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INDUCED INNOVATION AND SOCIAL INEQUALITY: EVIDENCE FROM INFANT MEDICAL CARE

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ABSTRACT

We develop a model of induced innovation where research effort is a function of the death rate, and thus the potential to reduce deaths in the population. We also consider potential social consequences that arise from this form of induced innovation based on differences in disease prevalence across population subgroups (i.e. race). Our model yields three empirical predictions. First, initial death rates and subsequent research effort should be positively correlated. Second, research effort should be associated with more rapid mortality declines. Third, as a byproduct of targeting the most common conditions in the population as a whole, induced innovation leads to growth in mortality disparities between minority and majority groups. Using information on infant deaths in the U.S. between 1983 and 1998, we find support for all three empirical predictions. We estimate that induced innovation predicts about 20 percent of declines in infant mortality over this period. At the same time, innovation that occurred in response to the most common causes of death favored the majority racial group in the U.S., whites. We estimate that induced innovation contributed about one third of the rise in the black-white infant mortality ratio during our period of study.

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Technological change is a source of substantial aggregate welfare improvements. Several studies suggest that technological change accounts for up to a third of aggregate economic growth (Jorgenson 2000). Yet overall welfare gains do not imply equal benefit for all individuals. If technological change is biased towards some industries or groups, some parts of the population will benefit more than others.

In this paper, we investigate biased technological change using a particular example – medical technology for treating at risk infants. Infant mortality provides a useful setting to learn about induced innovation because the outcome is easy to measure (deaths) and disparities in outcomes are so widely noted. Further, there has been enormous technological progress. In the early 1960s, about 25 of every 1,000 infants died before their first birthday, most before leaving the hospital. Much of this death was in premature infants – infants born before normal gestation, and typically low birth weight, or under 2500 grams.

The situation of newborns dying so young created a moral imperative to reduce those deaths. The highly publicized death of John F. Kennedy's infant son shortly after his premature birth attracted further attention to the magnitude of deaths to infants. Clinicians treating infants began to innovate, starting what would spur the development of neonatal intensive care units (Baker 1996; Anspach 1997). Grant money followed, and physicians and scientists became energized. Treatment progress was impressive. In the four and a half decades since 1960, mortality for low birth weight babies declined 70 percent, almost entirely as a result of improved medical care (Cutler and Meara, 2000).

The first part of our empirical analysis shows the link between humanitarian need and technological change. We look at the role of induced innovation using data on death

by cause. We investigate whether causes of death with higher mortality rates early in the time period experienced larger reductions in infant mortality over time. Our results support this prediction. Every death per 1,000 births attributed to a particular cause in 1983-85 is associated with a 20 percent greater reduction in mortality from that cause over the subsequent 13 years.

We then go on to examine the impact of these changes on social inequities in health. We focus specifically on the ratio of black to white infant deaths, which characterizes the relative rate of progress for blacks compared to whites. Since there are nearly four times as many white than black births, leading causes of death will inherently be those which whites suffer from relatively more. When progress was made on leading causes of death, therefore, it benefited white newborns more than black newborns. Using counterfactual simulations, we show that racial gaps in birth weight-specific mortality have widened over time as a direct result of the research progress that was made. As a result, medical need has led to improved aggregate outcomes, but with a disproportionate share of those benefits accruing to majority groups.

The paper is structured as follows. The first section presents a simple model of induced innovation in medicine that shows why research would be allocated to more common diseases and how induced innovation could increase disparities in health outcomes. The second section describes infant mortality trends in recent decades and presents a case study of a particular cause of death, respiratory distress syndrome (RDS). Section three presents the data, and section four empirically tests for induced innovation. Section five then translates these estimates into the social consequences of induced innovation. The last section concludes.

II. A Model of Induced Innovation

In this section, we use a simple framework to explore how the obvious and painful suffering of people near death could lead to technological change, and how that change might affect different population groups. For simplicity, we consider the setting we employ in our empirical analysis: survival of low birth weight infants.

A significant body of recent research has considered models of endogenous innovation. In most of these models, innovation is posited to respond to profits – either greater demand for some industries (Schmookler, 1966; Romer, 1990; Grossman and Helpman, 1991; Aghion and Howitt, 1992; Kremer, 2002; and Acemoglu and Linn, 2004) or differential factor costs (as in Newell, Jaffee, Stavins, 1999; and Popp, 2002).

The medical sector is not well characterized by pure profit motives, however. Most hospitals are not-for-profit and much innovation is done by independent, university-based researchers. We posit an alternative framework in which the humanitarian desire to improve health drives innovation. Lichtenberg (2001) and Bhattacharya and Packalen (2008) similarly model the allocation of public spending and innovation in the non-profit sector.

We consider a set of diseases that might result in death, abstracting from quality of life. Let the mortality rate for a particular diagnosis i at a point in time t be denoted d_i^t. We consider two periods, a base period $t = 0$ and a later period $t = 1$, where individuals from time 0 are no longer in the relevant population. The death rate is given by

$D^t = \sum_{i=1}^{n} d_i^t$, where n is the number of distinct diseases.

Medical research on a particular condition will improve survival according to a (probabilistically) known innovation possibility function. We model this as a function $f_i(r_i)$, that converts research between periods 0 and 1 into a survival probability at time 1. We assume straightforwardly that $f_i(0) = 0$, $f_i < 1$, $f_i' > 0$, and $f_i'' < 0$. The death rate for condition i at time 1 is then $d_i^1 = d_i^0 \cdot (1 - f_i(r_i^0))$, and the aggregate death rate in period 1 is $D^1 = \sum_{i=1}^{n} d_i^0 \cdot (1 - f_i(r_i))$.

Note that this formulation assumes no spillovers across diseases – that is, research conducted on one disease affects mortality only for that condition. We argue below that this assumption is conservative in our setting.

We consider a social planner wishing to maximize social welfare. This might be the National Institutes of Health, which funds a large share of basic biomedical research, or university researchers on their own, thinking about valuable projects to explore. The social planner wishes to minimize mortality in period 1, with a total research budget fixed at R.

The planner would then solve the following problem:

$$\sum_{i=1}^{n} d_i^0 (1 - f_i(r_i)) \quad \text{s.t.} \sum_{i=1}^{n} r_i < R . \tag{1}$$

The first-order condition is straightforward:

$$d_i^0 \cdot f_i'(r_i^*) = d_n^0 \cdot f_n'(r_n^*), \text{ for all } i. \tag{2}$$

Equation (2) states that the expected marginal benefit of research should be the same across all diseases. Provided the f_i functions are not too different across conditions,

this means that more common diseases deserve research that is less productive on the margin, and thus get more research funding.[1]

Disparities in health outcomes will be related to research innovation. Because more medical research is done for more common diseases, the socially optimal allocation of research dollars will tilt towards diseases that are relatively more common in larger population groups.

To see this, consider the case where there are two groups, a majority group *a* and a minority group *b* (in our empirical example, whites and blacks). Let the initial death rates per condition for two groups be $d^0_{a,i}$ and $d^0_{b,i}$ and their respective sums across conditions be D_a^0 and D_b^0. At time 1, the death rates are given by

$$d^1_{a,i} = d^0_{a,i} \cdot (1 - f_i(r_i)) \quad \text{and} \quad d^1_{b,i} = d^0_{b,i} \cdot (1 - f_i(r_i)),$$

and the mortality ratio at each time is given by D_b^t/D_a^t. This ratio increases over time if $D_b^1/D_b^0 > D_a^1/D_a^0$, which in our model expands to

$$\sum_{i=1}^{n}\left(1 - f_i(r_i)\right)\frac{d^0_{b,i}}{D^0_b} > \sum_{i=1}^{n}\left(1 - f_i(r_i)\right)\frac{d^0_{a,i}}{D^0_a}, \tag{3}$$

or

$$\sum_{i=1}^{n} f_i(r_i)\left(\frac{d^0_{a,i}}{D^0_a} - \frac{d^0_{b,i}}{D^0_b}\right) > 0\,. \tag{4}$$

Thus the mortality ratio rises when increases in survival probabilities ($f_i(r_i)$) are correlated with higher initial shares of deaths per condition among the majority group $\left(\frac{d^0_{a,i}}{D^0_a} - \frac{d^0_{b,i}}{D^0_b}\right)$.[2]

[1] In our work, we do not observe differences in the innovation function, so we consider it similar across diseases. Bhattacharya and Packalen (2008) attempt to model this empirically, assuming a structural model of research opportunity for pharmaceuticals that declines as drugs get older.

Such a correlation can be present for several reasons. The difference in the initial shares of deaths attributed to a given condition across groups may arise because a particular disease is more prevalent in the majority population than in the minority population, or more fatal for the majority group. For example, among infants born prematurely, black infants tend to have less severe illness than do white infants along a number of dimensions, holding gestation constant (Hulsey et al. 1993; Richardson et al. 1994; Richardson et al. 1999; Berman et al. 2001). In particular, at any given gestation, black infants are less likely to have RDS, and, on illness severity measures, black infants score better than whites even given the presence of a condition like RDS (Hulsey et al. 1993; Richardson et al. 1994). Thus, death rates from RDS are greater among whites than among blacks, even within narrow birth weight categories. As a result, research on RDS will disproportionately benefit whites over blacks. Thus, if research favors common causes of death, we can expect ($f_i(r_i)$) to be positively correlated with higher death rates in a majority group.

In this model, the increasing disparity occurs as a consequence of the differences in population sizes, not because deaths are valued differently by the social planner. To see this more formally, consider two diseases, one with a greater prevalence among whites and the second with a greater prevalence among blacks. Let e_1^0 be the prevalence of disease 1 among whites in the base period, and θe_1^0 be the prevalence among blacks, where $\theta<1$. Conversely, let e_2^0 be the prevalence of disease 2 among blacks and θe_2^0 be the prevalence among whites. To simplify notation, suppose that each case of the disease is fatal, so that death rates are equal to prevalence rates.

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[2] Note that in the model, increases in survival probabilities ($f_i(r_i)$) are assumed to be constant for the two groups.

The overall mortality rate from disease 1 is $d_1^0 = \frac{e_1^0 N_a + \theta e_1^0 N_b}{N_a + N_b}$, where N_a and N_b are the number of white and black births respectively. Similarly, the mortality rate for disease 2 is $d_2^0 = \frac{\theta e_2^0 N_a + e_2^0 N_b}{N_a + N_b}$. Combining these mortality rates with equation (2) gives a formula for the marginal product of research on each disease in equilibrium:

$$\frac{f_1'(r_1)}{f_2'(r_2)} = \left(\frac{e_2^0}{e_1^0}\right) \cdot \left(\frac{\theta N_a + N_b}{N_a + \theta N_b}\right) \tag{5}$$

If $\theta<1$ and $N_a > N_b$, the second expression on the right hand side of equation (5) is less than 1, and hence the overall expression on the right hand side is less than the ratio of disease incidence in the two groups. As a result, disease 1 should receive relatively greater funding than if the populations were the same, with the disparity rising as the population disparity rises.

The induced innovation hypothesis has several predictions, which we test in the empirical section of the paper. First, the theory predicts that initial death rates and subsequent research effort should be positively correlated. Second, innovation should be associated with more rapid mortality declines. As a result, induced innovation leads to growth in mortality disparities between minority and majority groups.

II. Background on Infant Mortality and Neonatal Medicine

Infant mortality, or death during the first year of life, used to be much more common than it is today. In 1915, for example, infant mortality was 150 babies per 1,000 born alive. With improved nutrition and advances in public health, that rate fell to 26 per 1,000 in mid-century.

The latter half of the 20th century witnessed continued declines in infant mortality. Figure 1 demonstrates that white infant mortality rates fell from 26 deaths per 1,000 live births in 1950 to 5.7 in 2004. Black infants experienced higher rates of mortality at every point in time, but infant mortality fell dramatically for blacks as well, from 43.9 deaths per 1,000 live births in 1950 to 13.8 deaths per 1,000 births in 2004. The economic value of this improvement is immense. Using the common estimate of $7 million per life, the value of reduced mortality is roughly $210,000 per black birth and $140,000 per white birth. With about 3 million black and white births per year in the United States, this translates to roughly $550 billion per year.

A good share of the reduction in infant mortality in the past half century has come from reduced mortality of low birth weight infants, consistent with the concentration of deaths among babies born low birth weight. In 2004, nearly 70 percent of infant deaths occurred among the 8 percent of babies born weighing under 2500 grams. Figure 2 depicts the gain in survival by birth weight. Mortality for the lightest infants (500-999 grams) fell from nearly 90 percent in 1960 to 30 percent in 1998. Mortality among infants weighing 1,000 to 1,499 grams fell from 50 percent to below 10 percent. Over half of improved survival for all infants between 1960 and 2005 was a result of lower mortality in low birth weight infants.[3]

Unlike early in the century, when improved nutrition and public health were the keys to improved survival, advances in medical care were much more important in the last few decades. Low birth weight infants die of many causes, but respiratory-related

[3] Compared with the actual infant mortality rate of 6.86 per 1000 live births in 2005, the infant mortality rate would have been 14.79 in 2005 based solely on improved rates of survival among low birth weight infants. Compared to the 1960 mortality rate of 25.14, this represents 57 percent of the actual improvement in survival from 1960 to 2005.

conditions and congenital anomalies are particularly important. Among the very lightest, or Very Low Birth Weight births (< 1500 g), respiratory-related conditions were the most common cause of death in 1980. An infant's lungs do not develop the capacity to transfer oxygen into the blood until about 23 to 25 weeks of gestation, and even after that time difficulties breathing are common. In the 1970s and 1980s, RDS and the related bronchopulmonary dysplasia, which often resulted as a consequence of treatment with ventilators, were the primary causes of mortality among Very Low Birth Weight infants, and an important condition for low birth weight infants. At that time, RDS caused about 10,000 deaths per year. A major part of medical care for premature infants is helping them breathe. Other major causes of infant death include sudden infant death syndrome, congenital anomalies (especially heart defects), infections, and pneumonia.

Starting in the 1960s and continuing today, neonatal intensive care emerged as a field of medicine to treat those conditions. Neonatal intensive care embodies hundreds of small innovations often adopted from care for adults, but adapted to very light infants. The innovations range from improving the technology for ventilation, improving the ability to monitor newborn blood and respiratory function, to the development of synthetic surfactant that can be administered to infants with RDS. These innovations do not guarantee survival, but they increase its chances. Cutler and Meara (2000) show that improved care during the neonatal period for critically ill infants collectively accounted for essentially all of the reduction in neonatal mortality after 1960.

Even as overall infant mortality fell from 1950 on, the ratio of black to white infant death rates rose. In 1950, the ratio of black to white infant mortality was about 1.6. In the early 1980s, it was just above 2.0. By the late 1990s, the ratio was about 2.5. The

increase in this ratio has been widely noticed. The U.S. government's Healthy People 2010 initiative has called for the elimination of racial disparities in health outcomes. Yet the most prominent indicator of racial inequality is moving adversely to goals. As a result, there has been a good deal of focus on how to improve black infant outcomes (AHRQ 2001; AHRQ 2008; Howell 2008).

Some of this adverse trend for blacks relative to whites is a greater incidence in low birth weight births among blacks. But that is not the entire story; our calculations (described below) indicate that adverse trends in the birth weight distribution account for only one-third of the increase in the black-white infant mortality ratio. The rest comes from differential improvements in survival at any given birth weight -- racially-biased technological progress.

An Example: Respiratory Distress Syndrome

To understand the effects we analyze, consider the specific example of RDS.[4] Somewhere between 24 and 28 weeks of gestation, a healthy, developing fetus begins to produce surfactant. The role of surfactant is to help keep the lung sacs, or alveoli, open. Without ample surfactant, the alveoli collapse during breathing, causing damaged cells to collect in airways, and impeding breathing ability. Death is a frequent result. By 35 weeks gestation, most babies have developed enough surfactant to maintain appropriate surface tension in lung airways. In the interim between 24 weeks and 35 weeks (approximately), infants are at decreasing risk of death from RDS.

[4] A review by Clements and Avery (1998) characterized the progress leading to modern day treatment for RDS in detail.

The first observations about the biological process of RDS were made as early as 1903. However, it was not until after 1950 that surfactant was discovered in lung extracts and eventually connected to what is now known as RDS. Between the late 1950s and the early 1990s, a wave of government, industry, and academically sponsored research helped to uncover the treatments for RDS. The first scientists experimented in uncontrolled settings and reported the use of animal surfactant from rabbits and cows. This early research, reported by 1980, spurred other researches to launch controlled clinical trials using animal surfactant. Synthetic surfactant was developed later on, with continuing trials to determine the appropriate timing of treatment and optimal delivery of the drug. The effort was immense; an estimated 30,000 infants across North America, Europe, and Japan were enrolled in clinical trials of surfactants by 1990. The results of a subset of these, over 35 randomized controlled trials of 6,000 infants, have been reported. Surfactant was approved for wide use by the FDA in August, 1990.

Figure 3 shows the resulting trend in RDS mortality. To hold constant need, we present RDS mortality for two specific weight ranges: 500-999 grams, and 1,000-1,499 grams. These ranges correspond to the greatest respiratory impairment.

Both blacks and whites had high mortality rates from RDS in the early 1980s. The rate for whites was about 120 deaths per 1,000 births for the lighter infants, and 50 deaths per 1,000 births for the heavier ones. The rate for blacks was high as well, although a bit lower in each case. As noted above, black infants experience faster maturation of lungs than white infants (Richardson et al. 1999; Berman et al. 2001).

Because surfactant was so promising, a large share of very low birth weight babies was enrolled in clinical trials of the drug in the 1980s. Thus, the decline in RDS

mortality in the 1980s is likely due to surfactant. The approval of surfactant in 1990 coincided with a further immediate decline in death rates and a continuing decline over the next few years. Between 1983 and 1998, RDS-related mortality for black and white babies combined fell 63 percent among those weighing 500 to 999 grams, and by 87 percent among those weighing 1,000 to 1,499 grams. Because RDS was more important for white babies than black babies, however, the reduction in RDS mortality led to significantly greater survival improvements for whites than for blacks. Thus, even with equal declines in mortality across racial groups, there was an increase in the racial gap in outcomes. Between 1983 and 1998, we estimate that about 20 percent of the increase in the ratio of black to white infant mortality resulted from improvements in RDS survival.

III. Data

To understand the sources of innovation in infant medical care, and the consequences of those innovations, we use data on mortality by race and cause, and on medical innovation. We describe the sources in turn.

The mortality data we employ come from cohort linked birth / infant death (LBID) files produced by the National Center for Health Statistics (NCHS, various years). These files contain a nearly universal sample of births and infant deaths in the United States, formed by compiling data from birth and death certificates.[5] Deaths that occur within one year of birth are matched back with their birth certificates to create the linked records. The files are organized by annual, calendar year birth cohorts, so deaths

[5] In 1983 and 1984, some states created records for only half of births, randomly selected, but all deaths were entered. Birth records have weights to adjust for the sampling.

may be from the same year or the following year as long as they occur within 365 days of birth.

The earliest LBID data is from 1960. However, only published tabulations of those data exist, and the published tabulations do not have the detail we need. The next year of linked data are from 1983, and are available in micro data. We thus use data from 1983-85 as our early time period. We pool years to improve death rate estimates for relatively rare conditions. Fortunately, changes in infant mortality were ongoing in this time period, and the profile of deaths by cause in 1983-85 is similar to that for 1960. As figure 2 shows, mortality among low birth weight infants roughly halved between 1983 and 1998. Changes in the coding of cause of death after 1998 led to substantial differences in cause-specific death rates among low birth weight births after that year, so our later time period uses data from 1996-98.

From these records, we use information on birth weight, the mother's race and Hispanic ethnicity, singleton or plurality of birth, and the underlying cause of death. We include births only to black or white mothers, excluding births to Hispanic mothers to limit the effect of increasing immigration over time. We further exclude plural births to limit the effect of secular changes in multiple births due to fertility treatment and a contemporaneous rise in maternal age, which increases the risk of multiple births (Blondel and Kaminski 2002).

Summary statistics for the mortality data are shown in Table 1. In 1983-85, the infant mortality rate for whites was 8.2 per 1,000 births and the rate for blacks was 17.1 per 1,000 births, for a corresponding black-white ratio of 2.09. Because blacks are a smaller share of the population than whites (84 percent of births are for whites, excluding

Hispanics), however, there are many more white deaths than black deaths. Over 70 percent of infant deaths are among white infants.

The next column of the table shows the substantial reduction in infant mortality over time. The infant mortality rate for whites fell by 37 percent between 1983-85 and 1996-98; the comparable reduction for blacks was 23 percent. As a result, the black-white infant mortality ratio rose to 2.4, a 30 percentage point increase.

Our model does not address changes in the birth weight distribution over time. We thus purge from the increase in the black-white mortality ratio the contribution of unequal changes in birth weight by race. The fourth column of Table 1 shows a simulation of infant mortality rates in 1996-98 if the race-specific distribution of births by 500 gram intervals had not changed over the time period. In both cases, infant mortality rates would be lower, reflecting a trend towards more low birth weight infants over time. But the changes are not large. Relative to the 30 percentage point increase in the unadjusted black-white infant mortality ratio, the adjusted increase is 21 percentage points.

In the final column, we modify the simulation further to give both races the same percent reduction in deaths for each cause within 500 gram weight groups. This eliminates changes in mortality ratios that would arise due to differential reductions in mortality for a given cause, for example because access to care differs. The only reason mortality changes differ in this scenario is because the causes of death differ for black and white babies, and relatively more progress is made on some causes of death. This simulation implies a 12 percentage point increase in the black-white ratio of infant mortality. It is this aspect of rising mortality ratios, the part that arises from induced

innovation, that we will explore with the model and empirical analysis that follow. The difference between the 21 percentage point growth in black-white mortality ratios and this 12 percentage point increase, or 9 percentage points, represents changes that occurred because of differential reductions in cause-specific mortality. That is, for some causes of death, the rate at which infant deaths fell was faster for whites than for blacks.

A faster survival gain for whites may relate to several underlying factors. If a disease manifests itself differently among white versus black infants, then efficacy of treatment may vary across races because induced innovation focuses more on the majority group, whites. If differential efficacy of treatments occurs, then we have underestimated the role of induced innovation by ignoring this portion of the rising mortality ratio, since we have no way to disentangle this from two other potential sources of differential survival gains: access and quality of care. Differences in survival gains might also reflect differential access to care, or differential quality of care conditional on access. These latter two explanations do not relate to induced innovation.

Causes of death

For each infant who dies, the LBID data reports an underlying cause of death. We use this to calculate death rates for specific conditions, separately by race. The causes of death are grouped into categories based on an NCHS categorization of the *International Classification of Diseases -9th Revision* (ICD-9) codes (the 61 Cause Recode); the data appendix has details, including a full list of the condition categories. After making adjustments to the causes, we identified 69 independent conditions, and one residual category.

Death rates by leading cause of death are shown in Table 2a. To highlight the role of differing causes within birth weights, we show mortality rates as if blacks and whites had the same distribution of births in each weight category. The 14 conditions listed in the table account for half of all infant deaths in 1983-85. The top three causes, Sudden Infant Death Syndrome (SIDS), RDS, and congenital heart anomalies (heart defects), stand out in magnitude, accounting for nearly 30 percent of deaths. SIDS primarily occurs outside of the hospital setting and affects children of all birth weights. The other causes are predominant in low birth weight babies.

The ranking of the conditions is different for blacks and whites. Respiratory conditions tend to be a greater cause of death for whites than blacks. In contrast, issues that arise because of conditions at the time of delivery such as birth asphyxia, or complications relating to the placenta or umbilical cord are more likely to cause deaths among blacks. For example, RDS is the second most important causes of death for whites (accounting for 9.1 percent of deaths), but the third most important cause for blacks (accounting for 5.6 percent of deaths). Among low birth weight infants, death rates due to RDS in 1983-85 (14 per 1000) were over 50% higher than they were for black infants (9 per 1000), as table 2b shows. Thus, any advance in RDS led to more rapid reductions in infant mortality among whites.

Measures of Innovation

We construct two measures of innovation related to infant conditions: the number of NIH grants associated with each condition, and the number of peer-reviewed journal publications associated with each condition. Each of these has been used as a measure of

innovation in past studies. The grants data come from the Computer Retrieval of Information on Scientific Projects (CRISP) database of biomedical research grants, maintained by the National Institutes of Health. Each entry includes a thesaurus of key words, allowing us to search for relevant grants. A complete list of our search terms is available from the authors upon request. In cases when the use of multiple search terms returned duplicate grants, we removed duplicates from the final counts. We created counts of grants for two periods, 1975-82 and 1983-98, to capture new innovative effort during our study period as well as earlier research that may have produced clinically useful results between 1983 and 1998.[6]

The publications data come from the MEDLINE database of medical journal articles, maintained by the National Library of Medicine. This database has an index of hierarchical subject headings, so identifying articles that are relevant to a particular cause of death can be accomplished by searching on the appropriate headings, when available. First, we identified subject headings denoted as "major" topics, and searched on these major topics that closely matched causes of death.[7] In some cases, there were no "major" topic subject headings matching the cause of death (or category) sufficiently, so we searched for terms in the titles and abstracts of articles. A complete list of our search commands is available from the authors upon request. For all causes, we counted articles that were published during the study period (1983 to 1998).

[6] We elected to start counting grants in 1975 due to data availability at the start of this study (at that time data were not available earlier), and because we felt that eight years offered ample time for grant activity to begin to disseminate through various channels. In practice, our results show little sensitivity to the choice of time period, except that earlier grants are somewhat stronger predictors of mortality changes compared with later grants.

[7] We identified the appropriate headings by searching the database of subject headings for terms from the NCHS categories and ICD-9 entries.

For many of the conditions we study, peer-reviewed publications reflect innovation that has already occurred, since they tend to summarize evidence from completed clinical trials. In some cases, as in the case of treatments for premature infants, multi-site trials may actually affect a large share of the relevant population. Thus, our measure of innovation likely captures innovation that occurs just before and during our period of study.

In some cases, categories are too broad to identify a relevant subject category for grants or publications (e.g., "viral diseases," or "remainder of diseases of respiratory system"). In the absence of a subject heading that captured a given condition, we did not include that grant or article. Out of 69 possible categories (not counting the residual category), we successfully constructed grants counts for 49 conditions and journal article counts for 41 conditions. The conditions with both measures account for over 85 percent of deaths not in the residual category, or 66 percent of all deaths, in the initial period. Over the 1983-98 time period, the mean number of grants per condition was 136, with an interquartile range of 34 to 156. Journal articles are more numerous than grants. The average condition had 1,315 journal articles devoted to it over the 1983-98 time period, with an interquartile range of 336 to 1,810.

IV. Testing for Induced Innovation

In this section, we test the predictions of the induced innovation model. We start with the prediction that initial death rates and subsequent research should be related. We estimate equations of the form:

$$r_i = \alpha_0 + \alpha_1 d_i^0 + \varepsilon_i \qquad (6)$$

Figure 4 shows the relation between initial mortality rates and research graphically. The upper panel shows that conditions with higher mortality rates in 1983-85 have more journal articles devoted to them in the subsequent 15 years. The lower figure shows that the same is true about the number of NIH grants. In both cases, a good part of the line is defined by conditions with very high mortality rates. That is not necessarily problematic, although we examine the sensitivity to this in our results below.

The magnitude of these relations, and potential other correlates, are shown in Table 3. The first column shows the relation graphed in figure 4(a). Each death per 1,000 births due to a particular cause in 1983-85 is associated with nearly 500 NIH grants on that disease from 1983 to 1998. The second column explores the sensitivity of this result to SIDS, RDS, and heart defects. Without those three conditions, the coefficient estimate is still positive, but smaller and not statistically significant. At least some of our results are related to the fact that very big causes of death get more research effort. Since there is no obvious reason to exclude these causes of death from our analysis, our subsequent findings include those data.

The third column shows that each death per 1,000 births is associated with over 2,700 articles in the 1983-98 time period. As with the grant data, the coefficient falls and is no longer statistically significant when SIDS, RDS, and heart defects are excluded. As the fifth, sixth, and seventh columns show, the initial death rate is proxying for grant activity – generally with a lag. Every additional grant in the 1975-82 time period is associated with eight additional articles in the 1983-98 time period. More recent grants have a smaller impact on articles, although this may be a function of the specific timing of grants in this interval.

The second part of the analysis looks at the impact of initial mortality and research on subsequent mortality changes. The theory predicts that declines in mortality should be negatively correlated with higher initial mortality rates, and that this relationship should be mediated by research activity. To look at the relation between initial mortality and subsequent mortality changes, we estimate equations of the form:

$$\ln(d_i^1 / d_i^0) = \beta_0 + \beta_1 d_i^0 + \varepsilon_i \qquad (7)$$

Because of the heteroskedasticity induced by very low mortality rates, we express the dependent variable in logarithms. We also weight the regressions by the theoretical standard error.[8]

To control for changes in the birth weight distribution over time, we measure both initial and final mortality using a constant birth weight distribution, equal to the birth weight distribution at baseline in 1983-1985.

Taken literally, our theoretical model implies $\beta_0 = 0$; a condition with no deaths should have no research, and hence no change in the death rate. In reality, the constant term captures a variety of departures from the model, such as a reduction in overall mortality risk over time that results independent of research (due to improved nutrition, for example), innovations that affect all conditions, and spillovers in research across conditions. Also, to the extent that research allocation is non-optimal (for example, due to political pressure) or the f_i are not equal, β_1 would be diminished and β_0 would differ from zero.

[8] In particular, we weight the regressions by the inverse of the standard error of the dependent variable, the ln change in mortality. This is calculated using the Delta method and the asymptotic variances of the individual mortality rates.

Figure 5 shows the relation between initial mortality and the decline in subsequent mortality. There is a negative relationship between the two, again with a large component played by SIDS, RDS, and heart defects. The first column of Table 4 shows the corresponding regression coefficient. Across all 69 conditions, initial mortality is negatively and statistically significantly related to mortality changes. The coefficient on initial mortality implies that each additional death per 1,000 births is associated with a 20 percent greater mortality reduction. The constant term is negative and implies a 31 percent reduction in mortality over time. The relatively large magnitude indicates that induced innovation is not the only effect in the data. As the second column shows, the coefficient on initial mortality is actually larger excluding SIDS, RDS, and heart defects, but the standard errors are larger as well. The big causes of death are a large part of our story.

The theory suggests that the relation between initial mortality and subsequent mortality declines will be mediated by the amount of research devoted to the disease. We test this in the next columns. The third column shows the same regression as in column (1), but limited to the 41 conditions for which we have journal article data. The coefficient on initial mortality is negative, although smaller than the corresponding coefficient in column (1). The fourth column shows very little relationship between articles and changes in mortality. The coefficient on journal articles is near zero and insignificant, and the coefficient on initial mortality remains negative.

The next two columns show the analogous results using research grants as the measure of innovation. Initial mortality rates are negatively related to changes in mortality, and this relationship is mediated by the number of research grants. Controlling

for the number of research grants, the initial mortality rate is positively related to subsequent mortality changes, although not statistically significantly so.

Grants and research articles are both noisy measures of innovation. Assuming the errors in each are uncorrelated, we can use one to instrument for the other and obtain more efficient estimates. The last column of the table instruments for journal articles with the number of research grants.[9] The coefficient on research articles is now large and negative, and the coefficient on initial mortality is positive. The constant is much lower as well; more of the results are explained by the number of journal articles related to the condition.

The results in Table 4 permit two estimates of the magnitude of induced innovation. One estimate comes from the constant term. The difference between the average rate of mortality reduction and the constant term in equation (7) indicates the average mortality reduction due to induced innovation. The second measure is the change in predicted final death rates attributable to the relation between initial mortality and subsequent mortality. Because of the non-linear model,[10] the first estimate understates the inducement effect, while the second overstates it.

The constant term in the first column of Table 4 implies a decline of 31 percent in mortality (i.e. $-.31 = e^{-.374}-1$) compared with the total reduction in the death rate of 37 percent. This suggests that about 6 percent, or one-sixth of the actual improvement in mortality is due to induced innovation. The contribution of induced innovation implied by the constant term in column 7, the instrumental variables regression that includes

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[9] The F-statistic on the instrument is 4.43.

[10] In a linear model, these would be the same, but our model is not linear. The regression equation implies $E[d_i^1] = d_i^0 \exp(\beta_0) \exp(\beta_1 d_i^0)$, so the relative change $d_i^1 / d_i^0 - 1$ equals $\exp(\beta_0) \exp(\beta_1 d_i^0) - 1$. The two measures of induced innovation are thus $D^1/D^0 - [\exp(\beta_0) - 1]$, the impact calculated based on the constant term, and $E[\exp(\beta_1 d_i^0)] - 1$, the impact of the slope coefficient.

research articles devoted to the condition, is even larger. Using the estimate of β_1 to calculate predicted mortality, in contrast, generates a 7.5 percent decline in mortality (i.e. $E[\exp(\beta_1 d_i^1)]$-1), about one-fifth of the actual change.[11] In column 7, using the implied change in mortality from the coefficient on research articles, the magnitude of the role for induced innovation rises to two thirds of the change.[12] Conservatively choosing estimates that are closer to our lower bound, we estimate that about one fifth of the reduction in infant mortality over time is a result of induced innovation.

The role of competing risks

One potential concern about our estimates is the possibility of competing risks. If vulnerable infants saved from one disease are still likely to die of another, decreased mortality for one cause of death will not result in as great a reduction in total mortality. Alternatively, if research in one disease leads to progress in another, we will understate the impact of research on improved outcomes. However, infants differ from adults in the sense that, unlike older adults who are at risk of multiple conditions (hypertension, high cholesterol, and diabetes are all related to obesity, for example), infants saved from one condition are much less likely to develop other conditions. In contrast to older adults, infants face lower risks of disease incidence as they age, mitigating the disease risk problem.

To test for competing risks though, we return to the RDS example. RDS is important both because of its contribution to the overall evaluation and because respiratory distress is so central to infant mortality. We take advantage of the time series

[11] For this estimate, the death rate for the residual category was held constant.

[12] For this estimate, the death rates for all categories without publications data were held constant. The implied change in mortality is a 24 percent decline.

variation to look for competing risks. If a substantial portion of infants in low birth weight groups who died from RDS were at risk from other diseases as well, we would expect years with large drops in the RDS death rate to have smaller reductions in deaths from other causes.

Figure 6 shows annual mortality change for RDS and non-RDS causes among births 1000-1499 g. The results are, if anything, the opposite of the competing risk explanation. In years where RDS mortality fell most – especially 1990, the year of widespread diffusion of surfactant – non-RDS mortality fell as well; the correlation between mortality changes for RDS and all other causes is 0.34. These results suggest no issue of competing risks. If anything, it appears that as a major cause of death like RDS falls, the innovations that contribute to this fall may reduce deaths from other causes as well. One example of this would be bronchopulmonary dysplasia, a condition related to extended periods of mechanical ventilation in premature infants. With the advent of surfactant, time on ventilation fell, thus reducing the incidence of bronchopulmonary dysplasia (Soll 1998). Other NICU technologies developed in the treatment of infants with RDS could also benefit infants with unrelated conditions. So, for example, improved ventilation techniques developed in response to the wave of RDS infants, might also benefit other infants on ventilators. To the extent that such spillovers exist, our estimates of induced innovation yield a lower bound, since they only capture the differential mortality reductions that occur across causes of death receiving more research effort, and not any potential spillover from induced research effort.

Is Innovation Race Neutral?

In our model, medical innovation is race-neutral: doctors and research funders seek to reduce the major causes of death, whether they affect blacks or whites. This may not be right, however. For example, research might be tilted toward conditions that whites suffer from, ignoring conditions that are common among blacks.

One way to test this is to differentiate black and white deaths in the equation for subsequent mortality changes. Consider equation (8), an expanded version of equation (7):

$$\ln(d_i^1 / d_i^0) = \beta_0 + \beta_1 dw_i^0 + \beta_2 db_i^0 + \varepsilon_i \qquad (8)$$

where *dw* and *db* refer to race-specific deaths as a share of all births. Thus, *dw* and *db* are not standard death rates since the race-specific number of deaths are divided by the sum of black and white births. One can thus view *dw* as capturing the death rate from the bundle of causes that kill white infants and *db* as capturing the death rate from the bundle of causes that kill blacks. A theory of racially biased innovation suggests that black deaths should count less than white deaths, i.e., $|\beta_2| < |\beta_1|$.

Table 5 shows results of regressions separating black and white deaths. The first three columns present results for the impact of initial mortality on subsequent changes in mortality. Independently, black deaths count more than white deaths for subsequent mortality changes (column 1 versus column 2), although the standard errors on each are large. The regression has difficulty determining the relative weight to put on the two when included in the regression jointly (column 3), but the coefficient on black deaths is negative while the coefficient on white deaths is positive. However these coefficients are not statistically different from each other.

The next columns show the relation between race-specific initial mortality and the number of journal articles and NIH grants. When included together, white deaths are more associated with journal articles than are black deaths, but black deaths are more associated with NIH grants. In these regressions the coefficients are statistically different, but their magnitudes suggest a co-linearity problem. Overall, we find no consistent pattern of race-based bias in the innovative process.

Induced Innovation and Social Inequality

As shown in our model, induced innovation can have an unintended consequence on disparities in health outcomes. In the model, greater gains in survival occur for causes of death that are relatively more common among the majority group. Thus, the overall disparity of health outcomes widens. As discussed in section II, this result arises mechanically when the difference in initial death rates across races (majority – minority) are positively correlated with survival gains. We test for evidence of this positive correlation empirically, by correlating changes in condition-specific death rates to the difference in initial shares of death for majority and minority groups. The correlation of 0.08 is indeed positive and statistically significant.[13]

The overall impact of this correlation on subsequent inequality changes is shown in Table 1. The last column of the table shows the simulated mortality rates if mortality for each cause declined at the same rate for whites and blacks.[14] In this scenario, the black-white ratio would change only because white and black babies die of differing causes. As the table shows, the black-white infant mortality ratio still rises by 12

[13] Here we control for birth weight by calculating death rates and survival gains within 500-gram intervals and then taking a weighted average based on the number of births in each interval.

[14] As in the preceding column, the analysis is conditional on birth weight.

percentage points in this scenario. Thus, about one-third of the increase in the black-white mortality disparity, (.12/.30), is a result of induced innovation. Another one-third, (.21-.12)/.30, results from the differentially greater reduction in mortality for whites relative to blacks within causes. The remaining one-third, (.30-.21)/.30, is a result of differential growth of low birth weight babies among blacks. Notably, the magnitude of rising black-white mortality ratio is comparable to that of the role of induced innovation in mortality reductions documented earlier. In other words, black-white infant mortality ratios in the US are responsive to induced innovation, growing with induced improvements in survival for infants.

A Falsification Exercise: Education-Based Disparities

One concern about our findings is that we may be measuring the impact of being in an economically disadvantaged group, not necessarily a minority population group. It may be that the economically disadvantaged are doing worse over time, regardless of whether they are majority or minority groups. Since blacks are both a population minority and economically disadvantaged, we cannot completely differentiate between these theories with our data.

We test this using data on deaths by maternal education, restricting the sample to non-Hispanic whites. Women with some college education represent a minority of births (40 percent), but are better off economically. Thus, if the effect we find is a result of economic disadvantage, babies of women with some college should fare better over time than babies of women who never attended college. If the result is due to population size, babies of women who attended college should fare no better than babies of less educated

women. Table 6 displays numbers similar to those in table 1, but dividing mothers into groups based on college attendance. As the second column of the table shows, women who never attended college have a 53 percent higher mortality rate in 1983-85 than women who attended some college. That ratio increased to 88 percent in 1996-98.

About one-third of that increase is a result of adverse trends in the birth weight distribution for women who did not attend college relative to women who did. Our model does not explain such effects. The entirety of the remainder is a result of more rapid declines in mortality within cause for women with some college education. Indeed, as the last column shows, there is no increase in the mortality ratio, or if anything a slight decrease, when mortality reductions by cause are assumed to be the same. Thus, our results do not suggest that the causes of death predominant among the economically advantaged are declining by more than the causes among the economically disadvantaged. Indeed, the two seem about the same.

The reason why women with some college education benefitted more from progress within causes of death than women without any college education is not clear. It may reflect differences in access to care or the quality of that care, which we do not explore. Overall, however, these results support the conclusion that it is induced innovation resulting from minority status itself that leads to less rapid mortality declines for blacks, and hence lagging health outcomes.

VII. CONCLUSIONS

The dynamics of the medical sector have been a subject of much debate. Most of medical care cost increases are a result of technological progress (Newhouse, 1992), and

some studies suggest that health benefits emanate from the same source (Cutler, 2004; Cutler, Rosen, and Vijan 2006).

For at least the last two decades, economists have speculated about the underlying source of these technological innovations (Weisbrod, 1991). The idea that progress is a result of perceived need has been common in the literature, although not tested to any great extent. We test the importance of induced innovation by considering care for a group that is particularly needy: low birth weight infants.

We reach two primary conclusions. First, there is a strong impact of induced innovation on technological change. Disease conditions with higher initial mortality rates had more grant effort devoted to them, saw more journal articles about them, and experienced a greater reduction in subsequent mortality. Induced innovation explains about 20 percent of the reduction in mortality over time.

But endogenous technology also benefits majority groups over minority groups. Majority groups are of necessity a larger share of total deaths than are minority groups. Thus, conditions that affect them more will receive more research attention. Our results show that this leads to a significant increase in the disparity between blacks and whites as innovation allows premature white newborns to "catch up" to their black counterparts, who, for a given gestation and birth weight, tend to have better health.

Our results do not arise because medical research is racially biased; our best estimates suggest that it is not. Rather, growing disparities result from the seemingly benign tendency of 'treating what we see'. If we want disparities to fall over time, our results suggest that we will need to treat based on factors beyond the headline numbers.

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Appendix

Cause of Death Data

We start with cause of death as identified on ICD-9 forms and tabulated by the National Center for Health Statistics. We then modify this in several ways. First, we formed 10 additional categories by breaking apart two NCHS categories that grouped together distinct conditions with over 100 deaths in 1983-85: 770.xx and codes 775.2-775.9. We also formed five categories by identifying conditions within the NCHS residual group with over 250 deaths in this period, based on ICD-9, 3-digit codes. In addition, we moved four of the NCHS categories into a residual category because they do not identify a specific condition, but are rather residual catchall categories (e.g., "...unspecified," or "all other").

Specifically, our modifications from the NCHS 61 Cause Recode were: the category for "other respiratory conditions of newborn" (ICD-9 code 770) was subdivided into congenital pneumonia (770.0), massive aspiration syndrome (770.1), interstitial emphysema and related conditions (770.2), pulmonary hemorrhage (770.3), primary atelectasis (770.4), other and unspecified atelectasis (770.5), and chronic respiratory disease arising in the perinatal period (Bronchopulmonary dysplasia, Wilson-Mikity syndrome) (770.7); "all other and ill-defined conditions originating in the perinatal period" (codes 775.2-775.9, and 776.1-779) was subdivided into disseminated intravascular coagulation in newborn (776.2), necrotizing enterocolitis in fetus or newborn (777.5), and hydrops fetalis not due to isoimmunization (778.0); and categories were created for disorders of fluid, electrolyte, and acid-base balance (276),

cardiomyopathy (425), primary pulmonary hypertension (416.0), cardiac arrest (427.5), and renal failure, unspecified (586). The final 69 categories are shown in Table A1.

Table A1. Cause of Death Categories

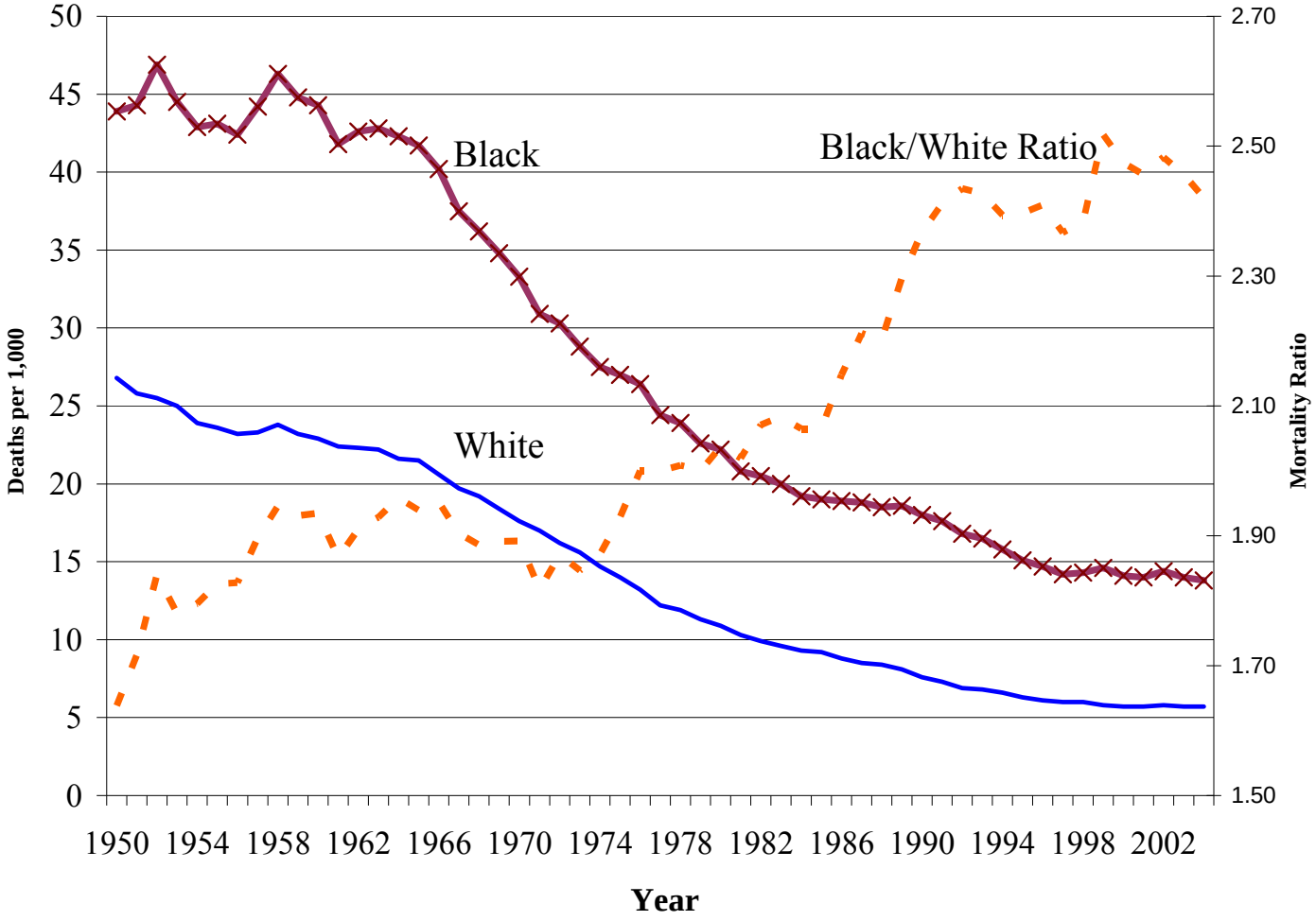
ICD-9 Codes	Condition(s)	Articles* 1983-98	NIH Grants* 1983-98	NIH Grants* 1975-82
001-007, 010-032, 034-035, 037, 039-041, 042-044, 080-088, 091-139	Remainder of infectious and parasitic diseases	a	a	a
008-009	Certain intestinal infections	b	172	38
033	Whooping cough	b	113	17
036	Meningococcal infection	b	b	b
038	Septicemia	2466	160	94
045-079	Viral diseases	a	a	a
090	Congenital syphilis	b	23	3
140-208	Malignant neoplasms, including neoplasms of lymphatic and hematopoietic tissues	a	a	a
210-239	Benign neoplasms, carcinoma in situ, and neoplasms of uncertain behavior and of unspecified nature	a	a	a
254	Diseases of thymus gland	b	b	b
276	Disorders of fluid, electrolyte, and acid-base balance	1640	145	62
277	Cystic fibrosis	b	224	158
280-289	Diseases of blood and blood-forming organs	3689	30	8
320-322	Meningitis	1630	223	48
323-389	Other diseases of nervous system and sense organs	a	a	a
416	Primary pulmonary hypertension	336	277	54
425	Cardiomyopathy	924	17	0
427.5	Cardiac arrest	287	77	23
460-465	Acute upper respiratory infections	a	a	a
466, 490-491	Bronchitis and bronchiolitis	694	186	109
480-486	Pneumonia	1810	351	107
487	Influenza	b	156	61
470-478, 492-519	Remainder of diseases of respiratory system	a	a	a
520-534, 536-543, 562-579	Remainder of diseases of digestive system	a	a	a
535, 555-558	Gastritis, duodenitis, and noninfective enteritis and colitis	b	0	0
550-553, 560	Hernia of abdominal cavity and intestinal obstruction without mention of hernia	b	b	b
586	Renal failure, unspecified	1088	120	68
740	Anencephalus and similar anomalies	681	31	52
741	Spina bifida	777	153	69
742.3	Congenital hydrocephalus	1301	88	60

ICD-9 Codes	Condition(s)	Articles* 1983-98	NIH Grants* 1983-98	NIH Grants* 1975-82
742.0-742.2, 742.4-742.9, 743	Other congenital anomalies of central nervous system and eye	b	52	0
745-746	Congenital anomalies of heart	8334	364	375
747	Other congenital anomalies of circulatory system	2525	147	61
748	Congenital anomalies of respiratory system	1137	64	5
749-751	Congenital anomalies of digestive system	2717	27	3
752-753	Congenital anomalies of genitourinary system	980	8	0
754-756	Congenital anomalies of musculoskeletal system	2047	34	12
758	Down's syndrome	1242	153	156
758.1-758.9	Other chromosomal anomalies	474	74	1
760	Newborn affected by maternal conditions which may be unrelated to present pregnancy	b	b	b
761	Newborn affected by maternal complications of pregnancy	1144	119	25
762	Newborn affected by complications of placenta, cord, and membranes	419	87	3
763	Newborn affected by other complications of labor and delivery	b	34	0
764	Slow fetal growth and fetal malnutrition	b	b	b
767	Birth trauma	1815	74	47
768.2-768.4	Fetal distress in liveborn infant	289	101	38
768.5-768.9	Birth asphyxia	1161	19	1
769	Respiratory distress syndrome	2803	776	509
770	Congenital pneumonia	26	3	3
770.1	Massive aspiration syndrome	256	3	7
770.2	Interstitial emphysema and related conditions	319	42	12
770.3	Pulmonary hemorrhage	86	37	2
770.4	Primary atelectasis	73	37	13
770.5	Other and unspecified atelectasis	b	b	b
770.7	Chronic respiratory disease arising in the perinatal period (Bronchopulmonary dysplasia, Wilson-Mikity syndrome)	879	621	29
771	Infections specific to the perinatal period	1162	125	37
772	Neonatal hemorrhage	825	150	54
773-774	Hemolytic disease of newborn, due to isoimmunization, and other perinatal jaundice	2287	55	54
775.0-775.1	Syndrome of "infant of a diabetic mother" and neonatal diabetes mellitus	b	b	b
776	Hemorrhagic disease of newborn	153	34	20
776.2	Disseminated intravascular coagulation in newborn	106	23	10
777.5	Necrotizing enterocolitis in fetus or newborn	624	163	28
778	Hydrops fetalis not due to isoimmunization	1	2	4
798	Sudden infant death syndrome	2703	673	387
E911-E912	Inhalation and ingestion of food or other object causing obstruction of respiratory tract or suffocation	c	c	c
E913	Accidental mechanical suffocation	c	c	c

ICD-9 Codes	Condition(s)	Articles* 1983-98	NIH Grants* 1983-98	NIH Grants* 1975-82
E800-E910, E914-E949	Other accidental causes and adverse effects	c	c	c
E967	Child battering and other maltreatment	c	c	c
E960-E966, E968-E969	Other homicide	c	c	c

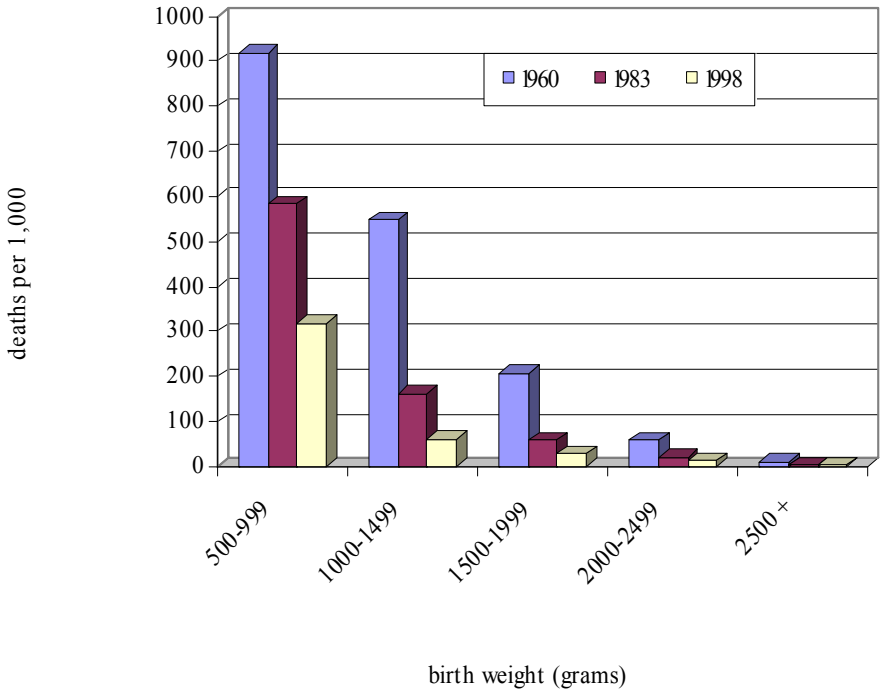
* Data are not available for all categories. See the discussion on page 19 for further information. Specific reasons for the absence of data are as follows:
a. Overly broad category.
b. Lack of appropriately targeted search terms. In some cases, in the MEDLINE database, we were unable to effectively restrict searches to infant conditions despite the presence of an "infant" qualifier.
c. Not a medical condition.

Figure 1: Infant Mortality by Race, 1950-2004



Source: Data are from Vital Statistics of the United States

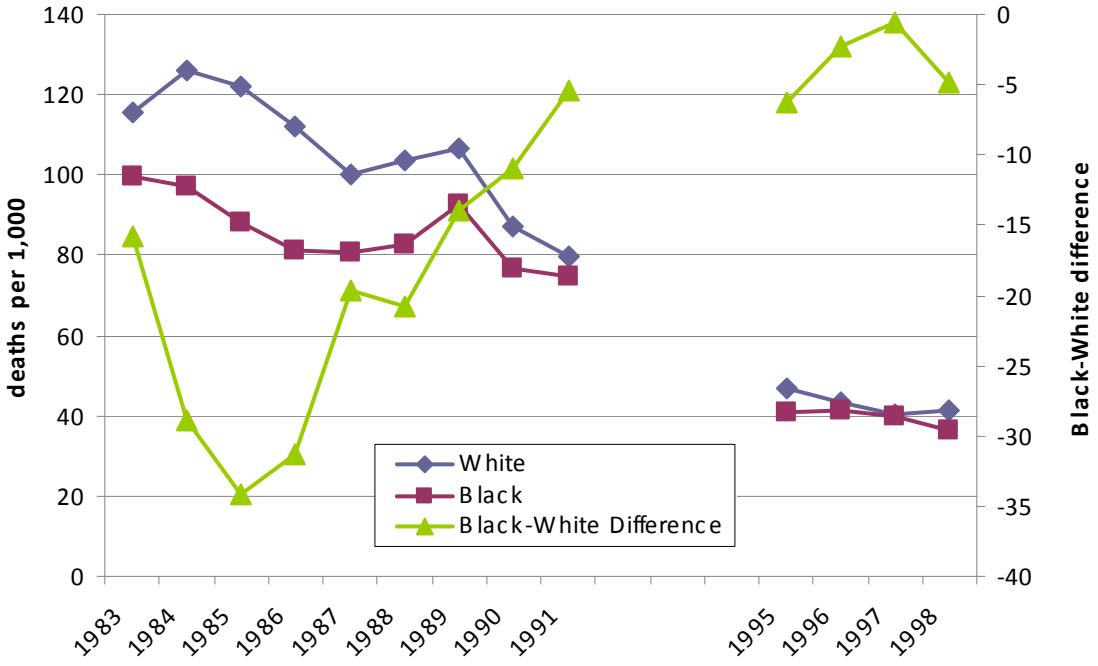
Figure 2: Infant Mortality by Birth Weight, 1960, 1983, and 1998



Note: Data for 1960 do not present all birth weights. Infant mortality rate for births below 1,000 grams are assumed to be for infants weighing 500-999 grams.

Figure 3: The RDS Example

(a) 500-999 gram babies



(b) 1,000-1,499 gram babies

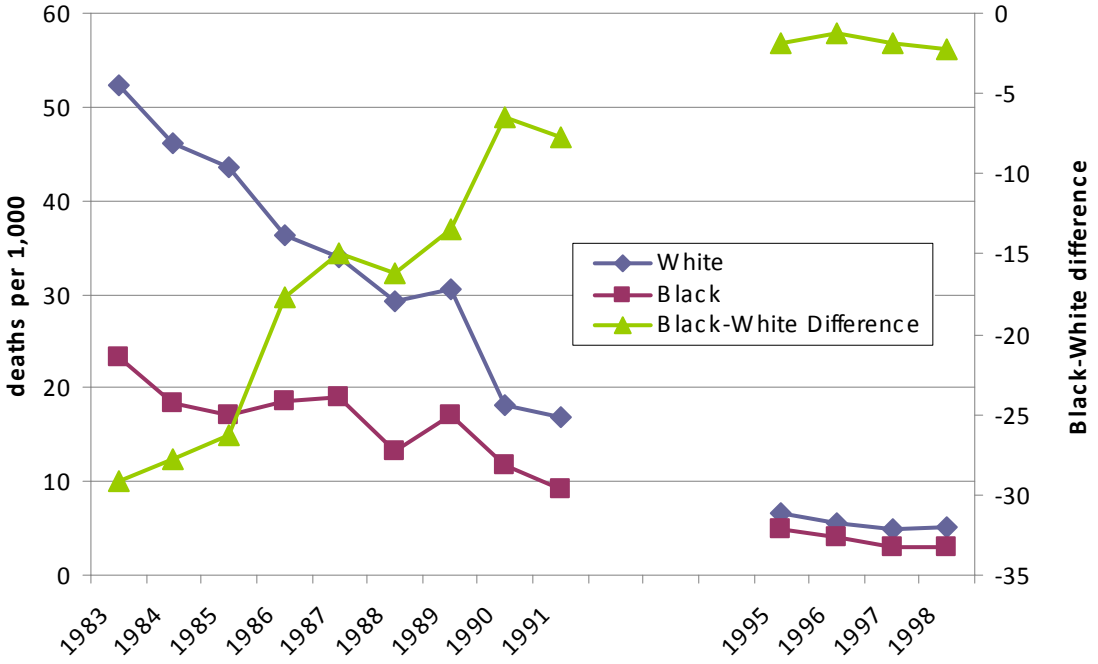
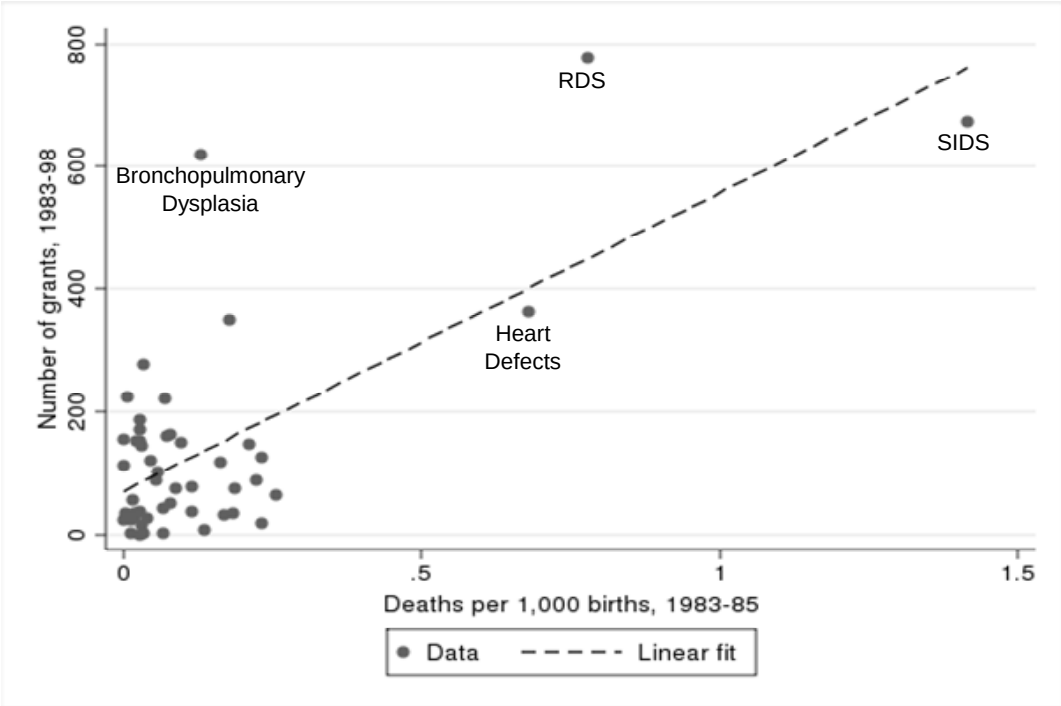


Figure 4: Relationship between initial mortality rate and research effort

Panel A: Number of NIH grants, 1983-98



Panel B: Number of journal articles, 1983-98

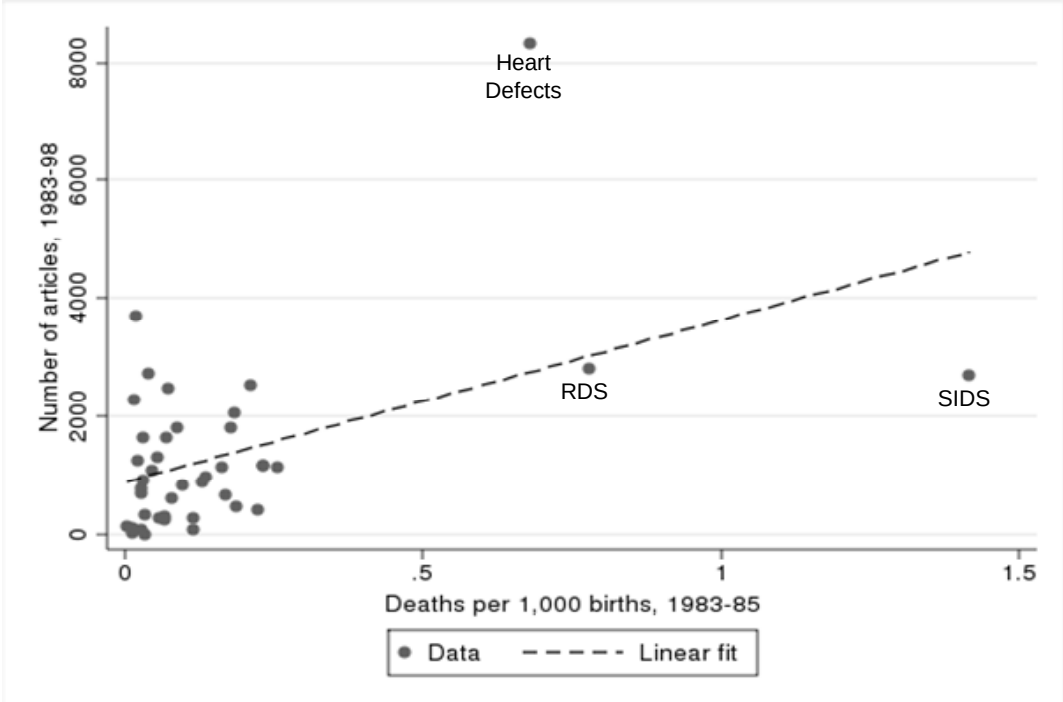


Figure 5: Relationship between initial mortality rate and declines in death rates

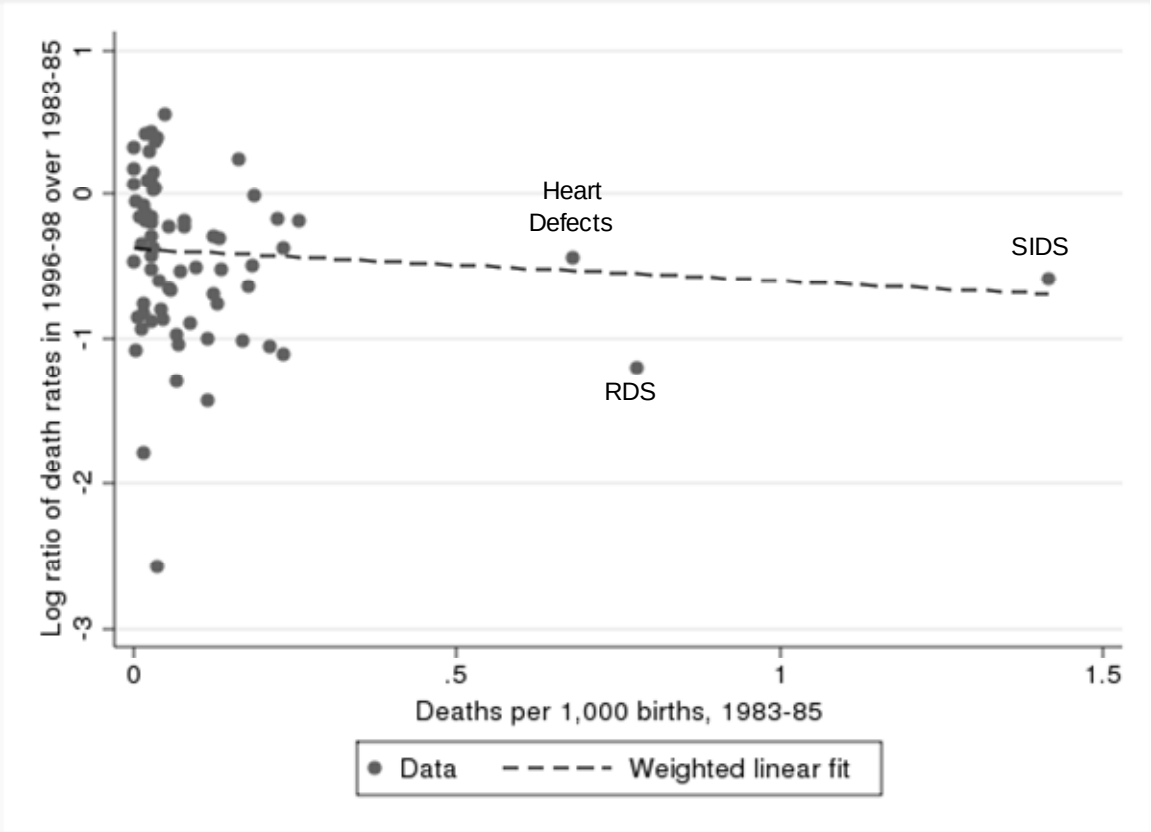


Figure 6: Annual Change in RDS and Other Mortality

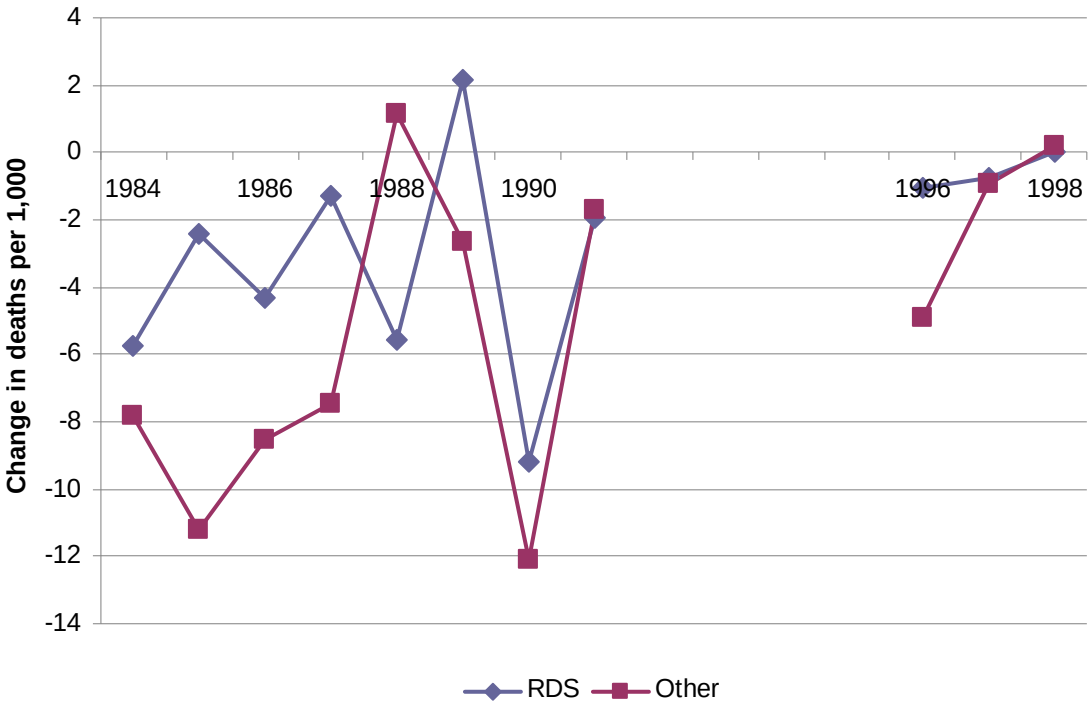


Table 1: Actual and Simulated Change in Racial Disparity

	1983-85		1996-98		
Race	Share of Births	Infant Mortality Rate	Actual Infant Mortality Rate	Holding birth weight constant	Holding birth weight constant and same change in cause specific mortality rates
White	84%	8.2	5.2	5.0	5.0
Black	16%	17.1	12.4	11.4	11.0
Black/White		2.09	2.40	2.30	2.21
Change in ratio			0.30	0.21	0.12

Note: Data are based on the linked birth-death infant data. Deaths are per 1,000 births. In the 1996-98 columns, the second column is a simulation showing changes in infant mortality if the birthweight distribution were the same in that time period as in the 1983-85 time period, separately by race. The third column includes that assumption and also simulates the same change in infant mortality rates by cause for blacks and whites. The decline for both races is assumed to be the rate observed for whites.

Table 2a: Leading causes of death among infants, all births (1983-85)

	All Births								
Cause of death (ICD-9)	All races	Rank	Share	Whites	Rank	Share	Blacks	Rank	Share
Sudden infant death syndrome	1.42	1	15%	1.29	1	13%	1.95	1	18%
Respiratory distress syndrome (RDS)	0.78	2	8%	0.89	2	9%	0.60	3	6%
Congenital anomalies of heart	0.68	3	7%	0.71	3	7%	0.61	2	6%
Congenital anomalies of respiratory system	0.26	4	3%	0.30	4	3%	0.17	9	2%
Birth asphyxia	0.23	5	2%	0.22	9	2%	0.29	4	3%
Infections specific to the perinatal period	0.23	6	2%	0.24	6	2%	0.23	7	2%
Newborn affected by complications of placenta, cord, and membrane	0.22	7	2%	0.26	5	3%	0.19	8	2%
Other congenital anomalies of circulatory system	0.21	8	2%	0.20	11	2%	0.27	6	3%
Other chromosomal anomalies	0.19	9	2%	0.23	7	2%	0.11	12	1%
Congenital anomalies of musculoskeletal system	0.18	10	2%	0.21	10	2%	0.11	11	1%
Pneumonia	0.18	11	2%	0.15	14	2%	0.28	5	3%
Anencephalus and similar anomalies	0.17	12	2%	0.23	8	2%	0.06	14	1%
Newborn affected by maternal complications of pregnancy	0.16	13	2%	0.20	12	2%	0.12	10	1%
Congenital anomalies of genitourinary	0.14	14	1%	0.17	13	2%	0.07	13	1%
All other causes	4.72	---	48%	4.50	---	46%	5.74	---	53%

Note: Sample restricted to singleton births that are non-Hispanic. Deaths are per 1,000 births and are adjusted to a common birth weight distribution.

Table 2b: Leading 5 causes of death among infants born under 2500 grams, 1983-85

Cause of death (ICD-9)	All races	Rank	Whites	Rank	Blacks	Rank
Respiratory distress syndrome (RDS)	12.40	1	14.35	1	9.17	1
Suddent infant death (SIDS)	4.24	2	3.70	2	5.27	2
Newborn affected by complications of placenta, cord, and membrane	2.97	3	3.69	3	2.00	5
Congenital anomalies of respiratory system	2.80	4	3.55	4	1.50	9
Infections specific to the perinatal period	2.67	5	2.82	6	2.38	3

Note: Sample restricted to singleton births that are non-Hispanic. Deaths are per 1,000 births and are adjusted to a common birth weight distribution.

Table 3: Regressions of Research Effort and Initial Infant Mortality Rates

	Grants (1983-98)		Journal Articles (1983-98)				
Independent Variable	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Initial mortality	488	135	2748	984	2456	-77	251
per 1,000 births	(73)	(222)	(797)	(1923)	(1142)	(1222)	(1220)
Grants (83-98)	---	---	---	---	0.59	---	-2.68
					(1.63)		(1.76)
Grants (75-82)	---	---	---	---	---	8.24	11.17
						(2.85)	(3.40)
Sample	All	Without SIDS, RDS, heart defects	All	Without SIDS, RDS, heart defects	All	All	All
N	49	46	41	38	41	41	41
R^2	0.484	0.008	0.234	0.007	0.236	0.371	0.409

Note: Each column is a separate regression. Standard errors are shown in ()s. Initial mortality is mortality in 1983-85 for singleton, non-Hispanic births.

Table 4: Regressions of Decline in Infant Death Rate, Initial Mortality, & Research Effort

		No SIDS, RDS, heart					IV model grants and
	All	defects	With article data		With grant data		articles
Independent Variable	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Initial mortality per 1,000 births	-0.222 (0.096)	-0.715 (0.726)	-0.146 (0.116)	-0.149 (0.127)	-0.168 (0.107)	0.374 (0.205)	0.243 (0.315)
Grants (75-98) (100s)	---	---	---	---	---	-0.070 (0.023)	---
Articles (83-98) (1000s)	---	---	---	0.002 (0.028)	---	---	-0.226 (0.144)
Constant	-0.374 (0.067)	-0.285 (0.112)	-0.457 (0.088)	-0.460 (0.098)	-0.432 (0.080)	-0.373 (0.076)	-0.117 (0.261)
N	69	66	41	41	49	49	41
R^2	0.074	0.015	0.039	0.039	0.050	0.207	NA

Each column is a separate regression. Birthweight distribution is held constant using 500 gram intervals. All regressions are weighted by 1/SE(ln-chg). The last column instruments for journal articles with the number of research grants.

Table 5: Testing for Race-Biased Innovation

	Change in Death Rates			Journal Articles			NIH Grants		
Independent variable	(1)	(2)	(3)	(1)	(2)	(3)	(4)	(5)	(6)
Black deaths per 1,000 births (both races)	-0.837 (0.335)	---	-2.515 (1.650)	8089 (3018)	---	-28672 (9660)	1856 (251)	---	2999 (953)
White deaths per 1,000 births (both races)	---	-0.298 (0.134)	0.682 (0.657)	---	3989 (1071)	14484 (3668)	---	648 (103)	-449 (361)
F test for equal coefficients	---	---	1.94 [0.169]	---	---	10.64 [0.002]	---	---	6.98 [0.011]
N	69	69	69	41	41	41	49	49	49
R^2	0.085	0.068	0.100	0.156	0.262	0.401	0.537	0.455	0.552

Note: Each column is a separate regression. Standard errors in ()s and p-values in []s. In columns 1-3, the regression are weighted by 1/SE(ln-chg) and the birthweight distribution is held constant using 500 gram intervals.

Table 6: Actual and Simulated Change in Educational Disparity

	1983-85		1996-98		
Race	Share of Births	Infant Mortality Rate	Actual Infant Mortality Rate	Holding birthweight constant	Holding birthweight constant and same change in cause-specific mortality rates
No College	60%	9.5	5.9	5.7	4.9
College attendee	40%	6.2	3.2	3.3	3.3
Ratio (No college:college)		1.53	1.88	1.73	1.49
Change in ratio			0.35	0.21	-0.03

Note: Data are based on the linked birth-death infant data for white births. Deaths are per 1,000 births. In the 1996-98 columns, the second column is a simulation showing changes in infant mortality if the birthweight distribution were the same in that time period as in the 1983-85 time period, separately by education group. The third column includes that assumption and also simulates the same change in infant mortality rates by cause for both groups. The change is assumed to be the rate observed for the high education group.