Eastern Economic Journal, Volume XIV, No 1, January–March 1988

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

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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) has developed 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 (renamed the Bureau of Health Care Delivery and Assistance in 1982), 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 (Lave and Leinhardt 1972; Hollister, Kramer, and Bellin 1974; Reynolds 1976; Davis and Schoen 1978; Roemer 1981).

This paper undertakes to assess the impact of the CHC program on health levels. 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 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 (Fuchs 1974). 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 (Grossman and Jacobowitz 1981; Harris 1982; Lewit 1983; Rosenzweig and Schultz 1983, 1987; Corman and Grossman 1985; Corman, Joyce, and Grossman 1987; Joyce 1987). 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.

Our focus on infant mortality is particularly timely in light of its dramatic 56 percent decline between 1965 and 1984—from 24.7 deaths per thousand live births in the former year to 10.8 deaths per thousand live births in the latter year. Despite this rapid decline (4.0 percent per

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year annually compounded), large cross-sectional differences in the infant mortality rate persist. The most notable of these is the excess death rate of black babies, which was approximately twice as large as the white rate both in 1965 and in 1984. Given that poverty rates are much higher for blacks than for whites and that CHCs service poverty populations, it is important to investigate the actual and potential contributions of the centers to the reduction in the excess mortality rate of black infants. We address this issue by estimating separate regressions for white and black birth outcomes.

METHODOLOGY

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

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

In this equation m_{jt} is the infant mortality rate in the j^{th} county of the United States in year t, c_{jt-1} is the number of community health centers per thousand population in the j^{th} county in year $t-1$, c_{jt-1}^2 is the square of the number of centers per thousand population, 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 $t-1$.

Equation (1) constitutes a quadratic specification of the effects of CHCs on infant mortality, one which allows for nonlinear effects from the placement of additional centers in the same county on a per capita basis. Since the infant mortality rate is a negative indicator of health status, the predicted sign of α_1 is negative. The predicted sign of α_2 is ambiguous. On the one hand, "health returns" to the centers could diminish as more are added ($\alpha_2 > 0$) if they simply compete for the same population. On the other, health returns could increase ($\alpha_2 < 0$) if the greater presence of centers prompts still greater acceptance and use of their services.

The specification of the impact function captures 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 of these effects are captured by including the lagged infant mortality rate in the regressions.

As the last comment suggests, equation (1) is a distributed-lag model because the effects of CHCs on infant mortality are spread over a number of periods. 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 equation (1), it constitutes a short-run model of the impact of CHCs.

The basic model can be viewed as the result of applying a Koyck (1954) transformation to an infinite-lag distributed-lag model with geometrically declining weights. It allows both for diminishing returns from the placement of a specific CHC in a county over time (since α_4 is less than one) and for diminishing returns to the placement of a second CHC in the same county (if α_2 is positive).

DATA AND MEASUREMENT OF VARIABLES

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 (SMSAs), 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 Tape. 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.[1]

Use of infant mortality data for the period ending in 1978 is consistent with other recent studies of the determinants of birth outcomes in the U.S. Corman and Grossman (1985); Corman, Joyce, and Grossman (1987); and Joyce (1987) investigate the determinants of cross-sectional differences in race-specific infant mortality among large countries of the U.S. in 1977. Rosenzweig and Schultz (1987) estimate birthweight production functions in the 1980 U.S. National Natality Followback Survey.

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 anomalies, 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.

Separate regressions are fitted for white infant mortality and for black infant mortality as well as regressions for infant mortality of all races.[2] As mentioned in the introduction, the incidence of infant mortality and poverty is much higher among blacks than among whites.

Since CHCs service poverty populations, it is important to investigate their role in the determination of black death rates. Moreover, in preliminary regressions we tested and rejected the hypothesis that slope coefficients of all variables (but not intercepts) are the same for whites and blacks. Non-race-specific regressions are shown to summarize the total impacts of CHCs on U.S. infant death 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 heterogeneous. 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 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 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 and the lagged infant mortality rate 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 (Fuchs 1974; Brooks 1976; Gortmaker 1979; Hadley 1982). 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 (1967 = 100) to obtain real family income. The role of the race-specific U.S. birth rate (not county-specific) is indicated when the results of those specifications that employ it as a regressor are discussed in the section which follows.

RESULTS

Table 1 contains regression coefficients and t-ratios of the community health center measures in four alternative regression models for each of the nine mortality rates. Model 1 is a linear specification because the only community health center variable is the number of centers per thousand population (CHC). Model 2 is a quadratic specification because the number of centers per thousand population squared (CHCSQ) is included as an independent variable. For

reasons indicated below, models 3 and 4 add the U.S. birth rate (births per thousand women aged 15 to 44) to the linear and quadratic specifications.

A potential problem in all the regressions is that of serial correlation of the residuals. Since the lagged dependent variable is a right-hand side variable, the application of ordinary least squares yields regression coefficients and standard errors that are biased estimates of the corresponding population parameters if the residuals are serially correlated. Consequently, we tested for the presence of first-order serial correlation using the Durbin h statistic (Durbin 1970).[3] In each of the 36 regressions, we accepted the hypothesis of no first-order serial correlation at the 5 percent level of significance.

Focusing on model 2 (the quadratic specification with the birth rate omitted), one sees that community health centers in general have negative and statistically significant impacts on the alternative infant mortality rates studied. The coefficient of the number of CHCs is negative and significant in eight of nine cases.[4] The exception pertains to the white postneonatal mortality regression. The coefficient of the square term always is positive, which implies that there are diminishing returns to the placement of additional centers in the same county. In the equations in which the CHC variable is significant, the square term also is significant at the 5 percent level except in the black neonatal and total infant regressions.[5]

The significance of the CHC coefficients is not an artifact of the nonlinear specification. When the square term is omitted, the eight negative CHC coefficients retain their signs and are significant except in the all races postneonatal mortality equation (see model 1). Moreover, the value of the number of CHCs per thousand population that "minimizes" the relevant infant mortality rate (that is, the value beyond which the infant mortality rate begins to rise) always exceeds the mean of the CHC variable by more than three standard deviations. For example, the partial derivative of the number of CHCs per thousand population in the neonatal mortality equation for all races is negative as long as the CHC variable is smaller than .0123. In fact the mean number of CHCs per thousand population equals .0007, and the value at three standard deviations above the mean amounts to .0061.

It is notable that each coefficient of the relevant lagged infant mortality rate is significantly less than one. In the quadratic specification, the largest lagged coefficient of .566 is found in the all-races infant mortality regression. This coefficient is significantly smaller than one at all conventional levels (t-ratio = −43.40). These results provide strong support for the Koyck distributed-lag model. They suggest that there are diminishing returns to the placement of a CHC in a given county over time. Simultaneously, mortality differentials between counties with CHCs and those without them widen over time. Since the coefficient of the lagged infant mortality rate is less than one, a model that relates the rate of change in the infant mortality rate ($m_{jt} - m_{jt-1}$) to the per capita number of CHCs in year $t - 1$ but omits the lagged infant mortality rate from the set of regressors is seriously misspecified.

During our sample period (1970–1978), the U.S. infant mortality rate fell at the extremely rapid rate of approximately 4.4 percent per year. This decline may have been caused by factors other than those included in our model. One method of controlling for these factors is to add a time trend to the set of regressors. A second method is to employ the national level at time t of a potential determinant of infant mortality with a pronounced trend during the sample period. One example is the U.S. birth rate (births per thousand women aged 15 to 44), which fell by 27 percent for whites, 25 percent for blacks, and 25 percent for all races from 1970 to 1978.

The birth rate is added to the linear and quadratic specifications in the third and fourth models in Table 1.[6] All CHC coefficients are reduced in absolute value by the inclusion of the

TABLE 1

Regression Coefficients of Community Health Centers per Thousand Population (CHC) and Community Health Centers per Thousand Population Squared (CHCSQ), Alternative Regression Models[a]

	Model 1	Model 2		Model 3	Model 4	
Race-Mortality Rate	CHC	CHC	CHCSQ	CHC	CHC	CHCSQ
All Races (n = 6,102)						
Neonatal	−88.531	−161.350	6,592.463	−40.376	−79.305	3,460.515
	(−4.80)	(−4.91)	(2.69)	(−2.26)	(−2.48)	(1.47)
Postneonatal	−5.079	−33.154	2,525.641	6.476	−13.694	1,796.543
	(−.54)	(−1.97)	(2.01)	(.68)	(−.80)	(1.43)
Infant	−88.366	−181.653	8,338.278	−32.907	−87.958	4,895.662
	(−4.15)	(−4.76)	(2.94)	(−1.59)	(−2.37)	(1.79)
Whites (n = 6,102)						
Neonatal	−82.010	−140.375	5,049.036	−20.664	−28.193	646.798
	(−4.36)	(−4.17)	(2.09)	(−1.14)	(−.87)	(.28)
Postneonatal	10.562	2.923	663.664	15.912	12.342	306.781
	(1.13)	(.17)	(.55)	(1.67)	(.72)	(.25)
Infant	−76.967	−145.072	5,898.554	−7.879	−20.438	1,079.109
	(−3.60)	(−3.79)	(2.14)	(−.38)	(−.55)	(.41)
Blacks (n = 3,222)						
Neonatal	−234.152	−309.369	8,797.011	−100.838	−95.508	−269.482
	(−4.26)	(−3.29)	(.99)	(−1.84)	(−1.06)	(−.03)
Postneonatal	−73.393	−163.686	10,582.840	−19.410	−81.932	7,228.682
	(−2.28)	(−2.97)	(2.02)	(−.59)	(−1.47)	(1.38)
Infant	−269.297	−414.284	16,938.980	−111.459	−166.515	6,364.083
	(−4.06)	(−3.66)	(1.58)	(−1.69)	(−1.48)	(.61)

[a]t-ratios in parentheses. The critical t-ratios at the 5 percent level are 1.64 for a one-tailed test and 1.96 for a two-tailed test.

birth rate. For all races, the CHC variable retains its significance in the linear and quadratic neonatal mortality regressions and in the quadratic infant mortality regression. For blacks, the CHC coefficients are significant in the linear neonatal and infant mortality regressions. For whites, no significant CHC effects are observed.

Insertion of time or the U.S. birth rate into the model should be viewed with caution because pure time effects in regression models are measures of ignorance. Lave and Seskin (1977) have considered this issue in the context of the effects of air pollution on mortality in a time series of SMSA cross sections. They write (1977, p. 166) "... the time variables will represent characteristics common to most SMSAs in a given year. When such characteristics are controlled, they may bias the estimated coefficients of the air pollution variables toward showing no effects. For example, if air pollution is higher (across SMSAs) in some years than in others, increased mortality from increased air pollution might be ascribed to the time variables rather than to the air pollution variables." In our context, given the strong upward trend in the per capita number of CHCs, there is a high degree of correlation between time or the U.S. birth rate and the per capita number of CHCs. This creates serious problems of multicollinearity, especially since the data span a short nine year period. Moreover, the lagged infant mortality rate obviously is negatively related to time. Therefore, the former variable controls in part for the effects of time.

In light of the above considerations, we view the regressions that omit the birth rate as giving upper-bound estimates of the impacts of CHCs on infant mortality and the regressions that include the birth rate as giving lower-bound estimates. Also in light of these considerations, we stress the results obtained without the birth rate in the remainder of this paper. Clearly, these results should be interpreted with caution. They are suggestive rather than definitive and must be refined along the lines indicated at the end of the paper before they can be used to formulate new public policy initiatives with regard to the delivery of health care services to the poor.

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 2. Specifically, we apply the regression coefficients of the quadratic specification without the birth rate to trends in the CHC measures in the period under consideration. To illuminate the nature of the 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 2, row 3, columns 1–3). The short-term 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 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.

The sixteen 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 1.1 deaths per thousand live births (the long-run 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. According to the figures in Table 2, 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 2 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 may 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 births 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 2
Contribution of Community Health Centers to Reductions in Infant Mortality Rates, 1970–1978, Quadratic Specification

Race-Mortality Rate	Rate in 1970[a]	Rate in 1978[a]	Reduction[a]	Short-run Contribution		Long-Run Contribution	
				Absolute[a]	Percentage	Absolute[a]	Percentage
All Races							
Neonatal	14.865	9.497	5.368	.108	2.01	.247	4.60
Postneonatal	4.685	4.174	.511	.021	3.70	.029	5.75
Infant	19.550	13.671	5.879	.119	2.02	.274	4.66
Whites							
Neonatal	13.454	8.308	5.146	.107	2.08	.206	4.00
Postneonatal	3.866	3.519	.347	b	b	b	b
Infant	17.320	11.827	5.493	.111	2.02	.208	3.79
Blacks							
Neonatal	22.769	15.496	7.273	.491	6.75	.801	11.01
Postneonatal	9.012	7.334	1.678	.236	14.06	.299	17.83
Infant	31.781	22.830	8.951	.636	7.11	1.071	11.96

[a]Deaths per thousand live births.
[b]Not computed since coefficients are not significant at the 5 percent level of confidence.

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 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 may be 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.

Of course, the above conclusions are tentative rather than definitive. They are subject to the qualifications mentioned in the discussion of the role of time in the regressions. In particular, studies that employ data for longer periods of time, focus on infant mortality rates of poverty populations as opposed to counties, and include measures of the utilization of CHCs clearly are warranted.

Our results are related to recent attempts by Corman and Joyce (1985) and Corman, Joyce, and Grossman (1987) to understand the sources of the rapid decline in the U.S. infant mortality rate since 1965. Based on extrapolations of cross-sectional infant mortality regressions for 1977, these studies highlight the importance of community health centers and such related Federal programs as maternal and infant care projects, subsidized family planning clinics, Medicaid, and the Special Supplemental Food Program for Women, Infants and Children (WIC program). In addition, the rise in the legal abortion rate and the increase in the quantity of neonatal intensive care are identified as contributing factors to the downward trend in infant deaths.

Although the infant mortality rate declined by 4.0 percent per year between 1965 and 1984, it declined by only 3.4 percent per year between 1981 and 1984. Moreover, the black postneonatal mortality rate, which has been trending downward at a faster pace than the white postneonatal mortality rate, actually rose between 1982 and 1983. Since the beginning of 1981, budget cutbacks by the Reagan Administration have curtailed the rates of growth of such poverty-related programs as WIC, maternal and infant care projects, community health centers, subsidized family planning clinics, and Medicaid. When inflation is taken into account, the absolute sizes of some of these programs declined in real terms. These developments have caused some persons to attribute the deceleration in the rate of decline in infant mortality to the Reagan Administration's policies (for example, Miller 1985).

Based on our study and the related studies by Corman, Joyce, and Grossman, the role of public policy in the above developments is not clearcut. The introduction and diffusion of new techniques in neonatology and the diffusion of abortion as a relatively new contraceptive technique slowed appreciably in the late 1970s and early 1980s. Medicaid and community health center funding cutbacks may have made it more difficult for pregnant low-income women to initiate prenatal care early in their pregnancies, although the recession of 1981–1982 also may have played a role. In summary, more research is required to provide a fuller explanation of the role of public policy in general and community health centers in particular in U.S. infant health outcomes. Our study represents a useful first step in this process.

FOOTNOTES

*We wish to thank Maureen Cropper, Karen Davis, and two anonymous referees for helpful comments and suggestions. We are indebted to Richard Bohrer and Robert Nelson of the Bureau of Health Care Delivery and Assistance, DHHS, for helping us to obtain the Bureau of Community Health Services (the former name of the Bureau of Health Care Delivery and Assistance) Common Reporting Requirements data tape, which is the basis for some of the research in this paper. We are also indebted to Allison Mann and Frank Chaloupka 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. An expanded version of the paper, which contains complete tables of regression results and mathematical proofs of certain statements made in the text, is available on request.

1. Note that, although the time series on infant mortality begins in 1969, the initial service dates of the CHCs can be as early as 1965.
2. 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.
3. It is well known that the Durbin-Watson statistic cannot be used to test for first-order serial correlation when the lagged dependent variable is a regressor. In computing the Durbin h statistic, we averaged the individual serial correlation coefficients obtained for each county. Note that our data span a nine year period. Therefore, some caution should be exercised in interpreting the results of the Durbin h test since its small-sample properties are not yet fully established.
4. A one-tailed test is employed because the alternative hypothesis is that each coefficient is negative. In general, statements concerning statistical significance in the text are based on one-tailed tests except when the direction of the effect is unclear on a priori grounds or when the estimated effect has the "wrong sign." In the latter cases two-tailed tests are used. One-tailed tests are applied to the coefficients of the number of CHCs per thousand population squared because almost all of the estimated effects are positive. Put differently, we adopt the hypothesis that there are diminishing returns to the placement of additional CHCs in the same county.
5. In the black total infant mortality regression, the coefficient of the square term is significant at the 6 percent level. In the black neonatal mortality regression, the corresponding coefficient is not significant at a level of confidence as high as 10 percent.
6. This specification is adopted in part to alleviate the serious problems of multicollinearity that are encountered when time itself is used as a regressor.

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