NBER WORKING PAPER SERIES

THE IMPACT OF PUBLIC HEALTH POLICY: THE CASE OF COMMUNITY HEALTH CENTERS

Fred Goldman

Michael Grossman

Working Paper No. 1020

NATIONAL BUREAU OF ECONOMIC RESEARCH
1050 Massachusetts Avenue
Cambridge MA 02138

November 1982

NBER Working Paper #1020
November 1982

The Impact of Public Health Policy: The Case of Community Health Centers

ABSTRACT

The aim of this paper is to assess the impact of the Community Health Center (CHC) on health levels in the U.S. Using infant mortality as the underlying health indicator, a time series of large counties as the data set, and multivariate regression techniques, we investigate the extent to which the presence of a program in a county affects future mortality. We find that CHCs have negative and statistically significant impacts on white and black infant mortality rates. The centers have larger effects on black infant mortality than on white infant mortality. The reduction in the black infant mortality rate between 1970 and 1978 due to the CHC system amounts to one death per thousand live births or approximately 12 percent of the observed decline. This result is particularly striking in light of the well-known higher infant mortality rate of blacks. A reduction in the excess mortality rate of black babies has been identified as a goal of public health policy for a number of years. Our results suggest that community health centers have the potential to make a substantial contribution to the achievement of this goal.

Fred Goldman
Columbia University School of Public Health
600 West 168th Street
New York, New York 10032
(212)694-3934

Michael Grossman
Department of Economics
City University of New York Graduate School
33 West 42nd Street
New York, New York 10036
(212) 790-4426

National Bureau of Economic Research
269 Mercer Street, 8th Floor
New York, New York 10003
(212)598-3446/3321

This is a study of the impact of a Federally sponsored health initiative on health status. Since 1965 a network of Federally funded community health centers (CHCs) have developed in the United States to deliver comprehensive ambulatory care, both primary and preventive, to poverty populations in medically underserved areas. The program to create and fund these centers, originally termed neighborhood health centers, was started by the Office of Economic Opportunity as part of the War on Poverty. By 1973 overall control of the centers had been shifted to the Bureau of Community Health Services (BCHS), Health Services Administration, U.S. Department of Health and Human Services, and the centers began to be referred to as community health centers. New and smaller variants of the basic CHC model were created in 1975 and 1978 by the introduction of the Rural Health Initiative and the Urban Health Initiative, respectively. Concomitant with these legislative developments, the number of CHCs increased from 51 in 1968 to 104 in 1974 and to approximately 800 in 1980.[1-6]

A preliminary version of this paper was presented at an invited session on Financing Versus Direct Care Delivery for Underserved Populations at the American Public Health Association annual meeting, Los Angeles, California, November 1-5, 1981. We wish to thank Maureen Cropper and Karen Davis for their comments on that version of the paper. We are indebted to Richard Bohrer of the Bureau of Community Health Services, Health Services Administration, DHHS, for helping us to obtain the BCHS Common Reporting Requirements data tape (the BCRR tape) which is the basis for some of the research in this paper. We are also indebted to Robert Nelson of the Division of Monitoring and Analysis, BCHS, for preparing our version of the BCRR tape and to Allison Mann for research assistance. Research for this paper was supported by Grant Number 5 R01 HS 04047 from the National Center for Health Services Research, DHHS, to the National Bureau of Economic Research. This paper has not undergone the review accorded official NBER publications; in particular, it has not been submitted for approval by the Board of Directors.

CHCs were only one component of a broad Federal initiative which was meant to address the "underlying" causes of poverty as well as the poverty population's health needs. The poor were mired in a "vicious cycle of poverty," a popularized shorthand which meant that they lacked employment or were underemployed and were politically disenfranchised. The War on Poverty which was launched under the auspices of the Federal government included, therefore, programs such as the Job Corps and

Community Action Programs as well as those specifically aimed at affecting the health needs of the poor such as CHCs, maternal and infant care programs, children and youth projects, and family planning clinics.

The programs were not, however, neatly compartmentalized in their aims: for example, health programs directed at health goals. Community health centers were "... viewed as shotgun attempts to operate simultaneously on all action variables."[1] In fact, proponents of the CHC program argued that the centers were best used to inject Federal funds into the community, act as a locus of legal and political support, and provide educational training as well as direct employment for community members. Given this, an evaluation of the medical care component of the CHCs and more importantly its impact on the community's health status is not easily accomplished.

The political and economic climate has shifted during the nearly two decades of the CHC program's existence. Whatever the reasons, these are times of fiscal austerity, and the momentum of public initiatives has shifted emphasis from equity to efficiency. Health programs now have improvements in health status as their overriding goal. Other beneficial outcomes such as improved community employment are welcome but unnecessary. Moreover, the very existence of a particular health program is predicated on actual or potential evidence that it is competitive with alternative programs. As Rush[7] recently has written of the WIC program: "If we are to rationally allocate resources, we must know whether programs work."

The aim of this paper is to assess the impact of the CHC program on health levels. That is, we look for an answer to Rush's query: "Does it work?" Using infant mortality as the underlying health indicator, a time series of large counties as the data set, and multivariate regression techniques, we investigate the extent to which the presence of a program in a county affects future mortality. Our estimates control for other determinants of infant mortality such as income and health manpower availability. Although CHCs are not limited in terms of the types of services provided or the age classes of those receiving services, we focus on infant mortality because it is generally accepted that where infant mortality rates are high health levels in all segments of the population are likely to be low.[8] Moreover, CHCs were designed in part to service target populations with high infant mortality rates. In addition, all centers must provide prenatal and post partum care and voluntary family planning services, each of which can have substantial impacts on infant mortality.[9-12] To the extent that health benefits of CHCs are conferred on other members of the population, our findings will understate and thus provide conservative estimates of the impact of the centers on health status.

I. Methods and Data

To estimate the effects of CHCs on health status, alternative versions of infant mortality multiple regression equations or impact functions are fitted. The two basic equations are given by

$$m_{jt} = \alpha_0 + \alpha_1 c_{jt-1} + \alpha_2 c^2_{jt-1} + \alpha_3 x_{jt} + \alpha_4 m_{jt-1} \qquad (1)$$

$$m_{jt} = \beta_0 + \sum_{i=1}^{4} \beta_i pi_{jt-1} + \beta_5 x_{jt} + \beta_6 m_{jt-1} \quad . \qquad (2)$$

In equation (1) m_{jt} is the infant mortality rate in the j^{th} county of the United States in year t, c_{jt-1} is the per capita number of community health centers in the j^{th} county in year t-1, c^2_{jt-1} is the square of the per capita number of centers, x_{jt} is a vector of other determinants of infant mortality such as family income and the per capita number of physicians, and m_{jt-1} is the infant mortality rate in year t-1. In equation (2) the per capita number of CHCs and its square are replaced by a set of four dichotomous variables given by pi_{jt-1}, $i = 1, 2, 3, 4$. Here pi_{jt-1} equals one if the initial service date of the i^{th} CHC in the j^{th} county (the year in which the CHC began to deliver medical care services) was as early as or earlier than year t-1. Four dichotomous variables are employed because very few counties in our sample (described below) had more than four CHCs during the sample period (1969-1978).

Equation (1) constitutes a quadratic specification of the effects of CHCs on infant mortality, while equation (2) constitutes a dichotomous variable specification. Both specifications are employed

because the appropriate way to measure the size of public programs such as CHCs and their impacts on health status remains an open issue. This is particularly true in our case because we have no data on the utilization or expenditures of CHCs. Moreover, the medical care services that they deliver are intended to affect the health of all segments of the poverty population and not just pregnant women and infants. The quadratic specification allows for nonlinear effects from the placement of additional centers in the same county, at least on a per capita basis. That is, the health "returns" to the centers could diminish as more are added if they simply compete for the same population. On the other hand, health "returns" could increase if the greater presence of centers prompts still greater acceptance and use of their services.

The dichotomous variable specification allows for more flexible nonlinearities than the quadratic specification and, in the absence of county-specific time series on the size of the poverty population, permits us to explore alternatives to per capita measurement. Note that the regression coefficients of p1, p2, p3, and p4 give the <u>marginal</u> effects of the placement of CHCs on infant mortality. The coefficient of p1 compares counties with no CHCs to those with one CHC. The coefficient of p2 compares counties with two CHCs to those with one, and the coefficient of p3 compares those with three to those with two. Finally, the coefficient of p4 compares counties with four or more CHCs to those with three. The sum of the four coefficients gives the difference in mortality rates between counties with four or more CHCs and those with none.

The specification of the impact functions recognize explicitly the plausible proposition that CHCs will affect infant mortality with a lag rather than instantaneously. The equation assumes a one-year lag between the year in which a CHC begins to deliver services and its

initial impact on infant mortality. To allow for the possibility that the length of the initial impact lag is more than one year, two- and three-year lag models also were estimated in preliminary research. The results of these models (not shown) did not improve upon those of the basic equations, possibly because of the gross nature of the CHC measures. The members of the x_j vector are not lagged because some variables can affect infant mortality within a relatively short period, particularly those that do not represent new innovations in the medical care delivery system for poverty populations.

Theoretically, the lagged infant mortality rate is an important variable to include in the regression equations because CHCs were designed to service target populations with poor health indicators. Consequently, estimates of their impacts are biased toward zero if the initial level of the mortality rate is omitted from the regression. That is, the presence of a center would be associated with high mortality. The use of the lagged rate as an independent variable also controls for unmeasured determinants of infant mortality that are correlated with the included variables. In addition, the effects of the placement of a CHC in a county will fall over time if there are upward trends in the percentage of the eligible population serviced and the amount of medical care delivered and diminishing returns to care. Simultaneously, mortality differentials between counties with CHCs and those without them will widen over time. Both these effects are captured by including the lagged infant mortality rate in the regressions.*

---

*This point can be demonstrated in a simple fashion by employing equation (2) and by assuming that counties either have one CHC or none. Suppose that county 1 receives a CHC in year t-1 while county 2

(continued on next page)

As the last comment and the footnote suggest, either equation (1) or equation (2) is a distributed-lag model because the effects of CHCs on infant mortality are spread over a number of periods. For instance,

---

* (continued)

does not. In addition suppose that $m_{1t-1} = m_{1t-2} = m_{2t-1} = m_{2t-2}$. Finally suppose that x_{1t} has no trend and $x_{1t} = x_{2t}$. Then

$$m_{1t} - m_{1t-1} = \beta_1$$

$$m_{1t+1} - m_{1t} = \beta_6 \beta_1$$

$$m_{1t+i} - m_{1t+i-1} = \beta_6^i \beta_1 \quad ,$$

where β_6 is the regression coefficient of the lagged infant mortality rate. Moreover,

$$m_{1t} - m_{2t} = \beta_1$$

$$m_{1t+1} - m_{2t+1} = \beta_1 (1 + \beta_6)$$

$$m_{1t+i} - m_{2t+i} = \beta_1 (1 + \beta_6 + \beta_6^2 + \ldots + \beta_6^i) \quad .$$

If β_6 is positive and smaller than one, then in the long run (as n goes to infinity)

$$m_{1t+n} - m_{1t+n-1} = 0$$

$$m_{1t+n} - m_{2t+n} = \beta_1/(1 - \beta_6) \quad .$$

Clearly, these equations imply that rates of change in infant mortality in county one decline over time (in absolute value since β_6 is negative), while mortality differentials between county one and county two grow over time.

if the square term (c^2_{jt-1}) in equation (1) is ignored, a one-unit increase in c_{jt-1} lowers m_{jt} by α_1. Since m_{jt+1} depends on m_{jt}, m_{jt+1} falls by $\alpha_1\alpha_4$. Similarly, m_{jt+2} falls by $\alpha_1\alpha_4^2$, and m_{jt+k} falls by $\alpha_1\alpha_4^k$. The sum of these distributed lag effects (assuming $\alpha_4 < 1$) is $\alpha_1/(1 - \alpha_4)$. The coefficient α_1 gives the short-run or immediate impact of CHCs on infant mortality, while $\alpha_1/(1 - \alpha_4)$ gives the long-run impact. The latter term shows the difference between the infant mortality rate in a year in the distant future and the rate in the year prior to the increase in the CHC variable. Since the lagged infant mortality rate is held constant in regressions (1) and (2), they constitute a short-run model of the impact of CHCs.

Note that the basic model may be viewed as the result of applying a Koyck[13] transformation to an infinite-lag distributed-lag model with geometrically declining weights.* Because the assumptions that underlie the Koyck lag structure are somewhat restrictive, an ad hoc distributed-lag model also is estimated. The quadratic version of this model is

$$m_{jt} = \gamma_0 + \gamma_1 c_{jt-1} + \gamma_2 c^2_{jt-1} + \gamma_3 x_{jt} + \gamma_4 m_{j0} \quad , \qquad (3)$$

---

*Based on the assumption in the previous footnote, the underlying model is

$$m_{jt} = \lambda_0 + \lambda_1 pl_{jt-1} + \lambda_2 pl_{jt-2} + \lambda_3 pl_{jt-3} + \cdots \quad ,$$

with $\lambda_k = \lambda_1 \rho^{k-1}$, $k = 1, 2, \ldots$ and $0 < \rho < 1$. For a detailed discussion of distributed-lag models, see Kmenta.[14]

while the dichotomous variable version is

$$m_{jt} = \Delta_0 + \sum_{i=1}^{4} \Delta_i pi_{jt-1} + \Delta_5 x_{jt} + \Delta_6 m_{j0} \quad . \tag{4}$$

The county-specific infant mortality rate (m_{j0}) in the initial year of the time series (1969 in our sample) is included in these regressions to control for the placement of CHCs in counties with poor health indicators.* In principal such regressors as, for example, c_{jt-2}, c_{jt-3} etcetera should be included in equation (3) to make it an ad hoc distributed-lag model. These variables are omitted because the appropriate lag structure is not known and because multicollinearity among members of the distributed-lag vector of CHC variables is high. Given this high degree of correlation, we interpret the coefficients of the CHC variables in equations (3) and (4) as long-run or cumulative effects and term these two equations a cumulative model.**

---

*Note that, although the time series on infant mortality begins in 1969, the initial service dates of CHCs can be as early as 1965.

**Suppose that the underlying ad hoc distributed-lag model is

$$m_{jt} = \phi_0 + \phi_1 c_{jt+1} + \phi_2 c_{jt-2} + \phi_3 c_{jt-3} \quad .$$

The short-run effect of CHCs on infant mortality is ϕ_1, while the long-run effect is $\phi_1 + \phi_2 + \phi_3$. Total differentiation of the equation with respect to c_{jt-1} yields

$$(dm_{jt}/dc_{jt-1}) = \phi_1 + \phi_2 (dc_{jt-2}/dc_{jt-1}) + \phi_3 (dc_{jt-3}/dc_{jt-1}).$$

In addition to relaxing the assumptions that underlie the Koyck lag structure, estimation of the cumulative model is desirable because it is possible that the short- and long-run effects of CHCs are understated in the Koyck model. This occurs if the contemporaneous correlation between a CHC measure and the infant mortality rate [for example, the correlation between c_{jt-1} and m_{jt-1} in equation (1)] becomes negative after some point in time. Initially, this correlation will be positive, reflecting the placement of CHCs in counties with above average infant mortality rates. Ultimately, however, it may become negative if c_{jt-2} has a substantial impact on m_{jt-1} and if the correlation between c_{jt-2} and c_{jt-1} is positive and fairly large. Under these circumstances, part of the impact of c_{jt-1} on m_{jt} may be captured by m_{jt-1}. Therefore, we view long-run effects computed from the Koyck model as lower-bound estimates.

Other members of the Federal health delivery system for poverty populations, such as maternal and infant care projects, children and

---

** (continued)

If $(dc_{jt-2}/dc_{jt-1}) = 1$ and $(dc_{jt-3}/dc_{jt-1}) = 1$, a regression of m_{jt} on c_{jt-1} with c_{jt-2} and c_{jt-3} omitted provides an estimate of the long-run effect. To the extent that (dc_{jt-2}/dc_{jt-1}) and (dc_{jt-3}/dc_{jt-1}) are smaller than one, the long-run effect is <u>underestimated</u>.

youth projects, and family planning centers, are omitted from the regressions. CHCs may be substitutes or complements for these components of the delivery system. They are substitutes if the availability of another program lowers the utilization of CHCs and complements if the reverse holds. For example, if a CHC and a family planning clinic are located in the same county, utilization of family planning services at the CHC may be smaller than if there were no family planning clinic. On the other hand, the existence of a CHC and a maternal and infant care project in the same proximity may encourage utilization of both via referrals.

Strictly speaking, if different types of projects tend to be located in the same areas, the omission of the other projects understates the impacts of CHCs on infant mortality if these projects are substitutes for CHCs and overstates the impacts of CHCs if they are complements. The reverse conditions hold if the presence of a CHC in a county is negatively related to the presence of other projects. But, as the preceding examples illustrate, the direction of the bias is not certain because locational patterns are not clear and because CHCs may be substitutes for some projects and complements for others. Moreover, the inclusion of the lagged mortality rate controls in part for the effects of other projects. Finally, it might be difficult to disentangle the effects of specific projects because of multicollinearity.

During our sample period, the U.S. infant mortality rate fell at an annually compounded rate of approximately 5 percent per year. The lagged infant mortality rate obviously is negatively related to time. Therefore, in the short-run specification, the former variable controls in part for the effects of time, and a time trend is omitted from the regression models. This is because the inclusion of a time trend creates serious problems of multicollinearity, especially since the data span a short nine year period. Since m_{j0} replaces m_{jt-1} as a regressor in the cumulative model, unmeasured trend effects are not held constant in that model. This factor alone biases upward in absolute value the CHC effects in the cumulative model. Note, however, from the previous footnote that the omission of certain members of the distributed lag vector of CHC variables biases downward the cumulative effect. Therefore, biases due to the omission of time are mitigated. Regression results without time are presented in the next section, and we indicate how these results are altered when time is included.*

---

*A potential remedy to problems associated with time trends and serial correlation is to take first differences of all variables. We did not employ this approach because it assumes that the regression residuals exhibit first-order serial correlation with a serial correlation coefficient equal to one. Preliminary results led to a rejection of this key assumption. The estimation of the precise nature of a serial correlation process, particularly in models with lagged dependent variables, is difficult, frequently has a high standard error, and is sensitive to alternative specifications. Consequently, we did not pursue this approach.

The basic data set employed in this paper is the Area Resource File (ARF). The ARF is a county-based data service, prepared by Applied Management Sciences, Inc., for the Bureau of Health Professions, Health Resources Administration, U.S. Department of Health and Human Services. It incorporates information from a variety of sources for 3,077 counties in the United States. These counties also can be aggregated into larger geographic areas such as county groups, Standard Metropolitan Statistical Areas, and states. Deaths by age, race, and sex for the years 1969 through 1978 are obtained from the National Center for Health Statistics (NCHS) Mortality Tape.* Births by race for those years are obtained from the NCHS Natality Tapes. County-specific time series pertaining to health manpower and facilities come from the American Medical Association, the American Hospital Association, and other sources. Demographic and socioeconomic characteristics for 1970 are taken from the 1970 Census of Population and from other sources for years before and after 1970. We have added information on the location and initial service dates of CHCs to the ARF. This information is derived from the Bureau of Community Health Services Common Reporting Requirements data tape (the BCRR tape).

---

*Since we use race- and age-specific infant mortality data (see below), the death data are based on the August 1978 and December 1980 versions of the ARF. Deaths for 1978 were provided to us directly by Applied Management Sciences.

In the regression estimates, a distinction is drawn between the two components of infant mortality: neonatal mortality and postneonatal mortality. Neonatal mortality refers to deaths of infants within the first 27 days of life. Postneonatal mortality refers to deaths of infants between the ages of 28 and 364 days. Neonatal deaths are usually caused by congenital anormalies, prematurity, and complications of delivery; while postneonatal deaths are usually caused by infectious diseases and accidents. Since the causes of the two types of infant deaths are dissimilar, CHCs may have different effects on each. This possibility is examined by using the neonatal, postneonatal, and total infant mortality rates as alternative dependent variables. In addition separate regressions are fitted for white infant mortality and for black infant mortality as well as regressions for infant mortality of all races.* This is because black infant mortality rates are much higher than white rates.

Counties rather than states or SMSAs are used as the units of observation. As indicated by their name, CHCs are intended to serve the residents of particular communities, and counties are the smallest geographic areas on the ARF. On the other hand, SMSAs and states are very large and sometimes heterogenous. Income, medical resources, and other relevant variables may vary greatly within an SMSA or a state. Since counties are much more homogeneous, these problems are reduced in

---

*In the non-race-specific regressions, the dependent variable pertains to whites, blacks, and other races. All infant mortality rates are expressed as deaths per thousand live births.

our research. A weakness with the use of counties is that the small size of some of these areas may mean that people may receive medical care outside the county. Moreover, the small number of births in certain counties may increase the importance of random movements or "noise" in the determination of regression coefficients.

We reduce these problems with county data by including in the regressions only counties with a population of at least 50,000 persons in 1970. A county must also have at least 5,000 blacks for inclusion in the black impact regressions. There are 678 counties in the white regressions and 358 counties in the black regressions.* In addition to selecting large counties, we attenuate random elements by estimating weighted regressions, where the set of weights is the square root of the total or race-specific number of births.

There are nine alternative dependent variables in the regression equations: the total, white, and black infant mortality rates; the total, white, and black neonatal mortality rates; and the total, white, and black postneonatal mortality rates. The lagged rate on the right-hand side of each equation corresponds to the rate that is being used as the dependent variable. The first observation on the dependent variable pertains to 1970 and the last observation pertains to 1978. This is because the age- and race-specific infant death series begins in 1969

---

*One county with a population greater than 50,000 in 1970 was eliminated from the sample because it was the only such county characterized as an isolated rural county with no incorporated place with a population of at least 2,500 persons in 1970.

and ends in 1978. Thus, the regression equations are fitted to a time series of the 678 largest counties (358 in the case of the black regressions) of the U.S. for the period 1970-1978.

The roles of the CHC variables, the lagged infant mortality rate, and the initial (1969) infant mortality rate in the regressions have been discussed in detail. In the non-race-specific regressions, the percentage of nonwhite births controls for the higher death rates of nonwhite babies. Real median family income and office-based physicians in private practice per thousand population have been stressed conceptually and empirically as basic determinants of infant mortality in previous research[8,15,16] and are included in the regressions. Race-specific family income is employed as a regressor in race-specific regressions. In all cases money family income is divided by the Consumer Price Index to obtain real family income.* Only the regression coefficients of the CHC variables are presented in the next section. It is important to realize,

---

*Race-specific median family income in 1969 by county is available from the 1970 Census of Population. Complete time series are obtained by assuming that the year-to-year percentage change in race-specific median family income equals the year-to-year percentage change in per capita income for all races. The same procedure is employed to compute a time series of median family income for all races. In the mortality regressions for all races, it makes little difference whether income is given by median family income or per capita income since the two variables are highly correlated. We select median family income to be consistent with the race-specific regressions.

however, that these are net or partial regression coefficients in the sense that the impacts of the variables just mentioned are held constant. (The full regression estimates are contained in the Appendix.)

II. Results

Table 1 contains regression coefficients of the community health center measures (CHC, the number of centers per thousand population, and CHCSQ, the square of the number of centers per thousand population) and related statistics in the two versions of the quadratic specification. The first version is the Koyck or short-run model and is obtained by including the relevant lagged infant mortality rate as a regressor. The long-run effects in that model obtained by dividing the CHC coefficients by one minus the coefficient of the lagged infant mortality rate, are shown in brackets. The second version of the quadratic specification is the cumulative model and is obtained by replacing the lagged infant mortality rate with the mortality rate in 1969. For reasons discussed in Section I, the CHC coefficients in the cumulative model are alternative estimates of long-run effects.

The results in Table 1 indicate that community health centers in general have negative and statistically significant impacts on the alternative infant mortality rates studied. The coefficient of CHC is negative and significant in sixteen of eighteen cases. The exceptions pertain to the two white postneonatal mortality regressions. The coefficient of the square term always is positive, which implies that there are diminishing returns to the placement of centers, at least on a per capita basis. It should be noted that the significance of the CHC coefficients is not an artifact of the nonlinear specification. When the square term is omitted, the sixteen negative CHC coefficients retain their signs and are significant except in the short-run, all races postneonatal mortality regression. Moreover, the value

of CHC that "minimizes" the relevant infant mortality rate (that is, the value of CHC beyond which the infant mortality rate begins to rise) falls outside the range of all observations.

The CHC regression coefficients associated with the dummy variable specification are presented in Table 2. Recall that the four dichotomous variables denote whether the initial service date of the first (P1), second (P2), third (P3), or fourth (P4) CHC in a given county was as early as or earlier than year t-1. The results in Table 2 are less clearcut than those in Table 1. Fifty-seven of the seventy-two regression coefficients (or 79 percent of the coefficients) are negative, but only twenty-three of the negative effects are significant. The hypothesis that no member of the set of four dichotomous variables has a nonzero effect is accepted in six of eighteen cases at the 5 percent level of significance and in eight of eighteen cases at the 1 percent level of significance (see the F-ratios in Table 2). The marginal effects of the placement of additional CHCs in the same county do not demonstrate a consistent pattern. For example, in the short-run infant mortality regression for all races, the mortality differential between counties with one CHC and counties with two CHCs (.3 deaths per thousand live births) exceeds the differential between counties with one CHC and those with none (.1 deaths per thousand live births). In addition, the excess mortality of counties with three CHCs compared to those with two (less than .1 deaths per thousand live births) is smaller than the excess mortality of counties with three CHCs compared to those with four (.2 deaths per thousand live births).

In spite of the above points, the findings in the two tables are consistent with each other. Both sets of results contain the implication that the growth in CHCs during the 1970s has contributed to the decline in infant mortality during that period. Both sets are shown because of the exploratory nature of this research and the crude measurement of the CHC variables. The negative effects that emerge from the quadratic and dichotomous variable specifications and from the short-run and cumulative versions of each specification strengthen our confidence in the basic findings. Since the coefficients of determination in the quadratic specification (not shown) are at least as large as (and frequently larger than) the corresponding coefficients in the dichotomous variable specification, the former is preferred to the latter. This suggests that it is important to take account of the size of the clientele of CHCs. Surely the number of users is positively related to the size of the population.

So far we have said nothing about the magnitudes of the negative impacts of CHCs on infant mortality. To address this issue, we examine the net or partial contribution of the centers to overall reductions in the nine infant mortality rates between 1970 and 1978 in Table 3. Specifically, we apply the regression coefficients of the preferred quadratic specification to trends in the CHC measures in period under consideration.* To illuminate the nature of the

---

*Based on the notation in equation (1), the short-run contribution in Table 3 is the absolute value of $\alpha_1(\bar{c}_{77} - \bar{c}_{69}) + \alpha_2(\bar{c}^2_{77} - \bar{c}^2_{69})$,

(continued on next page)

computations, note that the total infant mortality rate of all races fell by 5.9 deaths per thousand live births, from 19.6 in 1970 to 13.7 in 1978 (see Table 3, row 1, columns 1-3).** Based on the Koyck model, the short-run contribution of the CHC system to this reduction amounts to .1 deaths per thousand live births or 2 percent of the decline. The preceding computation ignores, for example, the reduction

---

*(continued)

where $\bar{c}_{69}$ is the mean of the per capita number of CHCs in 1969, and $\bar{c}_{77}$ is the mean of the per capita number of centers in 1977. These means pertain to the sample of 678 counties in the white and non-race specific regressions and to the sample of 358 counties in the black regressions. They are weighted by the year-specific total or race-specific number of births. Means for 1969 and 1977 are employed because the CHC variables are lagged one year in the regressions. The long-run contribution in Table 2 is the absolute value of $[\alpha_1(\bar{c}_{77} - \bar{c}_{69}) + \alpha_2(\bar{c}^2_{77} - \bar{c}^2_{69})]/(1-\alpha_4)$. Based on the notation in equation (3), the cumulative contribution is the absolute value of $\gamma_1(\bar{c}_{77} - \bar{c}_{69}) + \gamma_2(\bar{c}^2_{77} - \bar{c}^2_{69})$.

**The infant mortality rates in the first two columns of Table 3 pertain to the sample counties rather than to all counties in the U.S. They are weighted sample means, where the set of weights is the total or race-specific number of births. These rates are almost identical to rates for the U.S. as a whole.

in infant mortality in year t due to an increase in the per capita number of CHCs in year t-2. When these effects are incorporated, the long-run contribution is obtained. It amounts to a drop of .3 deaths per thousand live births or 5 percent of the observed decline. An alternative estimate of the long-run effect is provided by the cumulative model. The absolute and percentage variants of this estimate are contained in the last two columns of Table 3. In the case of the total infant mortality rate of all races, the long-run and cumulative contributions are almost identical.

The twenty-four computations of declines in various infant mortality rates due to the CHC system range from a low of less than .1 deaths per thousand live births (the short-run contribution to the decline in the non-race-specific postneonatal mortality rate) to a high of 1.2 deaths per thousand live births (the cumulative contribution to the decline in the black total infant mortality rate). When these effects are expressed as percentages of the observed reductions, they range from a low of 2 percent in several instances to a high of 18 percent in the instance of the long-run black postneonatal contribution. In general the cumulative effects are slightly larger than the corresponding long-run effects. The close agreement between these two alternative sets of estimates underscores the robustness of the computations. According to the figures in Table 3, CHCs have larger absolute effects on neonatal mortality than on postneonatal mortality. When, however, the contributions are expressed as percentages of the observed declines, the postneonatal impacts exceed the neonatal impacts. In part this finding reflects the fact that the postneonatal mortality rate is much smaller than the neonatal mortality rate.

The most notable finding in Table 3 is that CHCs have larger impacts on black infant mortality (total or age-specific) than on white infant mortality. This result emerges whether the effects are expressed as absolute contributions to observed reductions or as contributions as percentages of the corresponding reductions. This result is particularly striking in light of the well-known higher infant mortality rate of blacks. A reduction in the excess mortality rate of black babies has been identified as a goal of public health policy for a number of years. Our results suggest that community health centers have the potential to make a substantial contribution to the achievement of this goal. In particular, the long-run reduction in the black total infant death rate between 1970 and 1978 due to the CHC system amounts to one death per thousand live birth or approximately 12 percent of the observed decline. In appreciating the significance of such a decline, it is important to keep in mind that the centers were designed to affect the health of all segments of the poverty population and not just infants and pregnant women. Hence, there are many competing demands on their scarce resources: the goal of improvements in the delivery of prenatal care, perinatal care, and care for infants under the age of one competes in the allocation of CHC resources with the goal of improvements in the delivery of medical care services to children beyond the age of one and adults.

Table 4 contains alternative summary measures to those in Table 3 of the effects of community health centers on infant mortality. Specifically, cumulative mortality differentials between counties with

four or more centers and those with no centers are shown. Each race- and age-specific differential in the table is the sum of the regression coefficients of the four dichotomous variables in the relevant cumulative model. This model is used in the computations because it embodies more flexible assumptions about the nature of the lag structure than the Koyck model. The figures in the table suggest a substantial payoff to counties that have pursued an aggressive policy of investment in CHCs. For whites the total infant mortality rate in such counties (those with four or more CHCs) is smaller than the rate in counties with no centers by 1.5 deaths per thousand live births. The comparable figure for blacks is a whopping 2.9 deaths per thousand live births. Put differently, counties that have invested substantial resources in CHCs appear to have reduced both their white and black infant mortality rates by 10 percent when compared to counties that have made no investments in CHCs.

Still, some caution must be exercised in interpreting these results. We have stressed the difficulties with an empirical evaluation of as general a concept as the CHC and the techniques involved in such an evaluation. For example, the regression coefficients of the CHC variables are reduced in magnitude and lose their statistical significance when a time trend is included in the regressions. Given the short nine-year time span of our sample, this finding is not surprising. We wish to emphasize, however, that pure time effects in regression models are measures of ignorance. In this context it is important to note that our model does at least as good a job in explaining variations in infant mortality rates as a model in which eight time dummy variables alone are used as

regressors. Frequently, our model outperforms the pure time model based on the relevant coefficients of determination. Clearly, more research on the effects of CHCs on infant mortality is appropriate. Nevertheless, given the variety of evidence that we have presented and the robustness of this evidence to alternative specifications, we feel that it is reasonable to conclude that there is significant evidence that the centers have contributed to reductions in infant mortality.

III. Discussion

Since 1964 the U.S. infant mortality rate has declined at an annually compounded rate of more than 4 percent per year. This is an extremely rapid rate of decline compared to the decline of less than 1 percent per year during the prior decade, 1955 to 1964. Our study is the latest of several to attribute important roles in the dramatic fall in the infant mortality rate during the past two decades to a number of public policies and programs. Using a very different methodology from the one employed in this study, Grossman and Jacobowitz[9] report that the increase in the legal abortion rate was the single most important factor in reductions in white and black neonatal mortality rates between 1964 and 1977. In turn, the growth in the legal abortion rate occurred because of the reform of restrictive state abortion laws starting in 1967, the ruling by the Supreme Court in 1973 that restrictive state laws were unconstitutional, and the Federal and state funding of abortions for poor women under Medicaid.* Grossman and Jacobowitz also find that the growth in the use of organized family planning services by low-income women due to the expansion in Federal subsidies to clinics that deliver these services produced declines in race-specific neonatal mortality rates. Moreover, they indicate that

---

*Under the Hyde Amendment, which was in effect from June 1977 until February 1980 and continually since July 1980, Federal funding of abortions under Medicaid is banned except in cases when the woman's life is in danger. Note that, although we exclude the legal abortion rate from our regressions, we control for its effects by including the lagged infant mortality rate.

the delivery of prenatal and perinatal health services to poor pregnant women by maternal and infant care projects and the financing of these services under Medicaid caused reductions in black neonatal mortality. Results in studies by Hadley[17] and by Chachere and Verona* support Grossman and Jacobowitz's conclusions with respect to abortion and to Medicaid financing of prenatal and perinatal care. Taken together, these three studies and our study indicate that public financing and delivery of medical care services (defined to include abortion services and family planning services) can have substantial impacts on the health of the poor.

A full cost-benefit or cost-effectiveness analysis of the community health center program vis-a-vis other programs to reduce infant mortality, such as the construction and subsidization of neonatal intensive care units, is beyond the scope of this paper. Our results suggest, however, that the CHC system may have a very favorable cost-benefit ratio. The impact of the program on infant mortality, especially black infant mortality, appears to be substantial. Moreover, the cost of the program probably is smaller than the cost of constructing and maintaining sophisticated neonatal intensive care units if, for example, these were competing programs. In addition, the CHC system's benefits are understated in our research because the centers deliver services to all age-classes of low-income people and can affect health measures besides infant mortality.

---

*Chachere B, Verona D: Medicaid Programs and the Health Status of SMSA Residents: An Econometric Analysis. Washington, D.C.: Office of Research, Demonstrations, and Statistics, Health Care Financing Administration, DHHS, Working Paper No. OR-12, 1980.

Clearly, our research findings are relevant with regard to the current policy debate on the effectiveness, efficiency, and ultimate fate of public health policy in general and the CHC system in particular. The growth of the CHC system has been curtailed sharply in real terms by the Reagan Administration's budget cutbacks. Although the centers were exempted from the Administration's block grant program in the fiscal 1982 budget, starting in 1983 individual states have the option to take over the CHC program or leave it in Federal hands. In the fiscal 1983 budget, the centers are combined with family planning clinics, migrant health centers, and black lung clinics into a single block grant. Moreover, a provision under which states that choose to take control of the program must match a portion of the Federal support is eliminated. If the CHCs are relatively inefficient producers of ambulatory medical care services and have little or no impact on health levels, the policies of the Reagan Administration have some merit. On the other hand, if the centers are relatively efficient, the merit of these policies can be questioned.

Our results seriously challenge the conventional wisdom that public sector production is less efficient than private sector production. We have shown in this study that the CHC system has played an important role in recent reductions in infant mortality, especially black infant mortality. In addition, in another study,[18] we have found no evidence that allocative inefficiency (increases in production costs due to the use of inappropriate combinations of inputs) is more

widespread among CHCs than among private sector physicians. Although there are statistically significant departures from cost-minimizing behavior in the CHC system, their impacts on the cost of ambulatory medical care are modest. To be specific, the cost saving associated with the use of a more appropriate combination of physicians, physician aids, and medical support personnel amounts to 6 percent of the CHC system-wide total cost of ambulatory medical care in 1980. Thus, we conclude from our two studies that the CHC program is an effective vehicle to achieve the goal of improvements in the health of the poor. Substantial improvements appear to have been accomplished, and the costs in terms of departures from the optimal utilization of inputs appear to be small. Our results suggest that infant mortality rates, especially black rates, may fall more slowly than otherwise and may even rise if the CHC program is subjected to substantial budget cutbacks. In general this study and the related studies discussed in this section raise serious questions with regard to current public health policy.

References

1. Lave JR, Leinhardt S: The delivery of ambulatory care to the poor: a literature review. Management Science 1972; 19:78-99.

2. Hollister RM, Kramer BM, Bellin SS: Neighborhood Health Centers. Lexington, Mass: Lexington Books, 1974.

3. Reynolds RA: Improving access to health care among the poor: the neighborhood health center experience. Milbank Mem Fund Q 1976; 54:47-82.

4. Seacat MS: Neighborhood health centers: a decade of experience. J Community Health 1977; 3:156-170.

5. Davis K, Schoen C: Health and the War on Poverty. Washington, DC: The Brookings Institution, 1978.

6. Roemer MI: Ambulatory Health Services in America: Past, Present, and Future. Rockville, Md: Aspen Systems Corporation, 1981.

7. Rush D: Is WIC worthwhile? Am J Public Health 1982; 72:1101-1102.

8. Fuchs VR: Some economic aspects of mortality in developed countries. In: Perlman M (ed): The Economics of Health and Medical Care. London: MacMillan, 1974, 174-193.

9. Grossman, M. Jacobowitz S: Variations in infant mortality rates among counties of the United States: the roles of public policies and programs. Demography 1981; 18:695-713.

10. Harris JE: Prenatal medical care and infant mortality. In: Fuchs, VR (ed): Economic Aspects of Health. Chicago: University of Chicago Press for the National Bureau of Economic Research, 1982, 15-52.

11. Rosenzweig MR, Schultz TP: The behavior of mothers as inputs to child health: the determinants of birth weight, gestation, and rate of fetal growth. In Fuchs VR (ed): Economic Aspects of Health. Chicago: University of Chicago Press for the National Bureau of Economic Research, 1982, 53-92.

12. Lewit, EM: The demand for prenatal care and the production of surviving infants. In: Sirageldin I, Salkever D, Sorkin A (eds): Research in Human Capital and Development, Vol III. Greenwich, Conn: JAI Press, 1982.

13. Koyck LM: Distributed Lags and Investment Analysis. Amsterdam: North-Holland Publishing Company, 1954.

14. Kmenta J: Elements of Econometrics. New York: MacMillan Publishing Company, 1971.

15. Brooks, CH: Infant mortality in SMSAs before Medicaid: test of a causal model. Health Services Research 1978; 13:3-15.

16. Gortmaker SL: Poverty and infant mortality in the United States. Am Sociological Review 1979; 44:280-297.

17. Hadley J: More Medical Care, Better Health? Washington, DC: the Urban Institute Press, 1982.

18. Goldman F, Grossman M: The production and cost of ambulatory medical care in community health centers. In Scheffler RM, Rossiter LF (eds): Advances in Health Economics and Health Services Research, Vol IV. Greenwich, Conn: JAI Press, 1983.

TABLE 1
Regression Coefficients of Community Health Center Variables, Quadratic Specification*

Race-Mortality Rate	Short-Run Model		Cumulative Model	
	CHC	CHCSQ	CHC	CHCSQ
All Races (n = 6,102)				
Neonatal	-162.350 (-4.91) [-371.510]	6,592.463 (2.69) [15,085.727]	-396.847 (-10.30)	16,297.950 (5.66)
Postneonatal	-33.154 (-1.97) [-46.369]	2,525.641 (2.01) [3,532.365]	-47.112 (-2.78)	2,598.068 (2.05)
Infant	-181.653 (-4.76) [-418.555]	8,338.278 (2.94) [19,212.621]	-441.259 (-9.95)	18,648.650 (5.63)
Whites (n = 6,102)				
Neonatal	-140.375 (-4.17) [-269.952]	5,049.036 (2.09) [9,709.685]	-300.882 (-8.01)	10,828.710 (4.00)
Postneonatal	2.923 (.17) [3.241]	663.664 (.55) [735.769]	-11.261 (.67)	1,251.544 (1.03)
Infant	-145.072 (-3.79) [-272.180]	5,898.554 (2.14) [11,066.705]	-316.592 (-7.48)	12,256.170 (4.02)
Blacks (n = 3,222)				
Neonatal	-309.369 (-3.29) [-504.680]	8,797.011 (.99) [14,350.752]	-604.551 (-6.01)	24,783.360 (2.58)
Postneonatal	-163.686 (-2.97) [-207.460]	10,582.840 (2.02) [13,412.978]	-167.951 (-3.00)	7,664.397 (1.44)
Infant	-414.284 (-3.66) [-697.448]	16,938.980 (1.58) [28,516.801]	-760.146 (-6.24)	31,359.350 (2.70)

*t-ratios in parentheses. The critical t-ratio at the 5 percent level is 1.64 for a one-tailed test. Long-run effects associated with short-run model in brackets. The F-ratio associated with each regression (not shown) is statistically significant at the 1 percent level of significance.

TABLE 2

Regression Coefficients of Community Health Center Variables, Dichotomous Variable Specification*

Race-Mortality Rate	Short-Run Model P1	P2	P3	P4	F-ratio**	Cumulative Model P1	P2	P3	P4	F-Ratio**
All Races (n = 6,102)										
Neonatal	-.174 (-1.95) [-.400]	-.296 (-1.99) [-.680]	-.003 (-.02) [-.007]	-.189 (-.85) [-.434]	5.94	-.426 (-4.07)	-.779 (-4.47)	-.160 (-.70)	-.794 (-3.04)	38.42
Postneonatal	.096 (2.10) [.134]	-.052 (-.69) [-.073]	.124 (1.24) [.173]	-.198 (-1.72) [-.276]	1.96	.094 (2.04)	-.115 (-1.50)	.192 (1.90)	-.391 (-3.40)	4.15
Infant	-.120 (-1.16) [-.278]	-.289 (-1.68) [-.671]	-.014 (-.06) [-.032]	-.193 (-.75) [-.448]	3.61	-.355 (-2.94)	-.891 (-4.45)	-.012 (-.05)	-1.179 (-3.92)	59.82
Whites										
Neonatal	-.157 (-1.71) [-.304]	-.136 (-.85) [-.263]	-.050 (-.23) [-.097]	-.059 (-.23) [-.114]	2.52	-.327 (-3.17)	-.464 (-2.57)	-.126 (-.51)	-.512 (-1.79)	17.09
Postneonatal	.052 (1.13) [.065]	.094 (1.17) [.117]	.044 (.40) [.055]	-.099 (-.78) [-.123]	1.92	.038 (.82)	.049 (.61)	.058 (.53)	-.192 (-1.51)	1.08
Infant	-.134 (-1.29) [-.253]	-.071 (-.39) [-.134]	-.068 (-.27) [-.128]	-.089 (-.30) [-.168]	1.30	-.303 (-2.61)	-.440 (-2.17)	-.078 (-.28)	-.715 (-2.23)	13.40

(continued on next page)

TABLE 2 (concluded)

Race-Mortality Rate	Short-Run Model P1	P2	P3	P4	F-ratio*	Cumulative Model P1	P2	P3	P4	F-ratio**
Blacks (n = 3,222)										
Neonatal	-.079 (-.34) [-.130]	-.044 (-1.20) [-.072]	-.153 (-.36) [-.251]	-.648 (-1.40) [-1.062]	3.44	-.287 (-1.13)	-.830 (-2.13)	-.328 (-.71)	-1.138 (-2.28)	11.69
Postneonatal	.265 (1.91) [.337]	-.059 (-.28) [-.075]	.100 (.25) [.127]	-.367 (-1.34) [-.466]	1.44	.300 (2.14)	-.179 (-.83)	.256 (1.00)	-.612 (-2.22)	2.39
Infant	.122 (.43) [.207]	-.360 (-.82) [-.611]	-.211 (-.41) [-.358]	-.744 (-1.33) [-1.263]	2.05	-.016 (-.05)	-1.004 (-2.12)	-.094 (-.17)	-1.739 (-2.87)	9.21

*See note to Table 1.

**The F-ratio pertains to the test of the hypothesis that no member of the set of four CHC variables has a nonzero effect. The critical F-ratio is 2.37 at the 5 percent level of significance and 3.32 at the 1 percent level of significance.

TABLE 3
Contribution of Community Health Centers to Reductions in Infant Mortality Rates, 1970-1978, Quadratic Specification

Race-Mortality Rate	Rate in 1970*	Rate in 1978*	Reduction*	Short-Run Contribution		Long-Run Contribution		Cumulative Contribution	
				Absolute*	Percentage	Absolute*	Percentage	Absolute*	Percentage
All Races									
Neonatal	14.865	9.497	5.368	.108	2.01	.247	4.60	.263	4.90
Postneonatal	4.685	4.174	.511	.021	3.70	.029	5.75	.031	6.07
Infant	19.550	13.671	5.879	.119	2.02	.274	4.66	.292	4.97
Whites									
Neonatal	13.454	8.308	5.146	.107	2.08	.206	4.00	.231	4.49
Postneonatal	3.866	3.519	.347	**	**	**	**	**	**
Infant	17.320	11.827	5.493	.111	2.02	.208	3.79	.242	4.41
Blacks									
Neonatal	22.769	15.496	7.273	.491	6.75	.801	11.01	.929	12.77
Postneonatal	9.012	7.334	1.678	.236	14.06	.299	17.83	.255	15.20
Infant	31.781	22.830	8.951	.636	7.11	1.071	11.96	1.167	13.04

*Deaths per thousand live births.

**Not computed since coefficients are not significant at the 5 percent level of confidence.

TABLE 4

Mortality Differentials: Counties with Four or More CHCs Compared to Counties with No CHCs, Cumulative Model

	Differential*	Percentage Impact**
All Races		
Neonatal	2.159	17.32
Postneonatal	.220	4.98
Infant	2.437	14.43
Whites		
Neonatal	1.429	12.92
Postneonatal	***	***
Infant	1.536	10.46
Blacks		
Neonatal	2.583	13.29
Postneonatal	.235	3.01
Infant	2.853	10.47

*Deaths per thousand live births. All differentials are negative.

**Differential as a percentage of the average predicted race- and age-specific mortality rate for counties with no CHCs for the period 1970-78.

***Not computed since coefficients are not significant at the 5 percent level of confidence.

Appendix

Ordinary least squares regression estimates of the Koyck or short-run model are contained in Tables A1, A2, A3, A4, A5, A6, A7, A8, and A9. Each table pertains to one of the nine alternative infant mortality rates. Both the dichotomous variable specification and the quadratic specification are shown. Estimates of the cumulative model are not shown.

The notation in the tables is as follows. Variable names ending in an asterisk denote race-specific measures.

1.	IMR_{t-1}, IMR^*_{t-1}	Infant mortality rate in year t-1; deaths of infants less than one year old per thousand live births
2.	NMR_{t-1}, NMR^*_{t-1}	Neonatal mortality rate; deaths of infant less than 28 days old per thousand live births
3.	$PNMR_{t-1}$, $PNMR^{*b}_{t-1}$	Postneonatal mortality rate; deaths of infants between the ages of 28 and 364 days per thousand live births
4.	NWB_t	Percentage of nonwhite births
5.	$MFINC_t$, $MFINC^{*c}_t$	Real median family income in hundreds of dollars; money median family income divided by the CPI
6.	MD_t	Office-based physicians in patient care (sum of active non-federal office based general practitioners and active non-federal office-based specialists) per thousand population

7.	$P1_{t-1}, \ldots, P4_{t-1}$	Set of four dichotomous variables; Pk_{t-1} equals one of the initial service date of the k^{th} CHC in a given county was as early as or earlier than year t-1
8.	CHC_{t-1}	Number of community health centers per thousand population
9.	$CHCSQ_{t-1}$	Square of preceding variable

TABLE A1

Weighted Neonatal Mortality Regressions, All Races*
(n = 6,102)

Independent Variable	Dichotomous Variable Specification		Quadratic Specification	
	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio
NMR_{t-1}	.565	54.64	.563	54.44
NWB_t	.039	15.86	.038	15.91
$MFINC_t$	-.016	-8.77	-.017	-9.19
MD_t	-.234	-1.16	-.384	-2.01
$P1_{t-1}$	-.174	-1.95	..	..
$P2_{t-1}$	-.296	-1.99	..	..
$P3_{t-1}$	-.003	-.02	..	..
$P4_{t-1}$	-.189	-.85	..	..
CHC_{t-1}	..	..	-162.350	-4.91
$CHCSQ_{t-1}$	..	..	6,592.463	2.69
Constant	5.999	23.55	6.191	24.095
R^2	.483		.484	
F	712.78		952.78	

*The critical t-ratios at the 5 percent level of significance are 1.64 for a one-tailed test and 1.96 for a two-tailed test. The two F-ratios are statistically significant at the 1 percent level of significance.

TABLE A2

Weighted Postneonatal Mortality Regressions, All Races*
(n = 6,102)

Independent Variable	Dichotomous Variable Specification		Quadratic Specification	
	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio
$PNMR_{t-1}$	.283	23.37	.285	23.60
NWB_t	.025	19.74	.025	21.08
$MFINC_t$	-.013	-13.15	-.013	-13.11
MD_t	-.294	-2.83	-.195	-1.99
$P1_{t-1}$	.096	2.10	..	..
$P2_{t-1}$	-.052	-.69	..	..
$P3_{t-1}$	.124	1.24	..	..
$P4_{t-1}$	-.198	-1.72	..	..
CHC_{t-1}	..	..	-33.154	-1.97
$CHCSQ_{t-1}$	..	..	2,525.641	2.01
Constant	4.032	32.74	4.000	32.688
R^2	.279		.279	
F	295.03		392.69	

*See note to Table A1.

TABLE A3

Weighted Infant Mortality Regressions, All Races*
(n = 6,102)

Independent Variable	Dichotomous Variable Specification		Quadratic Specification	
	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio
IMR_{t-1}	.569	55.65	.566	55.35
NWB_t	.053	18.17	.053	18.63
$MFINC_t$	-.023	-10.65	-.024	-11.03
MD_t	-.394	-1.69	-.477	-2.16
$P1_{t-1}$	-.120	-1.16	..	..
$P2_{t-1}$	-.289	-1.68	..	..
$P3_{t-1}$	-.014	-.06	..	..
$P4_{t-1}$	-.193	-.75	..	..
CHC_{t-1}	..	..	-181.653	-4.76
$CHCSQ_{t-1}$	..	..	8,338.278	2.94
Constant	8.281	26.14	8.485	26.59
R^2	.539		.539	
F	888.95		1,189.78	

*See note to Table A1.

TABLE A4

Weighted Neonatal Mortality Regressions, Whites*
(n = 6,102)

Independent Variable	Dichotomous Variable Specification		Quadratic Specification	
	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio
NMR^*_{t-1}	.483	43.73	.480	43.39
$MFINC^*_t$	-.022	-11.38	-.023	-11.78
MD_t	-.046	-.22	-.119	-.61
$P1_{t-1}$	-.157	-1.71	..	..
$P2_{t-1}$	-.136	-.85	..	..
$P3_{t-1}$	-.050	-.23	..	..
$P4_{t-1}$	-.059	-.23	..	..
CHC_{t-1}	..	..	-140.375	-4.17
$CHCSQ_{t-1}$	..	..	5,049.036	2.09
Constant	7.733	28.45	7.912	28.79
R^2	.298		.300	
F	369.59		521.37	

*See note to Table A1.

TABLE A5

Weighted Postneonatal Mortality Regressions, Whites*
(n = 6,102)

Independent Variable	Dichotomous Variable Specification		Quadratic Specification	
	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio
$PNMR^*_{t-1}$	.198	15.77	.199	15.94
$MFINC^*_t$	-.014	-14.57	-.014	-14.49
MD_t	.039	.37	.153	1.56
$P1_{t-1}$	.052	1.13	..	..
$P2_{t-1}$	.094	1.17	..	..
$P3_{t-1}$	.044	.40	..	..
$P4_{t-1}$	-.099	-.78	..	..
CHC_{t-1}	..	..	2.923	.17
$CHCSQ_{t-1}$	..	..	663.664	.55
Constant	4.395	35.66	4.359	35.35
R^2	.094		.093	
F	90.19		124.97	

*See note to Table A1.

TABLE A6

Weighted Infant Mortality Regressions, Whites*
(n = 6,102)

Independent Variable	Dichotomous Variable Specification		Quadratic Specification	
	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio
IMR^{*}_{t-1}	.470	42.40	.467	42.06
$MFINC^{*}_{t}$	-.032	-14.18	-.033	-14.52
MD_{t}	.003	.01	-.006	-.03
$P1_{t-1}$	-.134	-1.29	..	..
$P2_{t-1}$	-.071	-.39	..	..
$P3_{t-1}$	-.068	-.27	..	..
$P4_{t-1}$	-.089	-.30	..	..
CHC_{t-1}	..	..	-145.072	-3.79
$CHCSQ_{t-1}$	..	..	5,898.554	2.14
Constant	10.809	32.16	10.989	32.40
R^2	.309		.311	
F	389.60		549.18	

*See note to Table A1.

TABLE A7

Weighted Neonatal Mortality Regressions, Blacks*
(n = 3,222)

Independent Variable	Dichotomous Variable Specification		Quadratic Specification	
	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio
NMR^*_{t-1}	.390	25.16	.387	24.862
$MFINC^*_t$	-.001	-.24	-.006	-.92
MD_t	.399	.74	.480	.93
$P1_{t-1}$	-.079	-.34	..	..
$P2_{t-1}$	-.044	-1.20	..	..
$P3_{t-1}$	-.153	-.36	..	..
$P4_{t-1}$	-.648	-1.40	..	..
CHC_{t-1}	..	..	-309.369	-3.29
$CHCSQ_{t-1}$	..	..	8,797.011	.99
Constant	11.277	20.52	11.619	21.21
R^2	.174		.176	
F	97.01		137.19	

*See note to Table A1.

TABLE A8

Weighted Postneonatal Mortality Regressions, Blacks*
(n = 3,222)

Independent Variable	Dichotomous Variable Specification		Quadratic Specification	
	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio
$PNMR^{*}_{t-1}$	.213	12.85	.211	12.79
$MFINC^{*}_{t}$	-.019	-5.27	-.019	-5.38
MD_{t}	-1.152	-3.64	-.609	-2.00
$P1_{t-1}$	.265	1.91	..	..
$P2_{t-1}$	-.059	-.28	..	..
$P3_{t-1}$	.100	.25	..	..
$P4_{t-1}$	-.367	-1.34	..	..
CHC_{t-1}	..	..	-163.686	-2.97
$CHCSQ_{t-1}$	..	..	10,582.840	2.02
Constant	7.930	24.92	7.897	25.09
R^2	.076		.077	
F	37.88		53.82	

*See note to Table A1.

TABLE A9

Weighted Infant Mortality Regressions, Blacks*
(n = 3,222)

Independent Variable	Dummary Variable Specification		Quadratic Specification	
	Regression Coefficient	t-Ratio	Regression Coefficient	t-Ratio
IMR^{*}_{t-1}	.411	26.93	.406	26.50
$MFINC^{*}_{t}$	-.014	-1.91	-.018	-2.48
MD_{t}	-.523	-.81	-.004	-.01
$P1_{t-1}$	.122	.43	..	..
$P2_{t-1}$	-.360	-.82	..	..
$P3_{t-1}$	-.211	-.41	..	..
$P4_{t-1}$	-.744	-1.33	..	..
CHC_{t-1}	..	..	-414.284	-3.66
$CHCSQ_{t-1}$	..	..	16,938.980	1.58
Constant	16.616	23.03	16.983	23.57
R^2	.200		.202	
F	114.46		163.03	

*See note to Table A1.