THE INCREASING RACIAL DISPARITY IN INFANT MORTALITY: RESPIRATORY DISTRESS SYNDROME AND OTHER CAUSES*

W. PARKER FRISBIE, SEUNG-EUN SONG, DANIEL A. POWERS, AND JULIE A. STREET

Although substantial declines in infant mortality rates have occurred across racial/ethnic groups, there has been a marked increase in relative black-white disparity in the risk of infant death over the past two decades. The objective of our analysis was to gain insight into the reasons for this growing inequality on the basis of data from linked cohort files for 1989–1990 and 1995–1998. We found a nationwide reversal from a survival advantage to a survival disadvantage for blacks with respect to respiratory distress syndrome over this period. The results are consistent with the view that the potential for a widening of the relative racial gap in infant mortality is high when innovations in health care occur in a continuing context of social inequality. As expected, the results for other causes of infant mortality, although similar, are less striking. Models of absolute change demonstrate that among low-weight births, absolute declines in mortality were greater for white infants than for black infants.

Although major absolute declines in infant mortality have occurred across racial/ethnic groups over the past several decades, the relative gap between blacks and whites has persisted. More important for the present purposes, racial disparities have not only persisted but actually *increased*, especially from the 1980s onward. In 1980, the black-white ratios for the infant mortality rate (IMR) and the neonatal mortality rate (NMR) stood at 2.0 and 1.9, respectively, but by 1997, both ratios had risen to 2.3 (Guyer et al. 1998). By 2000, the black-white IMR ratio had risen to 2.4–2.5 (Hoyert et al. 2001; Mathews, Menacker, and MacDorman 2002; Minino et al. 2002). Absolute declines in rates have quite deservedly received considerable attention. More recently, the widening of the relative racial difference in infant mortality has generated increasing concern (Gortmaker and Wise 1997; Guyer et al. 1998; Wise 2003), but explanations for this persistent and, now, growing disparity have remained elusive.

In this analysis, we concentrated on the increasing disparity in the relative risk of infant mortality between the non-Hispanic white (hereafter "white") and non-Hispanic black (hereafter "black") populations. We also estimated models of absolute change in infant mortality. An exclusive focus on relative differences yields an incomplete picture in that it neglects the notable achievement represented by overall reductions in infant mortality.[1] Nevertheless, the primary impetus for our study can be found in the conclusion by

*W. Parker Frisbie, Population Research Center, 1 University Station, G1800, University of Texas at Austin, Austin, TX 78712; E-mail: frisbie@prc.utexas.edu. Seung-eun Song, Daniel A. Powers, and Julie A. Street, University of Texas at Austin. We gratefully acknowledge the support for this analysis provided by the National Institute of Child Health and Human Development under Grant RO1 HD41147. We are also grateful for the computational assistance of Starling G. Pullum and for the careful reading and helpful suggestions provided by our colleague, Robert A. Hummer, anonymous referees, and the editor.

1. We acknowledge the insight provided by editorial and reviewer comments pointing out the need for (and benefit of) modeling the absolute black-white gap.

Wise (2003:343) that "[t]he analytic challenge for any assessment of disparities in infant mortality . . . is not merely to document that disparities exist but rather to explain why they persist in the face of enormous reductions in absolute levels in mortality."

RECENT CHANGES IN THE STRUCTURE OF INFANT MORTALITY

In a recent review, Gortmaker and Wise (1997:152) argued that "the past two decades have witnessed the most profound alterations ever recorded in the structure of infant mortality patterns in the United States." Among the most prominent changes are the following. Medical advances have made it increasingly possible for women to deliver live infants under conditions that previously would have led to stillbirths. At least a decade ago, infants born as early as 22 weeks gestation were being resuscitated (Sowards 1997; see also Hack and Fanaroff 1993). This, along with the increase in the incidence of plural births, is likely a major part of the reason that rates of compromised birth outcomes (i.e., low-weight and preterm births) increased from the 1980s into the 1990s. However, while rates of low birth weight (LBW) and prematurity have risen among whites and many other racial/ethnic groups, modest *declines* in rates of these adverse outcomes have been recorded for blacks (Demissie et al. 2001; Frisbie and Song 2003).[2] Another largely unanticipated temporal shift is that the well-documented survival advantage of preterm and LBW black infants, compared with their white counterparts (Kline, Stein, and Susser 1989; Wilcox and Russell 1986, 1990), appears to have eroded in recent years (Hamvas et al. 1996; Malloy and Freeman 2000; Ranganathan et al. 2000). The latter trend is especially distressing in that the large and long-standing black-white gap in infant mortality would have been even more extreme in the absence of this "return to risk" advantage for LBW black infants.

A promising place to look for an explanation of the growing black-white disparity in infant mortality is the point at which the interests of social and medical sciences intersect. That is, although the recent "profound alterations in the structure of infant morality" have been fueled by "major technological advances in the clinical management of the high-risk pregnancy and the critically ill new born" (Gortmaker and Wise 1997:155), "disparities in infant mortality are rooted fundamentally in social stratification and the exercise of power" (Wise 2003:342). In short, there is a potential for a widening of the relative gap between black and white infant mortality because social inequalities are likely to translate into differential knowledge of, and access to, preventative and curative innovations (Gortmaker and Wise 1997; Link and Phelan 1995).

A particularly pertinent example with important implications for the overall growth in black-white disparity involves the introduction of pulmonary surfactant therapy—an innovation that has proven to be of substantial efficacy in the treatment of respiratory distress syndrome (RDS). RDS results from a deficiency of naturally occurring surfactant in the lungs of the fetus such that the functioning of the alveoli (air cells in the lungs) may be compromised and gas exchange may fail.[3] It is largely a problem of preterm (or LBW) infants in that prior to 26 weeks gestation, there is usually little or no natural secretion of surfactant (British Columbia Reproductive Care Program 1993; Halliday 1997; Malloy and Freeman 2000).

An expanded investigation of racial disparities in infant mortality that distinguishes RDS from other causes of infant mortality is important for a number of reasons. From

2. One interpretation of the upward inflection in the rates of low-weight and preterm births among white women is that fertility-enhancement procedures have resulted in an increased proportion of multiple births, a phenomenon not observed among black women (Demissie et al. 2001; see also Blondel et al. 2002).

3. RDS, which appears as a single code (769) in the *International Classification of Diseases*, 9th revision, includes pulmonary hyaline membrane disease and idiopathic respiratory distress (*Dorland's Illustrated Medical Dictionary* 1988).

1980 to 1998, RDS ranked as the fourth leading cause of infant death in the United States. Over that period, however, the RDS mortality rate was reduced almost fivefold (U.S. Census Bureau 2001), with much of the reduction strongly linked to the introduction of surfactant replacement about midway through this period (Hamvas et al. 1996; Malloy and Freeman 2000).

The availability of data on black-white variation in the risk of RDS mortality in the periods before and after the approval of surfactant therapy for general use offers an opportunity to gain insight into the possible differential impact of advances in perinatal health care as conditioned on social factors. Following the approval of surfactant replacement by the U.S. Food and Drug Administration (FDA) in August 1990, there was a much greater decline in the rate of infant death from RDS among whites than among blacks (Halliday 1997; Hamvas et al. 1996; Malloy and Freeman 2000; Ranganathan et al. 2000). For example, in 1989, the RDS mortality rate per 100,000 live births was 172.2 for blacks and 74.7 for whites (National Center for Health Statistics 1993: table 2-5)—a rate ratio of 2.3. By 1999, the ratio was greater than 2.7; the IMR from RDS was 61.9 and 22.5 for blacks and whites, respectively (Hoyert et al. 2001: table 28). Thus, the decrease in infant deaths that were associated with RDS has contributed substantially to the overall decline in infant mortality in the past few decades (Ranganathan et al. 2000) and likely to the widening black-white difference in the risk of infant mortality.

Hamvas et al. (1996) examined clinical records from St. Louis–area hospitals concatenated with data from the National Center for Health Statistics (NCHS) linked birth and infant death files and showed that the NMR among white infants with very low birth weight (VLBW; infants born weighing 500–1,500 grams) dropped by 41% between 1987–1989 and 1991–1992. Meanwhile, no significant change was observed among VLBW black infants. Although this comparison of rates over time was accompanied by a logistic regression at the micro level, Hamvas et al. did not evaluate the effects of demographic, social, or economic covariates. The same is true for Ranganathan et al. (2000), who applied logistic regression in an analysis of the NCHS linked files "to approximate the time trends in the likelihood of death over time for each race" for the years 1985, 1988, and 1991 (p. 455), but included no social or economic risk factors (perhaps because of the changes in items on birth certificates after 1988).

Hamvas et al. (1996) found that the black-white rate ratio was actually *reversed* from 0.7 (a black survival advantage) in 1987–1989 to 1.3 (a white survival advantage) in 1991–1992, a result not observed in other research on racial differences in RDS mortality. One reason for this difference may be that relationships of interest are not the same in the St. Louis area as in other geographic locations. Furthermore, the severity of risk of death from any cause, including RDS, may be different when only VLBW infants are considered than when babies born at other or all weights are included in the analysis (as in Malloy and Freeman's 2000 research). In any event, it is unclear whether the kind of reversal from an RDS survival advantage to a survival disadvantage among black infants observed by Hamvas et al. occurred among infants nationwide.

Additional analyses of racial differences in RDS mortality are needed because past research on this issue consists mainly of studies that were based on clinical data from one or a few hospitals in a limited geographic area or studies that, although nationwide, do not adjust for the effects of social risk factors. Both types of research have been informative, but the former is limited in generalizability, and the latter is often descriptive, with a focus on RDS death rates and/or proportion of all infant deaths attributable to RDS.[4] As

4. A large body of research focuses on matters other than racial/ethnic differences, including evaluations of the benefits of surfactant therapy; the efficacy of natural (from animal lungs) as compared with synthetic surfactant; and the effectiveness of other interventions, such as administration of antenatal corticosteroids (American Academy of Pediatrics 1999; Kresh and Clive 1998; Walfisch, Hallak, and Mazor 2001).

far as we can determine, there is little research on racial differences in RDS mortality that involves multivariate modeling of individual risk for the United States as a whole.

One exception is a study by Muhuri, MacDorman, and Ezzati-Rice (2004), which examined cause-specific infant mortality by race/ethnicity between 1989–1991 and 1995–1997 and found substantial declines in risk for several causes, including RDS, sudden infant death syndrome (SIDS), and congenital anomalies. In searching for racial/ethnic differences, these authors found only one major race/ethnicity interaction: a disproportionately large decline in the risk of infant death among American Indians from SIDS and congenital anomalies. However, Malloy and Freeman (2000:420) argued that "the increasing disparity between blacks and whites in RDS mortality should serve as a sentinel event to reenergize efforts to address differences in social, behavioral, cultural, and economic factors that may be contributing to the disparity."[5]

It is also important to determine the extent to which recent trends in all other causes of infant death do or do not parallel the changes in RDS mortality. Although pulmonary surfactant therapy is a prime example of a technological advance that has dramatically improved the survival chances of infants with respiratory ailments and may be a key reason for the erosion of the black LBW and short-gestation survival advantage, there is no suggestion from previous research that this phenomenon can explain more than a fraction (albeit a sizable fraction) of the overall increase in the racial disparity in infant mortality. In the past few decades, many other innovations in neonatal care have emerged, including the continued development of high-tech infrastructure that characterizes neonatal intensive care units in tertiary-care hospitals (Horbar and Lucey 1995; Schwartz et al. 2000) and low-tech innovations, such as the "back to sleep" program that is associated with a reduction in deaths from SIDS (Gershan, Besch, and Franciosi 2002; Gibson et al. 2000).

ANALYTICAL AIMS

The general objective of our analysis was to provide insight into why racial disparities in infant mortality persist and, in fact, have increased while large reductions occurred in the absolute levels of infant mortality. Accomplishing this objective encompasses several specific aims: (1) documenting the trajectories in infant mortality attributable to RDS and other causes from the late 1980s and early 1990s to the late 1990s for blacks and whites; (2) determining whether the kind of reversal in RDS mortality from a survival advantage to a survival disadvantage for blacks that was found in data from St. Louis hospitals also occurred nationwide; and (3) estimating race and period effects, along with the influence of socioeconomic, sociodemographic, behavioral, and biomedical risk factors, on black-white variation in infant mortality from RDS and other causes of death. By comparing findings from periods before and after the introduction of surfactant therapy while adjusting for a wide range of other risk factors, we can make inferences regarding the proposition that growing racial disparities are associated with innovations in health technology because of social inequality. An ancillary aim, intended to place the analysis of relative differences in proper context, is to model racial differences in absolute changes in infant mortality from RDS and other causes.

THEORETICAL FRAMEWORK

The Underlying Conceptual Model

The general conceptual model guiding our analysis is grounded in the proposition that as advances in health care occur, the ability of individuals to reduce the risk of disease and

5. Nor, for that matter, have we been able to uncover any multivariate models of race differences in the absolute decline in rates nationwide.

death "is shaped by resources of knowledge, money, power, prestige, and beneficial social connections" (Link and Phelan 2002:730; see also Link and Phelan 1995). Link and Phelan (1995:81) defined social conditions to include "factors that involve a person's relationship to other people . . ." and thus, "in addition to factors like race, socioeconomic status, and gender," these authors included "stressful life events of a social nature (e.g., the death of a loved one, loss of a job, or crime victimization), as well as stress process variables such as social support." Many of these factors appear to be relevant mainly for adult health, but others pertain directly or indirectly to infants as well. For example, adverse social conditions may compromise a woman's health and thereby negatively influence pregnancy and parturition. We are aware of no data set that even approaches comprehensive coverage of the wide range of social risk factors discussed by Link and Phelan. We can, however, straightforwardly operationalize some of the more important maternal attributes, including certain aspects of maternal health endowments. Furthermore, knowledge of whether individuals are in categories that are known to be socially and economically disadvantaged (e.g., racial/ethnic minorities, unmarried mothers, teenage mothers) can be used to proxy social conditions.

The Model Applied to Infant Mortality

Gortmaker and Wise (1997) provided what is perhaps the most directly salient conceptual framework in that it focuses directly on infant mortality. These authors warned that greater racial disparity in infant mortality may accompany advances in health-services technology because the "first injustice" (i.e., social and economic inequality) is apt to translate into differential access to health care. This does not imply that a high-risk black infant will, in a direct sense, be denied therapeutic intervention. Rather, we understand the Gortmaker and Wise argument to be similar to that advanced by Link and Phelan (1995, 2002), that is, that socially disadvantaged groups are less likely to have the information, the social networks, and/or the economic wherewithal to acquire access. The black-white disparity in infant mortality from RDS is only one example of how social variables may lead to differential benefits of advances in perinatal health care. It is a particularly salient example because of the well-documented benefits of surfactant therapy and the timing of the approval of its widespread use in the United States.

Although there is a consensus that surfactant therapy has had a more pronounced impact among white infants than among black infants, explanations for the racial difference in RDS trends vary. For example, the benefits of improvements in health care are geographically unevenly distributed, and infants (especially LBW infants) born at tertiary-care hospitals or regional neonatal intensive care centers have a greater probability of survival than those without access to such facilities (Horbar and Lucey 1995). Thus, one of the first access-limiting mechanisms to consider is that black women may be less likely to be admitted for delivery to facilities where a full array of options for neonatal intensive care is available. While plausible, this expectation is not supported by empirical research. In fact, Schwartz et al. (2000:7) demonstrated that "[b]lack high-risk women were *more* likely to deliver in a high-technology setting" than were their white counterparts (emphasis added). This may result from the fact that access to tertiary-care facilities is more restricted for rural residents (Schwartz et al. 2000), and blacks are much more likely than whites to reside in urban areas. The greater likelihood that black infants will be delivered in a high-technology facility, however, does not imply equality in health care across the board. For example, because blacks, compared with whites, are less apt to receive adequate prenatal care, have fewer economic resources (including medical insurance), and are more likely to reside in areas where exposure to environmental hazards (both natural and social) is greater, black women and infants are likely to be far more disadvantaged with respect to the quantity and quality of health care and social support received before and after parturition.

Two other explanations have been offered for the widening of the black-white gap in the risk of death from RDS. One is that, at least at very low birth weights, black infants "respond less favorably than white infants" to treatment (Ranganathan et al. 2000:458). The likely invalidity of a biological interpretation of racial differences is underscored by the finding from clinical records of no significant difference in RDS mortality between black infants and white infants who received surfactant therapy (Hamvas et al. 1996). Also, there is little evidence that genetic factors are responsible for racial/ethnic differences in infant mortality in general (Wise 1993).

Another explanation that is prominent in the literature on public health depends *not* on the proposition that surfactant replacement is less efficacious among black infants in need of this intervention than among their white counterparts, but rather that black infants are, on average, less likely to require intervention in the first place. Research based on data for years prior to the approval of surfactant replacement showed that RDS "occurs less frequently, is less severe, and is accompanied by fewer complications in black preterm infants" (Hulsey et al. 1993:572). Hamvas et al. (1996) summarized this perspective as follows: "Fetal pulmonary surfactant matures more slowly in white than in black fetuses, and therefore RDS is more prevalent among whites than among blacks" (p. 1635); this view is consistent with the notion that, in general, black infants mature at shorter gestations and lower birth weights (Kline et al. 1989; Wilcox and Russell 1986, 1990). A reasonable expectation, then, is that once surfactant replacement therapy became widespread, RDS mortality would be reduced "more among whites than among blacks" (Hamvas et al. 1996:1635).[6] Narrowly conceived, this explanation focuses on the *need* for intervention, whereas Gortmaker and Wise's perspective could be viewed as placing greater emphasis on *access* to intervention.

However, it is evident that the model exemplified in the work of Gortmaker and Wise (1997) draws on both medical and social models. Furthermore, taken in full context, the research on public health, which proposes a differential need for intervention as an explanation, also explicitly recognizes the need to take social risk factors into account. To see this, one needs only to recall that a central theme of Wise's (2003) contribution to the *Annual Review of Public Health* is that "disparities in infant mortality are rooted fundamentally in social stratification . . ." (p. 342) or Malloy and Freeman's (2000) call for an expansion of research on social, economic, and cultural factors as determinants of the RDS difference by race. Thus, the explanations of the differential need for intervention and the differential access to intervention are appropriately viewed as complementary, rather than competing. One should expect that both perspectives contribute to our understanding of racial disparities in infant mortality; we believe our analysis demonstrates this point.

Risk Factors

The basic demographic covariates we used for this analysis are maternal age, marital status, and nativity, along with parity and the sex of infant. The risk of infant mortality is higher for infants born to teenagers (Hummer et al. 1999; Moss and Carver 1998; Singh and Yu 1996). Maternal age needs to be considered jointly with parity because the risk of adverse outcomes is exacerbated among "primiparas 30 years of age and over and multiparas under 18 years of age" (Kleinman and Kessel 1987:751). A higher risk of infant mortality is generally observed among unmarried mothers (Cramer 1987; Hummer et al. 1999). Adverse pregnancy outcomes are less likely among immigrant women (Hummer et al. 1999), including black women (Cabral et al. 1990), probably because of the positive selection of migration (Frisbie forthcoming; Palloni and Morenoff 2001),

6. Differences in maturational timing by race may not be great enough to provide a full explanation (cf. Richardson and Torday 1994).

although some authors ascribe the immigrant advantage to cultural differences (Cobas et al. 1996; Scribner 1996). We controlled for sex because boys are less apt than girls to be born at low birth weights but are consistently more likely than girls to die in the first year of life (Frisbie, Forbes, and Hummer 1998; Moss and Carver 1998).

Maternal age and marital status, although typically categorized as demographic variables, might be conceptualized as fundamental social determinants of the resource base, whether "knowledge, money, power, prestige, [or] beneficial social connections" (Link and Phelan 2002:730) on which individuals may draw. For example, the finding that infant mortality is higher among teenage mothers has been attributed to a long history of exposure to social conditions that are deleterious to health beginning when these young women were children (Geronimus 1987; Geronimus and Korenman 1993). And the higher mortality rate among infants who are born to unmarried women is generally considered to be a reflection of inadequacy of social and economic resources and/or lifestyle differences (Cramer 1987; Eberstein, Nam, and Hummer 1990; Hummer et al. 1999).

Other somewhat more direct indicators of access that are available in our data set are maternal education and prenatal care. The risk of infant mortality decreases as maternal education rises. In addition to being an indicator of SES, maternal education may reflect knowledge of available medical services and of strategies for circumventing obstacles to access to them. The effect of education on infant mortality is usually attenuated, but often not erased, with controls for mediating variables (Cramer 1987; Hummer et al. 1999). The long-held conclusion that adequate prenatal care is of major benefit for the prevention of low-weight births and therefore a key to reducing infant mortality (Institute of Medicine 1988) has been challenged on the basis of evidence that the apparent beneficial effect stems primarily from selectivity bias (see Alexander and Kotelchuck 2001 for a useful discussion). Regardless of its influence on birth weight, we included prenatal care in the analysis because it represents a package of health-related services that are highly relevant to pregnant women (Alexander et al. 1999; Shiono and Behrman 1995). Adequate prenatal care is one way that women and medical personnel can become aware of existing maternal morbidities and/or problems in fetal development well before the onset of labor. If the receipt of prenatal care is an indication of degree of integration into the formal system of health care, then this, in turn, may have important implications for access to high-quality medical care both before and after childbirth.

Maternal health endowments have a powerful impact on pregnancy outcomes (Eberstein et al. 1990; Frisbie et al. 1998; Kallan 1993; Moss and Carver 1998). Hence, we included previous pregnancy loss and the presence of maternal medical risks (e.g., hypertension, anemia, chronic and pregnancy-related diabetes, eclampsia) as covariates. The same is true of complications of labor and delivery (Hummer et al. 1999), and we also controlled for this risk factor.

Smoking, particularly through its negative effect on birth weight, heightens the risk of infant mortality (Chomitz, Cheung, and Lieberman 1995; Frisbie et al. 1997; Kallan 1993). Maternal weight gain is included as an indicator of the adequacy of nutrition and because of its demonstrated relationship to fetal development (Chomitz et al. 1995). Gestational age and birth weight are the principal risk factors for RDS (British Columbia Reproductive Care Program 1993; Hamvas et al. 1996; Malloy and Freeman 2000). These birth outcomes have long been considered the strongest proximate predictors of infant mortality and mediate the influence of many other risk factors (Cramer 1987; Hummer et al. 1999; Kline et al. 1989; McCormick 1985).

DATA

A data set with a large number of cases is required for the construction of multivariate models from which reasonably stable estimates of the effects of risk factors on infant mortality risk and on specific causes of death can be derived. This essentially means that

we must have vital statistics. We used the NCHS linked birth and infant death cohort files for the years 1989–1990 and 1995–1998, which include all infants born alive in the United States during those years. The data set contains millions of cases each year, and the match rate is exceptional—as early as 1989, more than 97% of the records were successfully linked (U.S. Department of Health and Human Services 1995). We divided the records into births occurring in the pre-surfactant period (1989–1990) and those occurring in the post-surfactant period (1995–1998). We included 1990 as a pre-surfactant year because surfactant therapy was not officially approved until August of that year. Probably for that reason, and perhaps because of a time lag between FDA approval of the therapy and its general availability, our preliminary analysis of relative risk showed only an insignificant difference in RDS mortality between 1989 and 1990.

Linked cohort files exist for years prior to 1989, but they are far less rich than those from later years. For example, no information on maternal smoking, weight gain during pregnancy, medical risks, or labor and delivery complications is available in the pre-1989 data. Also, before 1989, no data on maternal education were collected by California and Texas, and these two heavily populated states and other large states (e.g., Michigan and Ohio) did not report information on marital status. Unfortunately, no linked cohort files exist for the years 1992–1994. Beginning in 2003, birth certificates contain an item on whether surfactant replacement occurred, but this information (which is not likely to be available for public use until at least 2006) would not speak to the core issue in our research, namely, the major changes in the patterns of infant mortality that occurred in the 1990s. Some of the infants born to the 1998 cohort, of course, died in 1999, the year the 10th revision of the *International Classification of Diseases* came into use. This is not problematic in our data. The technical documentation for the 1998 linked files makes it clear that all causes of death for the 1998 cohort are coded according to the 9th revision, as they are for all the other years included in our analysis.

The cumulative percentage of cases with missing data on covariates varies from 17.7% in 1989–1990 to 7.1% in 1995–1998, excluding maternal smoking and weight gain, which were not included on certificates for a few states (most notably, California). Where information on smoking and weight gain was missing, we adopted the conventional strategy of assigning a missing category for these covariates (Frisbie et al. 1998; Singh and Yu 1996).

For other variables, the percentage of missing data ranged from 0% to 8.2% (education in 1989). We do not believe that this leads to any serious distortion of our results. For example, following the deletion of cases with missing data, the black-white IMR ratio based on the linked files for 1995–1998 stands at 2.3—identical to the 1997 ratio reported by Guyer et al. (1998). It is logical to suppose that information is more apt to be missing for cases in which pregnancy outcomes are highly adverse and among blacks who are less likely than whites to have adequate health care. This implies that errors in estimation will be conservative—that is, the degree of racial inequality is likely to be even *greater* than our estimates are able to demonstrate.

Nevertheless, the difference in proportions of missing data between the two periods remains somewhat troubling. Therefore, we also applied the methodology used for smoking and weight gain to four other variables for which the number of cases with missing data was 5% or greater, and then reestimated all models. Our conclusions were unchanged regardless of the methodology we used and, in fact, even the magnitudes of coefficients were quite similar.

A major limitation of our data is that the linked birth and infant death files do not include information on whether surfactant therapy was administered. Such information is available only in clinical records that are limited in geographic coverage and contain too few cases to allow multivariate modeling of a wide range of risk factors on the risk of infant death from specific causes. However, because we knew the date that surfactant

therapy gained FDA approval for general use, we could use a "before-and-after" design to examine indirectly the impact of surfactant replacement on racial differences in RDS mortality.

METHODS

Measures

The outcome variable consists of three categories: infant death from RDS, infant death from other causes, and infant survival. The two populations of interest are non-Hispanic whites and non-Hispanic blacks, which, for ease of exposition, we refer to simply as whites and blacks. As recommended by NCHS, we categorized the cases according to maternal race/ethnicity.

Most of the risk factors were measured in a conventional manner. However, the measures of prenatal care and parity warrant some elaboration. Prenatal care was operationalized in terms of the Kotelchuck Adequacy of Prenatal Care Utilization (APNCU) index. Among the advantages of the APNCU index is that it addresses the selectivity bias associated with women who experience problem pregnancies and who therefore exceed the standard number of prenatal care visits recommended by the American College of Obstetricians and Gynecologists through the addition of an "adequate plus" category to accompany the distinctions between inadequate, intermediate, and adequate care (Kotelchuck 1994a, 1994b). Parity was measured via the Kleinman-Kessel index (Kleinman and Kessel 1987), which combines maternal age and birth order to reflect the interaction of these two variables (see also Hummer et al. 1995). Our diagnostics indicated no problem of collinearity involving the Kleinman-Kessel measure and maternal age.

Models

We used multinomial logistic regression to model the three-category outcome. The results are presented in the form of odds ratios. Because our data set consists of all vital events, the conventional reason for the use of tests of statistical significance (i.e., assessing the probability of error in generalizing from a sample to a population) does not pertain. Hence, the greatest emphasis is placed on the direction and magnitude of the coefficients. Nonetheless, tests of significance retain utility "in order to rule out the simple 'chance processes' alternative" (Blalock 1979:242).

Like most previous research on infant mortality, our study focuses on singleton births. However, some studies of RDS have included plural births (Hamvas et al. 1996; Malloy and Freeman 2000), presumably because while the actual number and proportion of all births lost to the analysis when multiple births are excluded is small, using only singleton births leaves out an important subset of short gestation and low-weight births among whom RDS is apt to occur. Therefore, to make our work as widely comparable as possible, we estimated a supplementary set of models that include multiple births (while controlling plurality). Conclusions that were derived from models that include multiple births were the same as those that emerged from singleton-only models. Hence, the results of the supplementary analysis are not shown, but are available on request.

Also, some prior studies (e.g., Hamvas et al. 1996) have included only very low weight (< 1,500 grams) births. For present purposes, this seems overly restrictive in that (1) "[t]he largest and most consistent drop in RDS-related mortality occurred in the 2000 to 2499 gm birth weight and 33–36 week gestation groups" (Malloy and Freeman 2000:414), and (2) RDS occurs even among normal-weight infants. But because RDS is strongly associated with low-weight and preterm births, we extended our analysis to, and present tabular results from, a second phase in which only LBW infants were included. These additional regressions parallel the basic models in that two sets of regressions were again performed: one for the separate periods and one with period as a covariate.

We show certain tabulations from the LBW analysis because of differences in results between LBW and all-weight models. Models that are restricted to low-weight infants, of course, do not include birth weight or gestational age as risk factors. In the regressions containing births of all weights, both birth weight and gestational age were controlled.

In general, two types of models can be used in the "before-and-after" analysis. The first model, and the one we report on in the greatest detail, estimates the risk of infant death separately for the pre-surfactant (1989–1990) and post-surfactant (1995–1998) periods. The second model pools the data over time and includes period as a dummy variable. Evaluating the relationship separately for the two periods allows an assessment of the magnitude and significance of racial differences in risk without averaging the effects over time. Moreover, the results are more directly comparable to previous descriptive studies in which findings are often juxtaposed for pre- and post-surfactant periods. The two approaches use the same information, and conclusions from each should be (and are) identical. Still, because pooling the data includes period as a covariate and thereby allows an examination of the magnitude and significance of a race × period interaction term, a brief accounting of findings from the second model is presented.

Two problems that are encountered in research on infant mortality that relies on clinical data are small sample size (and thus instability of estimates) and limited generalizability. Conversely, a problem for all research that is based on the currently available national data sets of sufficient size to allow modeling the effects of a large number of factors on the individual risk of infant mortality is the lack of information on whether surfactant therapy was administered. Thus, a direct test of the differential impact of surfactant therapy cannot be conducted for the nation as whole. However, the linked NCHS files permit a reasonable, indirect "before-and-after" test because data (collected and coded with consistent protocols) exist on either side of the date at which surfactant therapy came into general use. Thus, although no definitive conclusions can be reached, we can at least determine whether the results are consistent with the proposition (Gortmaker and Wise 1997; Wise 2003) that technological innovations in health care are associated with greater racial disparities in infant mortality because of persistent social inequality.

Furthermore, we estimated a set of parallel models with neonatal mortality as the dependent variable to allow a comparison with some previous studies that have limited their analysis to that early period of life (e.g., Ranganathan et al. 2000).[7] No changes in our conclusions emerged from the neonatal models (not shown, but available on request).

Finally, our analysis was motivated by a desire to respond to the recent call for an expanded research agenda that is focused on the growth of a racial disparity in the face of absolute declines in infant mortality rates (Malloy and Freeman 2000; Wise 2003). However, for reasons delineated earlier, we also modeled the absolute change in infant mortality from RDS and other causes on a year-to-year basis. We began the modeling of absolute change as a way to insure that the analysis of relative differences would be presented in proper perspective and with the intent of briefly describing the findings. However, we believe that the findings from the latter models will be of substantial interest. The inclusion of these results in an Appendix does not represent a judgment regarding the importance of absolute change as compared with relative change. Rather, it reflects the fact that the modeling of absolute change requires an approach that is rather different from that required for the models that address the core of our analysis—namely, persistent and increasing relative disparities. Thus, the Appendix allows a more coherent presentation than could be achieved by interspersing this discussion throughout the body of the text.

7. Our thanks to a reviewer for suggesting this strategy.

Table 1. Infant Mortality Rates (IMR) per 1,000 Live Singleton Births, by Race: United States, 1989–1990 and 1995–1998

	1989–1990		1995–1998		Rate Ratios (Blacks/Whites)		Absolute Change in Rates 1995–1998 to 1989–1990	
	Whites	Blacks	Whites	Blacks	1989–1990	1995–1998	Whites	Blacks
All Birth Weights								
IMR	6.17	13.89	4.78	11.12	2.25	2.33	–1.39	–2.77
RDS	0.42	1.10	0.17	0.53	2.62	3.12	–0.25	–0.57
Other causes	5.75	12.79	4.61	10.59	2.22	2.30	–1.14	–2.20
Low Birth Weights								
IMR	69.54	78.25	52.81	68.40	1.13	1.30	–16.73	–9.85
RDS	9.14	9.26	3.49	4.64	1.01	1.33	–5.65	–4.62
Other causes	60.40	68.99	49.32	63.76	1.14	1.29	–11.08	–5.23

Source: NCHS linked birth and infant death files, 1989–1990 and 1995–1998.

DESCRIPTIVE RESULTS: DOCUMENTING CHANGES OVER TIME

The trends in the IMRs of whites and blacks between 1989–1990 and 1995–1998 are shown in Table 1 separately for all births and for low-weight births.[8] As shown in the first panel of Table 1, the IMR per 1,000 live births for both RDS and other causes dropped for both groups over time. Bearing most directly on the aims of this research, the relative racial difference (black/white rate ratio) increased, particularly for RDS mortality, with the ratio rising from 2.62 in 1989–1990 to 3.12 in 1995–1998. When attention is limited to LBW infants (i.e., infants born weighing < 2,500 grams), the results are generally similar, but in some ways more striking (see the second panel of Table 1). Rates per 1,000 live births were, of course, much higher among LBW infants. However, the relative risk of RDS mortality was almost identical for blacks and whites (1.01) in the earlier period, but was a third greater for blacks in the post-surfactant period. The risk ratio for all other causes stood at 1.14 in 1989–1990 and increased to 1.29 in 1995–1998. The final two columns of the table show that while the absolute decline in death rates for infants born at all weights was moderately larger for blacks than for whites, the pattern was reversed for LBW infants, among whom the absolute drop in rates was greater for whites than for blacks. This phenomenon is observable in the models of absolute change that appear in the Appendix.

The distributions of risk factors by race and period appear in Table 2. Consonant with all prior studies, blacks are quite disadvantaged with respect to their risk profile. Black mothers are more likely than whites to be in the teenage, unmarried, and high-parity categories. Educational levels are lower among blacks. Blacks are slightly more apt than whites to have had a previous pregnancy loss, to present with medical risks, and to have complications of labor and delivery, and are much more likely to gain only a small amount of weight during pregnancy and to receive prenatal care that is less than adequate. Even as late as 1995–1998, one fifth of black women continued to receive inadequate care.

8. The rates in our analysis will differ slightly from those found in published vital statistics because the latter often contain plural births.

Table 2. Percentage Distributions of Risk Factors of Infant Mortality, by Race: United States, 1989–1990 and 1995–1998

	1989–1990		1995–1998	
Variable	Whites	Blacks	Whites	Blacks
Place of Birth				
United States	95.94	93.53	95.26	91.19
Foreign born	4.06	6.47	4.74	8.81
Maternal Age				
Under 18	5.88	16.56	5.99	16.01
18 or older	94.12	83.44	94.01	83.99
Marital Status				
Unmarried	16.24	66.10	21.42	69.39
Married	83.76	33.90	78.58	30.64
Parity				
First birth	43.05	38.44	42.70	39.76
Low	46.14	38.27	46.49	38.23
High	10.81	23.29	10.81	22.01
Sex				
Male	51.37	50.74	51.30	50.77
Female	48.63	49.26	48.70	49.23
Education				
Less than 12 years	15.15	29.78	13.00	27.62
12 years	39.79	43.34	33.26	39.23
13 or more years	45.06	26.88	53.74	33.15
Previous Loss				
Yes	24.51	26.31	26.24	28.78
No	75.49	73.69	73.76	71.22
Medical Risks				
Yes	19.80	23.62	26.37	30.29
No	80.20	76.38	73.63	69.71
Labor/Delivery Complications				
Yes	31.23	33.19	33.38	34.24
No	68.77	66.81	66.62	65.76
Prenatal Care				
Inadequate	11.14	30.15	7.92	20.56
Intermediate	15.45	15.15	14.06	13.87
Adequate	48.74	30.30	48.51	35.39
Adequate plus	24.67	24.40	29.51	30.18

(continued)

However, black women are less likely to smoke during pregnancy than are white women. As reported elsewhere (Demissie et al. 2001), although the incidence of preterm and low-weight births is considerably greater among blacks, the rates of these adverse birth outcomes rose among whites and fell among blacks between 1989–1990 and 1995–1998.

(Table 2, continued)

Variable	1989–1990		1995–1998	
	Whites	Blacks	Whites	Blacks
Smoking				
Yes	17.64	13.44	14.08	8.78
No	65.51	69.95	70.21	80.73
Missing	16.85	16.61	15.71	10.49
Weight Gain				
< 15 lbs.	4.82	9.25	6.66	11.43
15–40 lbs.	64.88	56.98	65.48	59.93
40+ lbs.	12.73	10.97	16.10	14.59
Missing	17.57	22.80	11.76	14.05
Gestational Age				
Preterm (< 37 weeks)	7.35	17.46	8.11	15.88
Term (≥ 37 weeks)	92.65	82.54	91.89	84.12
Birth Weight				
Low (< 2,500 grams)	4.43	11.57	4.75	11.13
Normal (≥ 2,500 grams)	95.57	88.43	95.25	88.87
N	4,349,749	1,057,417	8,753,020	2,056,726

Source: NCHS linked birth and infant death files, 1989–1990 and 1995–1998.

RESULTS FROM REGRESSION MODELS

We begin the discussion of multivariate results with comparisons of black infants and white infants born at all weights. We then compare these results with those obtained among LBW infants.

Multivariate Results: All Birth Weights in the Pre- and Post-Surfactant Periods

The pre-surfactant period. The risk of infant mortality (at all birth weights) from RDS and all other causes for blacks compared with whites is examined in Table 3 for the pre-surfactant period (1989–1990) in terms of five models that progressively adjust for sets of risk factors. The first two columns display the familiar bivariate relationship in which the odds ratios that are associated with death from both RDS and other causes are more than twice as high for black infants. With sociodemographic covariates controlled (Model 2), the risk for blacks is considerably reduced. The magnitude of the decline is not unexpected; for example, black infants are much more likely to be born to teenage mothers and unmarried mothers, which are clearly socially and economically disadvantaged populations. Following adjustment for education and prenatal care (Model 3 of Table 3), the risk of infant death from RDS and other causes among blacks is reduced further. Controls for maternal health variables, beginning with previous loss up to and including weight gain during pregnancy (Model 4), result in another substantial decline in odds for blacks; the odds ratios (OR) are now only 1.367 and 1.375 for RDS and other-cause mortality, respectively. Also, as expected, adjustment for gestational age and birth weight (Model 5) has a sizable impact, with the risk of death among black infants from other causes becoming essentially identical to the corresponding risk for whites.

Table 3. Odds Ratios for Effects of Risk Factors on Infant Mortality: United States, 1989–1990

	Model 1		Model 2		Model 3		Model 4		Model 5	
Variable	RDS	Other Causes	RDS	Other Causes	RDS	Other Causes	RDS	Other Causes	RDS	Other Causes
Racial Group (White)										
Black	2.618**	2.242**	1.848**	1.575**	1.707**	1.499**	1.367**	1.375**	0.809**	0.985
Place of Birth (United States)										
Foreign born			0.928	0.872**	0.954	0.876**	1.035	0.922**	1.067	0.965
Age (18 or older)										
Under 18			1.321**	1.219**	1.155*	0.999	1.466**	1.179**	1.263**	1.119**
Marital Status (Married)										
Unmarried			1.742**	1.713**	1.594**	1.499**	1.484**	1.401**	1.146**	1.230**
Parity (First birth)										
Low			0.775**	1.083**	0.775**	1.059**	0.825**	1.092**	0.955	1.168**
High			1.094†	1.570**	1.033	1.395**	0.992	1.356**	1.052	1.386**
Sex										
(Female)										
Male			1.573**	1.250**	1.551**	1.241**	1.563**	1.246**	1.646**	1.309**
Education										
(13 or more years)										
Less than 12 years					1.357**	1.492**	1.213**	1.348**	1.025	1.218**
12 years					1.201**	1.192**	1.172**	1.157**	1.064	1.102**
Prenatal Care (Adequate)										
Inadequate					2.219**	1.758**	1.711**	1.521**	0.855**	1.135**
Intermediate					0.926	1.149**	0.914	1.143**	0.928	1.149**
Adequate plus					4.315**	2.314**	3.400**	2.033**	0.843**	1.046**
Previous Loss (No)										
Yes							1.350**	1.205**	1.137**	1.117**

(continued)

Moreover, the odds of a black infant dying from RDS become significantly *lower* than the odds for a white infant (OR = 0.809).

In general, the results support the view that social and health conditions are highly influential determinants of black-white inequality in infant mortality. In particular, the results demonstrate that with adjustment for all covariates, the RDS-specific risk for black infants was significantly lower than that for white infants during the pre-surfactant period.

Table 3 also shows that the effects of most risk factors are consistent with virtually all previous research and the conceptual model we discussed earlier, thereby rendering unnecessary a detailed discussion of these effects. However, a few of the relationships warrant at

(Table 3, continued)

Variable	Model 1 RDS	Model 1 Other Causes	Model 2 RDS	Model 2 Other Causes	Model 3 RDS	Model 3 Other Causes	Model 4 RDS	Model 4 Other Causes	Model 5 RDS	Model 5 Other Causes
Medical Risks (No)										
Yes							2.120**	1.667**	1.172**	1.212**
Labor/Delivery Complications (No)										
Yes							5.154**	2.289**	2.009**	1.516**
Smoking (Nonsmoker)										
Missing							0.729**	0.867**	0.818**	0.896**
Smoker							1.026	1.217**	0.750**	0.981
Weight Gain (15–40 lbs.)										
Missing							3.110**	1.833**	2.023**	1.498**
< 15 lbs.							6.982**	3.199**	3.074**	2.097**
40+ lbs.							0.268**	0.639**	0.789†	0.932**
Gestational Age (Term)										
Preterm									23.862**	2.811**
Birth Weight (Normal)										
Low									55.779**	7.390**
Intercept	–7.76**	–5.15**	–8.06**	–5.49**	–8.82**	–5.93**	–10.26**	–6.58**	–12.40**	–6.53**
–2 LL	502,902.4**		498,592.4**		491,868.5**		468,746.5**		409,058.4**	

Source: NCHS linked birth and infant death files, 1989–1990 and 1995–1998.

Note: Variables in parentheses are reference groups.

$^{\dagger}p \leq .10$; $^{*}p \leq .05$; $^{**}p \leq .01$ (two-tailed tests)

least brief mention. First, the odds ratios for racial differences that are associated with both RDS and other causes were greatly reduced in Model 4 before birth outcomes were controlled, thereby highlighting the power of social and maternal health factors in accounting for racial variation in infant mortality. As expected, the magnitudes of the effects of gestational age and birth weight are the largest. Furthermore, their effects are much greater in the case of RDS than for other causes of death, thus reconfirming the strong association of RDS with premature and low-weight births. The effects of most risk factors on infant death are substantially smaller with gestational age and birth weight controlled, as anticipated in light of the mediating role typically played by these birth outcomes. It does seem anomalous that the coefficient for smoking is reversed in the full model. One interpretation is that with the disadvantage of LBW and short gestation controlled, smoking serves as an indicator of monetary resources, for which we are unable to control directly here. More likely is that this result is a statistical artifact. Indeed, Almond, Chay, and Lee (2003) reported a similar finding; they noted that "maternal smoking and other health outcomes of the mother and infant are likely to be simultaneously determined, and that the coefficient on smoking may be biased" (p. 33).

Table 4. Odds Ratios for Effects of Risk Factors on Infant Mortality: United States, 1995–1998

	Model 1		Model 2		Model 3		Model 4		Model 5	
Variable	RDS	Other Causes	RDS	Other Causes	RDS	Other Causes	RDS	Other Causes	RDS	Other Causes
Racial Group (White)										
Black	3.071**	2.312**	2.412**	1.761**	2.252**	1.674**	1.766**	1.541**	1.098*	1.123**
Place of Birth (United States)										
Foreign born			0.951	0.830**	0.998	0.842**	1.021	0.870**	1.078	0.914**
Age (18 or older)										
Under age 18			1.091	1.252**	0.999	1.003	1.340**	1.223**	1.181*	1.168**
Marital Status (Married)										
Unmarried			1.604**	1.539**	1.465**	1.324**	1.471**	1.267**	1.187**	1.130**
Parity (First birth)										
Low			0.684**	0.995	0.679**	0.967**	0.654**	0.938**	0.875**	1.094**
High			0.829**	1.441**	0.791**	1.258**	0.709**	1.152**	0.840**	1.257**
Sex (Female)										
Male			1.484**	1.237**	1.464**	1.227**	1.487**	1.238**	1.542**	1.293**
Education (13 or more years)										
Less than 12 years					1.337**	1.603**	1.208**	1.421**	1.031	1.272**
12 years					1.357**	1.314**	1.296**	1.244**	1.189**	1.177**
Prenatal Care (Adequate)										
Inadequate					2.169**	1.917**	1.616**	1.605**	0.766**	1.127**
Intermediate					1.018	1.147**	0.962**	1.112**	0.993	1.124**
Adequate plus					4.223**	2.436**	3.526**	2.192**	0.825**	1.019†
Previous Loss (No)										
Yes							1.387**	1.191**	1.202**	1.115**

(continued)

The post-surfactant period. The patterns of risk-factor effects are similar in both periods. However, the estimates of racial disparity in the post-surfactant period (Table 4) differ from those in the pre-surfactant period in several important ways. First, a comparison of the bivariate relationship (Model 1 in Tables 3 and 4) shows that the relative odds of a black infant dying from RDS were higher in 1995–1998 (OR = 3.071) than in 1989–1990 (OR = 2.618). In fact, in every model, the odds of death from RDS among blacks in the post-surfactant period were higher than those in the pre-surfactant period. For example, when sociodemographic variables were controlled, the estimate of the risk of RDS death for blacks was about 85% higher than that for whites before the widespread use of pulmonary surfactant therapy (Model 2 of Table 3). In the post-surfactant era, the analogous

(Table 4, continued)

Variable	Model 1		Model 2		Model 3		Model 4		Model 5	
	RDS	Other Causes	RDS	Other Causes	RDS	Other Causes	RDS	Other Causes	RDS	Other Causes
Medical Risks (No)										
Yes							1.820**	1.587**	0.996	1.116**
Labor/Delivery Complications (No)										
Yes							4.466**	2.122**	2.158**	1.473**
Smoking (Nonsmoker)										
Missing							0.603**	0.775**	0.767**	0.860**
Smoker							1.012	1.320**	0.759**	1.058**
Weight Gain (15–40 lbs.)										
Missing							3.426**	2.116**	2.182**	1.681**
< 15 lbs.							6.355**	3.334**	3.120**	2.270**
40+ lbs.							0.132**	0.625**	0.338**	0.926**
Gestational Age (Term)										
Preterm									18.096**	2.976**
Birth Weight (Normal)										
Low									44.652**	8.091**
Intercept	–8.65**	–5.38**	–8.82**	–5.65**	–9.65**	–6.16**	–10.94**	–6.79**	–12.84**	–6.82**
–2 LL	804,289.5**		798,850.2**		786,424.5**		749,970.3**		651,605.8**	

Source: NCHS linked birth and infant death files, 1989–1990 and 1995–1998.

Note: Variables in parentheses are reference groups.

$^{\dagger}p \leq .10$; $^{*}p \leq .05$; $^{**}p \leq .01$ (two-tailed tests)

model estimates the relative odds to be over 140% greater (Model 2 of Table 4). In Model 4, which adjusts for all risk factors except gestational age and birth weight, the RDS disadvantage for blacks was moderate in 1989–1990 (OR = 1.367), but by 1995–1998, the odds ratio had increased to 1.766. The same pattern of results obtains in regard to infant deaths from other causes, but the size of the differences in odds ratios between the two periods is smaller compared with the risk associated with RDS. Finally, in the full model (Model 5), the black-white difference in RDS mortality is actually reversed. In the pre-surfactant period, net of the effects of all covariates, black infants were significantly less likely than white infants to die from RDS (OR = 0.809). The analogous model shows that the risk for black infants became significantly higher than the risk for white infants after the introduction of surfactant therapy (OR = 1.098). The odds of death among black infants from all other causes, which were statistically identical to the odds for white infants in 1989–1990 (OR = 0.985, not significant), became significantly higher in 1995–1998 (OR = 1.123).

Because we have data for the entire United States, the conventional reason for using tests of statistical significance to assess the probability of error in generalizing from a

sample to a population recedes in importance. Nevertheless, we tested whether the differences between the two periods are statistically significant. In every comparison for every model from Tables 3 and 4, the change in the risk of death from RDS and other causes among black infants, compared with white infants, was significantly different between 1989–1990 and 1995–1998—including, of course, the reversal just mentioned ($p \leq 0.01$).

The results of the regression models that were constructed separately by period for infants born at all weights provide support for the hypotheses regarding both the differential need for intervention and the differential access to intervention. As shown in Tables 3 and 4, although the bivariate estimate (Model 1) showed the risk of infant death for blacks to be 2.5 to 3 times greater than that for whites, controls for social and maternal health factors reduced the disparity considerably in both periods—a finding consistent with the notion that social inequality creates obstacles to receiving health care. On the other hand, the findings that the black-white difference in risk was consistently greater in the post-surfactant period for RDS mortality and that there was a reversal from a black RDS survival advantage before surfactant replacement therapy became widespread to a survival disadvantage after that innovation are consistent with the explanation of the differential need for intervention.

Multivariate Results: LBW Infants in the Pre- and Post-Surfactant Periods

Although birth weight and gestational age were not controlled in the logistic regression focusing on LBW infants, the nature (but not the magnitude) of the effects of the other covariates is highly similar to those estimated for infants born at all weights. Thus, any discussion or tabular presentation of these relationships would be largely redundant. (The full tabulations are available on request.)

Inasmuch as previous research has consistently found a survival advantage for black infants at very low birth weights, one would expect the relative disparity between black and white LBW infants to be smaller than that observed among all infants. Indeed, this is what we found. In Table 5, the odds ratios contrasting the mortality risk of black LBW infants with the risk for their white counterparts are again shown separately by period. When these values are juxtaposed with the analogous values for infants born at all weights (e.g., compare with the first row of figures of Models 1–4 in Tables 3 and 4), in every instance, the odds of black infant mortality are lower in Table 5. Also, the change in racial disparity shifted more dramatically among LBW infants between 1989–1990 and 1995–1998 than was the case for all infants. In the earlier period, the bivariate estimate (Model 1) demonstrates that the risk of death from RDS for black LBW infants was statistically identical to the risk for white LBW infants. In each of the other models, the risk of death from RDS was lower for blacks than for whites; the full model (Model 4 of Table 5) shows that the odds for black infants were about 17% lower in 1989–1990. The odds ratios associated with all other causes of death were modestly and significantly higher among black infants, except in Model 4, where the risk was modestly lower (OR = 0.958).

The findings for the post-surfactant period (1995–1998) present a sharp contrast. Although the odds ratios decline in magnitude as controls are added, in every model, black infants were significantly more likely to die from RDS and all other causes than were white infants. Furthermore, just as with the estimates from Tables 3 and 4, the changes in the risk of infant mortality for blacks (relative to whites) among low-weight births were statistically significant. Thus, it is clear that for low-weight births, just as for births at all weights, during the 1990s, the former advantage for blacks with respect to RDS was reversed, and the disparity in black-white infant mortality from all other causes increased.

Multivariate Results: Period as a Covariate

To quantify the effect of period, we merged the 1989–1990 and 1995–1998 data and performed regressions analogous to those on which Tables 3–5 are based, except that the

Table 5. Odds Ratios of Infant Mortality Among Black Low-Birth-Weight Infants, by Period: United States, 1989–1990 and 1995–1998

	Model 1		Model 2		Model 3		Model 4	
Period	RDS	Other Causes	RDS	Other Causes	RDS	Other Causes	RDS	Other Causes
1989–1990	1.022	1.153**	0.949	1.076**	0.947	1.073**	0.832**	0.958*
Intercept	–4.623**	–2.735**	–5.033**	–3.014**	–5.227**	–3.161**	–6.201**	–3.861**
–2 LL	181,837.9**		180,875.9**		180,351.9**		171,917.8**	
1995–1998	1.351**	1.314**	1.324**	1.301**	1.319**	1.293**	1.114*	1.133**
Intercept	–5.603**	–2.955**	–5.918**	–3.183**	–6.155**	–3.372**	–7.102**	–4.048**
–2 LL	304,452.3**		303,209.0**		302,706.7**		287,697.5**	

Source: NCHS linked birth and infant death files, 1989–1990 and 1995–1998.

Notes: White is the reference group. Model 1 is the bivariate relationship. Model 2 controls place of birth, maternal age, marital status, parity, and sex of infant. Model 3 includes the controls from Model 2, plus education and prenatal care. Model 4 includes the controls from Model 3, plus previous loss, medical risks, labor and delivery complications, smoking, and weight gain.

*$p \leq .05$; **$p \leq .01$ (two-tailed tests)

main effect of period and its interaction with race were added. Results are not presented in tabular form (but are available on request) because the conclusions remain the same as those derived from separate modeling of the pre- and post-surfactant periods. The model for all births demonstrates that the risk from RDS was approximately 56% lower (OR = 0.440) in the post-surfactant period than in 1989–1990, whereas the odds were lower by only about 19% in the case of mortality from all other causes. This is expected inasmuch as surfactant replacement is an innovation aimed primarily at reducing RDS mortality. Even more notable, the race × period interaction term indicates that for RDS-implicated mortality, the relative risk of being black was almost one-third greater in the post-surfactant period than in the pre-surfactant period (OR = 1.328). The direction of effect is the same for all other causes, but the size of the effect is smaller (OR = 1.135).

With one exception, the findings for low-weight births are highly similar to those uncovered for infants at all birth weights. The one exception parallels the results from Table 4: the black-white disparity in the bivariate model is much lower among LBW infants than among infants of all birth weights.

Models of Absolute Change in Infant Mortality

We examined two dimensions of absolute change in infant mortality by race: reductions over time and differences in reductions (i.e., second differences) over time. The methods and the results of our analyses of absolute change are described in some detail in the Appendix. Here, we summarize what we believe to be the most interesting results. Bivariate models for all births show what we already knew from our inspection of empirical rates: the absolute declines in infant mortality from RDS and all other causes for blacks exceeded those for whites. In the full models, net changes were, of course, much smaller, and whether greater improvement occurred among blacks or whites varied by the year and cause of death. Among LBW infants, the bivariate model showed greater improvement for whites, just as seen in Table 1. In the case of second differences, bivariate models indicate that absolute change for both RDS and other causes was more favorable among whites in every instance, whereas in the fully adjusted models, there were no significant differences by race. Thus, even in regard to absolute change, blacks are

disadvantaged in some respects, and adjustment for all covariates resulted in no significant differences by race.

CONCLUSIONS

To address our first specific aim, we examined black-white changes in infant death from RDS and all other causes (Table 1) and found that, for the entire United States, substantial reductions in the IMR for both groups were accompanied by a widening of the relative gap between blacks and whites, with the increased disparity being considerably greater for RDS mortality than for mortality from all other causes. We also found that while the absolute decrease in the IMR for all births was moderately larger for blacks, the absolute decline among low-weight births was *greater* for whites.

In regard to the second aim, multivariate models of individual risk that were constructed separately for 1989–1990 and 1995–1998 showed that for RDS mortality, there was a nationwide reversal from a survival advantage for black infants in the earlier (pre-surfactant) period to a survival disadvantage for them in the later (post-surfactant) period. This result is consistent with Hamvas et al.'s (1996) findings, which were based on clinical records from St. Louis–area hospitals, but not with studies of national-level data that did not adjust for social and biomedical risk factors.

Our third aim was a broader examination of the factors underlying the expanding risk for black infants relative to white infants. As part of this broader objective, we hoped to evaluate indirectly the perspective that innovations in health care and technology are likely to be associated with growing black-white disparity in health and mortality because of the "first injustice" (Gortmaker and Wise 1997), that is, continuing inequality in social resources, including "knowledge, money, power, prestige, and beneficial social connections" (Link and Phelan 2002:730). We addressed our third aim through a wide range of models: (1) models for all births and for low-weight births, (2) models for singletons only and models that included plural births, (3) separate models of the relationships of interest for 1989–1990 and 1995–1998 and models with period as a covariate, (4) models of neonatal mortality only, and (5) various combinations of the above. The conclusions we reached were virtually identical across all models. The relative difference between blacks and whites increased significantly over time, but the magnitude of the disparity was greatly reduced by adjusting for indicators of social inequality.

Because the timing of the FDA approval of surfactant therapy for general use is clearly established, and given strong evidence of the efficacy of surfactant replacement as a preventive and curative intervention aimed primarily at RDS, we reasoned that an analysis of variation in infant mortality by race in the pre-surfactant period, in comparison with the post-surfactant period, would shed some light on the tenability of the proposition that growth in the racial disparity in infant mortality is likely to follow innovations in health technology in the context of persistent social inequality. Data limitations, including the lack of clinical data and, most notably, the absence of any information on whether surfactant therapy was applied, prevented a definitive test. Still, if the hypothesis regarding the differential need for surfactant therapy has any validity, it was reasonable to anticipate (1) that estimates from multivariate analyses would show that the risk of infant death from RDS in the post-surfactant era was larger for blacks relative to whites than was the case prior to the FDA approval of this intervention, and (2) that the change for RDS would be more substantial than for all other causes of death. Inasmuch as both of these results emerged in our data, we can at least conclude that the "before-and-after" findings are consistent with the conceptual framework we advanced. The finding of a nationwide reversal from blacks' relative advantage in RDS survival in the pre-surfactant period to a relative disadvantage in the post-surfactant period provides some support for the interpretation of the differential need for intervention. On the other hand, the fact that controls for social risk factors resulted in a marked diminution of the disadvantage for

blacks relative to whites in regard to both RDS and other-cause mortality in both periods is evidence that differential access to intervention plays an important role in accounting for the black-white disparity in risk of infant mortality. Taken together, these findings are consistent with the conclusion that the social and medical models can appropriately be viewed as complementary, rather than competing.

Our estimates from race-specific bivariate models, based on logit approximations of absolute change in rates over time for all births, closely reflect what is observed in the empirical rates: absolute declines in infant mortality from RDS and all other causes were greater for blacks than for whites. However, more-detailed analyses indicate that, even in regard to absolute change, blacks are disadvantaged in some respects. For example, bivariate models focusing only on LBW infants suggest a small advantage for whites with respect to RDS and a larger advantage for whites for all other causes. An analysis in which the outcome of interest was the difference in reduction in the approximated rates (the change for blacks minus the change for whites) also produced mixed results. As shown in Appendix Table A3, the bivariate model for all births indicates a greater reduction in mortality for blacks, but with full controls, none of the second differences is significant. Among low-weight births in the bivariate models, absolute declines for both RDS and other-cause mortality were greater for white infants. In the fully adjusted models, the differences were statistically nonexistent.

We would be remiss if we did not explicitly recognize that disparities in infant mortality are the result of differences in composition as well as differences in "return to risk." Thus, while the risk of infant mortality at low birth weights may be lower among blacks (i.e., a more favorable "return to risk" as compared with whites), blacks are immensely disadvantaged in compositional terms owing to the much greater proportion of black births than white births that occur at low weights and short gestations. In this very important sense, then, the need of black infants for access to health care interventions is obviously greater than the need of white infants. Considered in this context, and taking into account that blacks are far more disadvantaged in terms of their risk profile than are whites and that controls for social factors substantially diminish the racial disparity in risk, we believe our results support the view that whatever differences exist in the need for intervention, social inequality remains a fundamental cause of disparities in health care, morbidity, and mortality.

APPENDIX

Approaches to Modeling Absolute Change in Infant Mortality

While descriptive analyses are readily available, we found no multivariate models of absolute change in nationwide race differences in infant mortality in the literature that would allow a direct comparison with change in individual relative risk. The approach to modeling absolute change that comes most immediately to mind involves regressing rates of RDS and other-cause mortality obtained for counties, cities, or other geographic units on aggregate characteristics of whatever spatial unit is selected. Ecological studies of this sort are of interest and proven utility, but to generalize findings from such research to relationships at the individual level would be to engage in an obvious ecological fallacy. Another strategy would be to conduct a longitudinal analysis in which years are the units of analysis, but the number of years for which comparable data are available are too few to support this approach.

An alternative exists for investigating absolute change in RDS and other causes of infant death over time by group by pooling over the individual data for the periods 1989–1990 and 1995–1998, while including a dummy variable for each of the six years, along with other covariates from the earlier analysis, and then fitting two multinomial logistic regression models to each of the racial groups. The constant term is excluded to

yield the cause-specific baseline log odds for each year, which can then be interpreted as a set of constant terms. In the model without controls, the exponentiated logits corresponding to the dummy variables for year provide approximations of the yearly observed mortality rates. The estimated odds can be interpreted approximately as rates when the cause-specific mortality probabilities tend toward zero. In this context, the cause-specific probabilities from the multinomial logit model without controls are virtually identical to the observed rates. In our data, the logits for RDS mortality for any year are in the range –5.6 to –4.4, while the logits for other causes range from –3.0 to –2.5. These logits yield cause-specific mortality probabilities (i.e., rates) in the range of 0.003 to 0.075. The denominators on which the multinomial probabilities are based are close to unity for logits of this magnitude.

Again, these estimates are odds, not rates, but they serve as reasonable approximations of rates. For example, consider the observed rates for whites from Table 1. In this case, the observed rate of RDS mortality is 0.42 per 1,000, and the mortality rate from other causes is 5.75 per 1,000; these rates translate into probabilities of 0.00042 and 0.00575, respectively. Let p_{RDS} and p_{OTH} denote the probabilities of death from RDS and death from other causes, respectively. The probability of survival is $p_{SURV} = 1 - (p_{RDS} + p_{OTH})$. The multinomial logit for mortality from RDS (versus survival) from a model based on these probabilities would be $\log[p_{RDS} / p_{SURV}] = -7.769$. For all other causes, the logit is $\log[p_{OTH} / p_{SURV}] = -5.1523$. Exponentiating these logits gives 0.000423 (0.423 per 1,000) for RDS and 0.005786 (5.786 per 1,000) for all other causes, values sufficiently close to the empirical rates to support the analysis.

To assess change over time, we evaluate estimated yearly differences in the odds, interpreted as approximate rates and changes in rates. Significance tests of differences are carried out based on large sample properties of functions of maximum-likelihood estimators. The estimated odds from a multinomial logit model have a normal sampling distribution in large samples, and the variance of the odds can be obtained using the delta method (Rao 1973). The standard errors of the yearly odds are then used to compute the standard errors of the difference between odds.

Results

The Appendix tables show absolute changes in approximated rates expressed per 1,000 live births for each year compared with 1989 as the reference year for singleton births for infants of all weights and low birth weights, based on both models without controls and those with full controls. As before, birth weight and gestational age are included as covariates for analyses involving all births, but not for models of LBW infants. Tests of statistical significance (z values) accompany every comparison. To reach the 95% confidence level ($p \leq 0.05$), a z value of 1.96 is required; for $p \leq 0.01$, z must be greater than or equal to 2.58.

Appendix Table A1 shows the absolute decline in approximated rates separately by race. The first panel presents bivariate and full models for absolute changes in RDS mortality, and the second panel repeats the process for all other causes. In the bivariate models, for both RDS and other-cause mortality, the changes closely reflect what is observed in the distribution of empirical rates, including consistently greater absolute declines for blacks than for whites. Models with full controls present a different picture. In the case of RDS, the estimated absolute decline was greater for whites than for blacks for every comparison except two: those for 1997 and for 1998, for which the drop in rates was identical for both races. For deaths from other causes, the pattern is less uniform, with sometimes one racial group and sometimes the other recording the greatest decrease.

Appendix Table A2 replicates the analysis for LBW infants. Two notable differences in the findings from the table demonstrate the utility of a separate consideration of infants born at low weights. For these high-risk infants, in the bivariate models for both RDS and

Appendix Table A1. Reduction in Logit Approximations of Infant Mortality Rates per 1,000, Contrasted to 1989, by Race: Births of All Weights

	Bivariate Model				Full Model			
	Whites		Blacks		Whites		Blacks	
Year	Estimate	z	Estimate	z	Estimate	z	Estimate	z
Infant Mortality—Respiratory Distress Syndrome								
1989	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.
1990	0.130	6.51	0.222	3.37	0.002	1.94	0.001	0.70
1995	0.301	16.72	0.663	11.00	0.004	5.56	0.003	2.70
1996	0.320	17.97	0.652	10.76	0.004	5.99	0.003	2.64
1997	0.329	18.62	0.695	11.62	0.004	6.20	0.004	2.88
1998	0.321	18.02	0.760	12.98	0.004	6.01	0.004	3.19
Infant Mortality—All Other Causes								
1989	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.
1990	0.355	4.84	0.654	2.90	0.073	2.69	0.068	0.77
1995	1.155	16.23	2.361	10.64	0.295	11.58	0.335	3.94
1996	1.355	19.16	2.306	10.34	0.348	13.85	0.314	3.67
1997	1.347	19.02	2.950	13.40	0.356	14.22	0.426	5.61
1998	1.486	21.09	2.789	12.67	0.384	15.49	0.383	4.53

Source: NCHS linked birth and infant death files, 1989–1990 and 1995–1998.

Note: $z = 1.96$ at $p \leq 0.05$; $z = 2.58$ at $p \leq 0.01$ (two-tailed tests).

Appendix Table A2. Reduction in Logit Approximations of Infant Mortality Rates per 1,000, Contrasted to 1989, by Race: Low-Weight Births

	Bivariate Model				Full Model			
	Whites		Blacks		Whites		Blacks	
Year	Estimate	z	Estimate	z	Estimate	z	Estimate	z
Infant Mortality—Respiratory Distress Syndrome								
1989	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.
1990	2.920	6.71	1.924	3.19	0.520	2.73	0.452	1.22
1995	7.228	17.32	5.814	10.51	1.341	8.23	1.515	4.78
1996	7.692	18.68	5.667	10.16	1.433	8.92	1.492	4.69
1997	7.884	19.25	6.085	11.06	1.470	9.19	1.606	5.13
1998	7.809	19.02	6.712	12.48	1.449	9.03	1.750	5.69
Infant Mortality—All Other Causes								
1989	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.
1990	3.144	0.07	2.314	1.38	0.779	1.17	0.399	0.31
1995	13.018	11.05	7.025	4.13	4.125	6.72	2.912	2.31
1996	14.957	12.78	6.873	4.01	4.913	8.13	3.026	2.40
1997	15.071	12.88	9.492	5.60	5.010	8.30	4.124	3.33
1998	14.864	12.69	7.121	4.17	4.864	8.03	3.008	2.39

Source: NCHS linked birth and infant death files, 1989–1990 and 1995–1998.

Note: $z = 1.96$ at $p \leq .05$; $z = 2.58$ at $p \leq .01$ (two-tailed tests).

Appendix Table A3. Black-White Differences in the Reduction of Approximations of Rates per 1,000

	Infant Mortality Respiratory Distress Syndrome				Infant Mortality All Other Causes			
	Bivariate Model		Full Model		Bivariate Model		Full Model	
Year	Difference in Estimate Blacks – Whites	z	Difference in Estimate Blacks – Whites	z	Difference in Estimate Blacks – Whites	z	Difference in Estimate Blacks – Whites	z
Births of All Weights								
1989	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.
1990	0.092	1.34	–0.000	0.29	0.299	1.26	–0.006	0.06
1995	0.362	5.76	–0.000	0.16	1.205	5.18	0.040	0.45
1996	0.333	5.27	–0.001	0.36	0.951	4.06	–0.034	0.38
1997	0.366	5.87	–0.000	0.27	1.603	6.93	0.070	0.80
1998	0.439	7.18	0.000	0.03	1.303	5.64	–0.002	0.02
Low-Weight Births								
1989	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.
1990	–0.996	1.30	–0.068	0.16	–0.830	0.02	–0.379	0.26
1995	–1.413	2.04	0.174	0.49	–5.993	2.90	–1.213	0.87
1996	–2.025	2.92	0.059	0.17	–8.085	3.90	–1.887	1.35
1997	–1.799	2.62	0.136	0.39	–5.578	2.71	–0.886	0.64
1998	–1.097	1.62	0.301	0.87	–7.743	3.74	–1.856	1.33

Source: NCHS linked birth and infant death files, 1989–1990 and 1995–1998

Note: $z = 1.96$ at $p \leq .05$; $z = 2.58$ at $p \leq .01$ (two-tailed tests)

other causes, the improvement for whites exceeded that for blacks, just as was true of the relative differences we examined earlier. In the full model estimating changes in RDS mortality, the improvement in survivorship was modestly greater for blacks (except for the 1990 comparison). The reverse is true in the full model for all other causes, in which the magnitude of reductions for whites exceeded those for blacks. The changes between 1989 and 1990 did not approach significance in three of the four comparisons.

Appendix Tables A1 and A2 allow an examination of changes by race and show which differences were significant. By contrast, Appendix Table A3 shows the direction, magnitude, and significance of black-white differences in changes in the rate approximations. That is, the models in Appendix Table A3 *begin* with the differences shown in Tables A1 and A2 and show how these differences changed from year to year. Specifically, the models analyze second differences in which the change for whites was subtracted from the change for blacks. An estimate with a positive sign represents a greater improvement for blacks, and a negative sign represents a greater improvement for whites.

The bivariate model for RDS among all births (Appendix Table A3) reflects what is already known about trends in rates available from NCHS vital statistics reports: absolute declines among blacks exceeded those for whites. The fact that the difference between 1989 and 1990 was not significant again supports the decision to combine the two years as a pre-surfactant period. With full controls, the RDS differences between blacks and whites disappear for every yearly comparison. The bivariate model for all other causes also indicates greater decreases in approximated rates for blacks. The full models show that when blacks are equated with whites on all risk factors, no significant differences remain.

When the relationships of interest are modeled for the LBW infants (lower panel of Appendix Table A3), in every instance in the bivariate models for both RDS and other causes, mortality declines reflect an advantage for whites (as indicated by the negative signs). Thus, the trend among LBW infants seen in the raw rates in Table 1 is reflected in Appendix Table A3. With controls for risk factors, however, there are no significant racial differences. The bivariate model for all other causes of death that occurred to LBW infants evidences the same pattern—that is, whites had larger decreases than did blacks, most of which are significant. A slight advantage for whites remains, net of the effects of risk factors, but none of the differences is statistically significant.

Several important inferences may be drawn from the modeling of approximated rates. First, as inspection of vital statistics descriptive reports shows, over time, absolute declines in rates for blacks surpass those of whites for infant mortality from RDS and all other causes. Thus, it is important to recognize that although the widening racial gap in relative risk of infant mortality is of major concern, the overall picture is one of great success in reducing rates of infant death for all racial/ethnic groups. However, when the focus is on high-risk (i.e., low-weight) births, the trend in terms of bivariate comparisons is one of rising racial inequality in the logit approximations of absolute changes in rates. With adjustment for risk factors, some evidence of racial inequality remains, but the effects are never statistically significant.

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