables (Schmidt 1981).

Many of the illustrative results in this paper were derived from a sample of black women's pregnancies. Since medical intervention may differ in its influence on the health of different races, the specific results cannot be applied generally at this time. No attempt was made in this paper to evaluate the quality, as opposed to the quantity, of care. It is not clear how such an evaluation can proceed in the absence of more refined experimental data bases.

Even if we infer a strong relation between prenatal care and pregnancy outcome, it is hardly obvious that some intervention during gestation was responsible. Mothers with no prenatal care in this analysis had pregnancy outcomes inferior even to those with minimal care. It is possible that the presence of prenatal care is merely indicative of a mother's access to other more critical types of medical intervention during the perinatal period. The finding that black mothers with care have lower rates of preterm delivery suggests that prenatal care may permit women with threatened abortions quick access to other types of treatment.

Although my analysis is confined to the evaluation of one type of medical intervention, many of the difficulties discussed here will apply to large population, non-experimental studies of other types of medical care. But that is a topic for another paper.

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