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The U.S. infant mortality rate, defined as the number of deaths before age one per 1000 live births, fell from 12.6 in 1980 to 6.9 in 2000, a decline of 45 percent.1 Over this same period, the total age-adjusted death rate in the United States fell by only 16.4 percent. We can decompose this decline in infant mortality into two components: changes in the healthiness of newborns and changes in the survival rate of newborns conditional on a given level of health. One widely used measure of newborn health, the rate of low birth weight births, is defined as the percentage of live births of babies who weigh less than 2500 grams or 5.5 pounds. The rate of low birth weight in the United States has actually risen since 1980, from 6.8 to 7.6 percent.2 A large portion of the increase is attributable to the rise in multiple births, which have grown from 2 to 3 percent of all live births over the same period. However, even if we adjust for multiple births, the underlying healthiness of newborns in the United States has remained largely unchanged since 1980. In short, the remarkable increase in the survival rate of infants has resulted almost exclusively from advances in the technology of newborn care.

Why, therefore, has the underlying morbidity of newborns, as proxied by the rate of low birth weight births, remained so immovable? Even more baffling, why has there been so little change in the rate of low birth weight despite increases in the prenatal inputs that many contend should lower its incidence. For instance, the percentage of women who initiate prenatal care in the first trimester increased from 76.3 in 1980 to 83.7 in 2000. The percentage of women who smoke during pregnancy fell from 18.4 in 1990 to 11.4 in 2002, while the number of infants served by the Special Supplemental Nutrition Program for Women, Infants and Children (WIC) has almost doubled since 1988.3

Recent research by my colleagues and me suggests that previous estimates of the efficacy of many inputs designed to improve newborn health is probably inflated by favorable selection. The women who initiate prenatal care early, or who participate in WIC, are likely to be more motivated, less stressed, and more risk averse than the women who start care late or who do not participate in WIC. Too often we lack empirical methods for overcoming the problems caused by selection. In addition, in vetting their results, economists often neglect the clinical literature. Consider studies of the effect of programs to enhance maternal nutrition on infant health. Economic theory is helpful in specifying the demand for nutrition, but the effect of nutrition on fetal growth is a physiological, not an economic, relationship. For example, the consensus in the literature has been that “WIC works.” In a recent study, economists reported that prenatal WIC participation was associated with a 50 percent decline in very preterm births, infants born before 33 weeks gestation.4 These results were consistent with a widely-cited study by economist Barbara Devaney and colleagues in which WIC was associated with a decline of between 2.2 and 6.2 percentage points in rate of preterm birth.5 Nationally, 9.7 percent of single births — versus twins, triplets, and other multiple births — were preterm in 1989. These are remarkable improvements, but they are strongly at odds with the clinical literature. In randomized trial after trial, clinical researchers have been unable to find any intervention that prevents preterm birth. In a candid editorial in the New England Journal of Medicine, a leading investigator writes:

“Trials measuring the effect of interventions at eliminating a single risk factor are numerous; uterine contractions have been suppressed, cervixes have been sewn shut, microorganisms have been eliminated, and social support, better nutrition, and prenatal care have been provided. When these factors have been studied in isolation, not one has resulted in a decline in preterm birth” (p. 54).

I am not arguing that social scientists have little to contribute to the clinical literature in matters of health.
However, the “theory” that guides the interpretation of treatment effects is often medicine and not economics. In such analyses, we can increase the credibility of our work if we use clinical findings to understand and perhaps challenge our results.

My colleagues Diane Gibson and Silvie Colman and I make these points in a recent NBER Working Paper.7 We use 14 years of birth certificates from New York City to analyze the effect of prenatal WIC participation on measures of fetal growth from 1988 to 2001. Because we have no convincing instruments, we take advantage of our large sample size to stratify the analysis by race, ethnicity, nativity, parity and the timing of prenatal care. The objective is to lessen unobserved heterogeneity by comparing similar women. For instance, we consider only women on WIC or on Medicaid. We further limit the sample to women with no previous live births and who thus have no experience with WIC from a prior pregnancy. We further limit the study to women who initiate prenatal care in the first months of pregnancy. These are likely to be the most motivated women with the longest exposure to WIC during pregnancy. Finally, we analyze two important subgroups separately: U.S.-born Blacks and foreign-born Hispanics. This latter stratification is motivated by our previous research on prenatal exposure to crack cocaine in New York City.8 The crack epidemic hit U.S.-born Blacks in New York City much more intensely than other groups. There is also rigorous evidence that cocaine use among pregnant Hispanic women has been minimal.9 If exposure to crack is an important omitted variable, then it is more likely to contaminate results among U.S.-born Blacks than foreign-born Hispanics.

Our results suggest that prenatal participation in WIC has had little impact on fetal growth in New York City between 1988 and 2001. However, we do find a strong association between WIC and rates of low birth weight among U.S.-born Blacks between 1988 and 1992 and relatively little association thereafter. We uncover no association between fetal growth and WIC among foreign-born Hispanics in any year. The results for U.S.-born Blacks, we suspect, are related to differences in prenatal exposure to crack cocaine between WIC and non-WIC participants during the peak years of the epidemic. As the epidemic waned, so did the association between WIC and low birth weight.

One criticism is that our results pertain to a relatively low-risk group of women. Previous researchers have found stronger effects of WIC on birth outcomes among unmarried women, teens, and smokers. We contend that stratification by such endogenous risk factors may exacerbate problems of omitted variables. As an alternative, we use “twinning” as an exogenous risk factor and we compare differences in fetal growth between WIC and non-WIC participants who delivered twins. Over half of twin births are low birth weight and the risk of anemia and inadequate weight gain are substantially greater among twins than single births. If WIC improves fetal growth, then it is more likely to be evident among twins. Again, we find little association between WIC and fetal growth among twins except for U.S.-born Black less than 25 years of age and only for selected years.

Demand for Health Inputs

The estimation of treatment effects is clearly a challenge in studies that use non-randomized research designs. In recent papers, my colleagues and I have focused on the demand for health inputs in which the treatment effects have been well established by clinical trials. For instance, there is widespread agreement among clinicians and epidemiologists that prenatal smoking stunts fetal growth. Our contribution was to analyze the determinants of prenatal smoking. We were not the first. William Evans and Jeanne Ringel used national natality files and demonstrated that cigarette tax evasion was a highly effective measure of both the quality of pediatric care and the improvement in health associated with vaccine-preventable illnesses. There are now 19 doses of vaccines that an infant should receive within the first 18 months, up from 8 in 1987. Costs of vaccines to fully immunize a child have risen from $116 in 1987 at private sector prices to $525 in 2002. However, new vaccines are much more expensive than older ones. The varicella and pneumococcal vaccines cost approximately $62.00 per dose, making them three times more expensive than either the combined vaccine for diphtheria,
acellular pertussis, and tetanus (DaPt) or the inactivated vaccine for polio (IPV). The number and costs of vaccines, as well as the complexity of vaccine schedules, suggest that up-to-date immunization rates may be sensitive to whether parents have health insurance that covers childhood vaccines. To test this, Andrew Racine and I used the recently released National Immunization Survey (NIS) to determine whether the State Children’s Health Insurance Program (SCHIP) was associated with a relative increase in vaccine coverage rates among poor and near-poor relative to non-poor children. The NIS is an annual population-based survey of households with at least one child between 19 and 35 months of age. The survey contains information from approximately 34,000 households per year from 1995 to 2002. Until the recent release of NIS, the United States had little consistent information at the state and metropolitan level with which to monitor immunization rates and to assess the effect of initiatives such as SCHIP.

We estimated a reduced-form model of immunization rates on the presence of SCHIP program. Identification came from variation in the timing of SCHIP implementation by states and the generosity of state programs, as measured by income eligibility thresholds above those that existed for infants and children through Medicaid. We found little evidence to suggest that SCHIP has had a major impact on narrowing the gap in immunization rates between poor and non-poor children. The one exception was the varicella vaccine in which differences in coverage rates between poor and non-poor children converged rapidly between 1997 and 2001. Moreover, convergence was faster among poor children from groups or areas with above average rates of uninsured children. However, in tests of robustness we found that the rise in varicella coverage rates often preceded implementation in SCHIP, which undermined a causal interpretation.

In summary, advances in perinatal care and the development of new pediatric vaccines are probably the two most important changes affecting infant and child health over the past two decades. Research as to which policies most effectively improve access and use of these inputs would be a useful area of work for economists.


12 See http://www.cdc.gov/nip/vfc/cdc_vac_price_list.htm