Increasing Racial Disparity in Infant Mortality: Respiratory Distress Syndrome and Other Causes *

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ABSTRACT

Dramatic innovations in perinatal care have led to a substantial decline in rates of infant death, but they have also fueled historic alterations in the structure of infant mortality. Among the most disturbing of recent trends is a significant increase in relative black-white disparity in risk of infant death. Based on data from linked cohort files for 1989-90 and 1995-98, our results are consistent with the view that the potential for a widening of the racial gap is high because advances in health care have occurred in a continuing context of social inequality. This conclusion is especially well-illustrated in the case of respiratory distress syndrome by comparisons drawn from time periods before and after the widespread use of pulmonary surfactant therapy, but the conclusion also appears valid for all other causes of infant mortality.

Increasing Racial Disparity in Infant Mortality:
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BACKGROUND

Although substantial declines in infant mortality have occurred across race/ethnic groups over the past several decades, the relative gap between blacks and whites has persisted. A central conclusion from a recent review of this topic is that "The analytic challenge for any assessment of disparities in infant mortality, therefore, is not merely to document that disparities exist but rather to explain why they persist in the face of enormous reductions in absolute levels of mortality" (Wise 2003:343).

More important for present purposes is the fact that racial disparities have not only persisted, but have actually increased, over the past 20 years. The black/white ratios for the infant mortality rate (IMR) and the neonatal mortality rate (NMR) stood at 2.0 and 1.9, respectively, in 1980, but by 1997, both ratios had risen to 2.3 (Guyer et al. 1998). The widening of the infant mortality gap has continued. For example, between 1998 and 1999, there was a decline of 3.8% in the death rate for white infants, as compared to a decline of only 0.3% among black infants (Hoyert et al. 2001: 5). Another largely unanticipated temporal shift is that the well-documented survival advantage of preterm and low birth weight black infants, compared to 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). Finally, although data are not yet available for multivariate analysis, the National Center for Health Statistics (NCHS) recently reported an increase in the IMR for the U.S. between 2001 and 2002—the first such increase since 1957-58 (Kochanek and Martin 2004).

These and other trends [1] support the conclusion by Gortmaker and Wise that "the past two decades have witnessed the most profound alterations ever recorded in the structure of infant mortality patterns in the United States" (1997: 152)—alternations fueled by dramatic technological advances in

perinatal care. These authors also warn that a substantial potential exists for a widening of the gap between black and white infant mortality because social and economic inequalities are likely to translate into differential access to preventative and curative innovations. The need for a clearer understanding of how these trends are related to technological advances and social processes constitutes the rationale for the general objective of the present research, which is to explore the reasons for the recent trends in both relative and absolute disparity in infant mortality by race/ethnicity in the United States. In pursuing this objective, we focus heavily, but by no means completely, on Respiratory Distress Syndrome for purposes of illustrating the relationships of interest and because of the implications of changing trends in death from this cause for the overall growth in relative black-white disparity**.** In later sections, comparisons are drawn with the Mexican origin population.

A thesis that has emerged primarily in the public health and medical literature is that a major reason for the diminution of the black infant survival advantage at short gestations and low birth weights is that whites have benefited to a greater extent than have blacks from the introduction of pulmonary surfactant therapy as an intervention aimed at reducing infant death from Respiratory Distress Syndrome (RDS). This, in turn, may have contributed substantially to the recent overall rise in the ratio of black to white infant mortality rates. RDS results from a deficiency of naturally occurring surfactant in the lungs of the fetus such that the functioning of the aveoli (air cells in the lungs) may be compromised and gas exchange may fail. [2] It is a largely (but not entirely) a problem of preterm (or low birth weight) 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).

Following approval of surfactant replacement by the U.S. Food and Drug Administration (FDA) in August 1990, there was a much greater relative decline in the white rate of infant death due to RDS than was the case in regard to black infant mortality from the same condition (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 (NCHS 1993: Table 2-5)—a rate ratio of 2.3. By 1999, the ratio had grown to 2.75, as the RDS infant mortality rate was 61.9 and 22.5 for blacks and whites, respectively (Hoyert et al. 2001: Table 28).

Based on an examination of clinical records from St. Louis area hospitals concatenated with data from the National Center for Health Statistics (NCHS) linked birth/infant death files, Hamvas et al. (1996) showed that the neonatal mortality rate (NMR) among very low birth weight (VLBW) white infants (in the Hamvas et al. study, infants born weighing 500-1500 grams) dropped sharply (41%) between 1987-89 and 1991-92; meanwhile, no significant change was observed among VLBW black infants. Although this comparison of rates over time was complemented by a logistic regression at the micro-level, there was no evaluation of the effects of demographic, social, or economic covariates in the Hamvas et al. research. The same is true of the Ranganathan et al. (2000) study which 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(2000: 455), but which included no social or economic risk factors (perhaps because of the changes in birth certificate items after 1988).

Interestingly, Hamvas et al. found that the black/white rate ratio was actually reversed from 0.7 (a black survival advantage) in 1985-87 to 1.3 (a white survival advantage) in 1991-92, a result not observed in other research on race differences in RDS mortality. [3] One reason for this difference may be that the relationships of interest are different in the St. Louis area than in other geographic locations. Further, the severity of risk of death from any cause, including RDS, is no doubt different when only VLBW infants are considered as compared to the risk when babies born at other or all weights are included in the analysis—as in the Malloy and Freeman (2000) research. In any event, it is unclear whether the kind of reversal in risk from a black RDS survival advantage to a survival disadvantage observed by Hamvas et al. (1996) occurred among infants nationwide.

An expanded investigation of race/ethnic disparities in infant mortality that includes both RDS and other causes is important, indeed crucial, for a number of reasons. Consider first the issue of RDS. From the mid-1970s to the mid-1990s, the decrease in infant death associated with RDS represents "the largest contribution among all potential causes of death to the overall decline in infant mortality" (Ranganathan et al. 2000: 454). While the black infant mortality rate in the U.S. has been substantially in excess of the white rate as far into the past as data exist (Buehler et al. 1987; Guyer et al. 1998), and although black infants are at higher risk of death at normal birth weight and term gestation, the gap would have been even greater if not for the existence of an offsetting survival advantage of black infants born at low weight and short gestation (Kline et al. 1989; Wilcox and Russell 1986, 1990). Of course, the compositional disadvantage so evident in the much higher proportion of low weight and short gestation births among blacks has, by far, overwhelmed the "return to risk" advantage at low birth weight (LBW). Nevertheless, if this long-standing advantage has been substantially reduced due to race differentials in the benefits from surfactant therapy, then the implications of recent changes in patterns of RDS mortality for the general rise in the black-white infant mortality ratio warrant further exploration.

Additional analyses emphasizing race differentials in RDS mortality are also needed because past research on this issue consists mainly of studies based on clinical data from one or a few hospitals in a limited geographic area or studies that, although nationwide in scope, do not adjust for the effects of social risk factors. Both types of studies have proven quite informative, but the former type is limited in generalizability and the latter tends to be rather descriptive in nature with a focus on RDS death rates and/or proportion of all infant deaths attributable to RDS.[4] As far as we can determine, there is little research on racial differences in RDS mortality that involves multivariate modeling of individual risk for the U.S. as a whole. [5] However, a recent study by Muhuri et al. (2004) included a multivariate model of cause-specific infant mortality by race/ethnicity between 1989-91

and 1995-97 and found substantial declines in risk from RDS, SIDS, and congenital anomalies, no significant change in mortality due to short gestation/low birth weight, and an increase in risk in the mortality of newborns associated with maternal complications. These authors found one major race/ethnic interaction, viz., a disproportionately large decline in risk of infant death among American Indians from SIDS and congenital anomalies. On the other hand, Malloy and Freeman conclude 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" (2000: 420).

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. While 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 well 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 infant mortality disparity. Moreover, the Gortmaker and Wise (1997) perspective seems quite general. In the past few decades many other innovations in neonatal care have emerged that, while more incremental in nature and impact than surfactant therapy, have, nonetheless, led to a substantial improvement in our capacity to preserve the life of infants at high risk of death from a wide range of conditions.

In this analysis, we concentrate on the increasing disparity in the relative risk of infant mortality between the non-Hispanic white (NHW) and non-Hispanic black (NHB) populations. (Hereafter, we use the terms "white" and "black" to refer to NHWs and NHBs, respectively, except where this might create ambiguities in comparisons.) We complement this by estimating models of absolute change in infant mortality. An exclusive focus on relative differences yields an incomplete picture both because it neglects the notable achievement represented by overall reduction in infant

mortality and leaves unexamined the conclusion that absolute declines have been uniformly greater among blacks, because black rates are so much higher and thus have "more room" to fall.

ANALYTIC AIMS

The general objective of our analysis is to provide insight into the question of why race/ethnic disparities in infant mortality persist, and in fact have increased, at the same time that large reductions were occurring in the absolute levels of infant mortality. Accomplishing this objective encompasses several specific aims including the following. (1) Black-White Comparisons: (a) documenting the black and white trajectories in infant mortality attributable to RDS and other causes between 1989-91 and 1995-98; (b) determining whether the kind of reversal in RDS mortality from a black survival advantage to a black disadvantage found in data from St. Louis hospitals also occurred nationwide, (c) estimating race and time period effects, along with the influence of socioeconomic, socio-demographic, behavioral, and biomedical risk factors, on black-white variation in infant mortality from RDS and other causes of death, and (d) modeling race differences in absolute changes in infant mortality from RDS and other causes. (2) Comparisons Involving the Mexican Origin Population: Inasmuch as the Mexican origin population, especially Mexican immigrants, on average have lower levels of education, less adequate prenatal care, and are less likely than either NHWs or NHBs to have insurance of any kind to pay for delivery (Frisbie et al. 1997), we repeat portions of the analysis, this time including Mexican origin births (divided by maternal nativity) in addition to non-Hispanic white and non-Hispanic black births. In this case, we (a) compare the raw rates and rate ratios and (b) estimate a preliminary model with all four groups analogous to that referenced in Aim 1c above.

THEORETICAL FRAMEWORK

Alternative Explanations

Although there is consensus that surfactant therapy has been more beneficial for white infants than for black infants, and thus may have contributed in an important way to the overall

increase in the black/white rate ratio, explanations for the differential in RDS trends vary. One possibility is that, at least at very low birth weights, black infants "respond less favorably than white infants" (Ranganathan et al. 2000: 458). Although Malloy and Freeman report a decline in the male/female risk of death differential from RDS, and note that this may be due to "a biologically mediated difference in the impact of surfactant on male infants compared to female infants" (2000: 419), they acknowledge the lack of evidence supporting biological explanations for the shift in black/white relative risk. The likely invalidity of an interpretation that relies on racial biological differences is underscored by the finding from clinical records of no significant difference between black and white RDS mortality among infants who received surfactant therapy (Hamvas et al. 1996). Indeed, there is little evidence that genetic factors are responsible for race/ethnic differentials in infant mortality in general (Wise 1993).

Another conceptual framework that has been specifically applied in the case of RDS mortality exists and can perhaps be most appropriately viewed as a "medical model." It 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. Support for this perspective is found in research showing that RDS "occurs less frequently, is less severe, and is accompanied by fewer complications in black preterm infants" (Hulsey et al. 1993: 572). The link between surfactant therapy and increasing black infant mortality disadvantage suggested most prominently in public health research has been succinctly delineated by Hamvas et al.: "Fetal pulmonary surfactant matures more slowly in white than in black fetuses, and therefore RDS is more prevalent among whites than among blacks" (1996: 1635)—a view 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] This

perspective need not be confined narrowly to explaining RDS differentials. It would seem quite applicable to other respiratory conditions that have been linked to pulmonary immaturity (Malloy and Freeman 2000), and likely to other causes of death for which preterm birth or low birth weight are prime risk factors.

The third explanation is one variant of a "social model" and appears to be the most general of the three alternatives. It is broader in the sense that it applies to all causes, not simply to causes of death that occur with greater frequency or severity in one population or another. And it seems equally pertinent to all advances in health care, whether these are incremental in nature or involve a single technological innovation. At the core of this conceptual framework is the proposition that, as advances in health care occur, the ability of individuals to reduce the risk of disease and death "is shaped by resources of knowledge, money, power, prestige, and beneficial social connections" (Link and Phelan 2002: 730). A facile application of the Link and Phelan formulation to infant mortality is complicated by their very broad definition of social conditions "as factors that involve a person's relationship to other people…(and thus) "in addition to factors like race, socioeconomic status, and gender, (these authors) include 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" (Link and Phelan 1995: 81). Many of these factors apply to adults (as is appropriate in a general theory of the relationship between social resources and health), but hold little direct relevance in the case of infants. Of course, a woman's health may well be compromised by adverse social circumstances, and thereby negatively and indirectly influence the processes of pregnancy and parturition. We can operationalize some of the more important maternal attributes in a fairly straightforward manner. Often, however, the effect of deleterious social conditions must be captured by measures of the mother's health, per se. No data set of which we are aware even approaches comprehensive coverage of the wide range of social risk factors discussed by Link and Phelan. On the other hand, knowledge of whether individuals are in certain categories known to be socially and economically

disadvantaged (e.g., race/ethnic minorities, unmarried women who give birth, teenagers who give birth), can be used to proxy social inequality.

As implied earlier, Gortmaker and Wise (1997) provide what is perhaps the most directly salient conceptual framework in that it focuses on infant mortality and draws to some extent on both medical and social models. These authors warn 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 innovations. This does not imply that a high risk black infant will be denied surfactant therapy or any other intervention. Rather, we understand the Gortmaker and Wise argument to be that socially disadvantaged groups are less likely to be integrated into the formal health care system and/or less likely to have the information, the social networks, and the economic wherewithal to acquire access.

A data set with a very 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, in general, and on specific causes of death, in particular, can be derived. This essentially means recourse must be made to vital statistics. These data do not allow us to unambiguously determine whether "differential need for intervention" or "differential access to intervention" constitutes a more valid explanation. However, we can at least assess whether the results based on comparisons of relationships over time are consistent with the view that innovations in perinatal technology are associated with greater racial disparities in infant mortality. While certain technological innovations pertain most directly to specific ailments, there have been tremendous improvements in neonatal care in general. The benefits of these improvements appear to have been unevenly distributed at least in the sense that infants (especially LBW infants) born at tertiary care hospitals or at regional neonatal intensive care centers, have a greater probability of survival than those without access to such facilities (Horbar and Lucey 1995).

What sort of empirical results would support the general notion that an increase in the black-white racial gap in infant mortality is associated with advances in health technology? We should, first of all, be able to replicate with our large and somewhat more up-to-date data set the basic bivariate finding from previous research, viz., that the ratio of black-to-white relative risk of death from RDS is greater in the post-surfactant period than in the pre-surfactant period. Given the general improvement in the capacity to preserve the life of high risk infants, epitomized by a wide range of interventions that are increasingly available in neonatal intensive care units (NICUs), the same pattern would be expected with respect to many other causes of death, as well. Still, if surfactant therapy had the sort of major impact postulated, it is to be anticipated that estimates from multivariate analyses will show that the black risk of infant death from RDS in the post-surfactant era was larger relative to that of their white counterparts, than was the case prior to FDA approval of this intervention.

We believe that the differential need for intervention explanation and the differential access to intervention explanation**s** should be viewed as complementary, rather than competing, hypotheses. It is difficult to discount, nor do we wish to discount, the evidence from public health and medical studies that black infants, on average, have less need for surfactant therapy because black fetuses mature at lower birth weights and shorter gestations, and thus (on average) the frequency and severity of RDS and other respiratory ailments are less among LBW black babies than among their white counterparts. Results from our multivariate analysis for the entire U.S. showing a nationwide reversal from a relative black "pre-surfactant" RDS survival advantage to a "post-surfactant" RDS survival disadvantage, would be consistent with the differential need interpretation. But since we know that a large number of black infants <u>are</u> in need of surfactant therapy or other interventions, socially limited access could well exacerbate any disparity produced by differential need. Further, to the extant that a black disadvantage relative to whites in regard to deaths from both RDS and other causes declines,

following adjustment for social risk factors in both time periods, the differential access to health care hypothesis is also supported.

There is considerable interest in Mexican origin infant mortality as seen in the large and growing literature on this topic. For this reason, and because our preliminary work including this population should be of some use in evaluating the two explanations, we later present selected findings comparing the mortality of infants born to Mexican American and Mexican immigrant women between 1989-90 and 1995-98 to that among their non-Hispanic white and non-Hispanic black counterparts. [7] For example, we are not aware of any research that suggests that differentials in timing of fetal maturation distinguish those of Mexican origin, but it does appear that such infants are more at risk for low weight and preterm birth than are NHW infants (Frisbie et al. 1998; Frisbie and Song 2003). Given the obstacles to access to medical care faced by the Mexican American population mentioned above, the fact that immigrants face even greater obstacles than their U.S.-born co-ethnics, and that immigrants, in general, are less apt to receive timely health care, (LeClere et al. 1994), it seems reasonable to expect that, whatever the risk of RDS mortality for this group relative to NHWs in the pre-surfactant era, the change in risk for infants born to Mexican immigrant women in the post-surfactant era will be less favorable than that for NHWs—if access to intervention plays a key role.

At this juncture, it is useful to bear in mind the limitations of the data. For example, even if social factors were completely preeminent, controls for these risks would not result in a zero differential between the risk of majority and minority infant mortality, except in the unlikely event that all important covariates are known, measured, and actually included in the models being estimated. Nor, in the absence of clinical data, can we reject the differential need explanation**.** Nevertheless, based on "before and after" comparisons, we believe we can shed at least some light on these important issues.

Risk Factors

Basic demographic covariates for this analysis include maternal age, marital status, and nativity, along with parity, plurality, and sex of infant. Infant mortality risk is higher for infants born to teenagers (Singh and Yu 1996), and this relationship, net of controls, is observed for both endogenous and exogenous causes of death (Moss and Carver 1998) and across most race/ethnic groups (Hummer et al. 1999). Maternal age needs to be considered jointly with parity since 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). 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 due to the positive selection of migration (Palloni and Morenoff 2001; Frisbie 2004), though some authors ascribe the immigrant advantage to cultural differences (Cobas et al. 1996; Scribner 1996).

Perhaps because it is so well-established that plural births are at higher risk than singletons, research on pregnancy outcomes is often limited to the latter. However, "Respiratory Distress Syndrome (RDS) is the major cause of morbidity and mortality in preterm twin deliveries" (Turrentine et al. 1996: 351). Interest among demographic and public health researchers in plural births has been heightened by the finding that a major reason for 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 to white women—but this phenomenon is not observed among black women (Demissie et al. 2001; see also Blondel et al. 2002). Rates of LBW and preterm birth have also increased for Hispanics in recent years (Frisbie and Song 2003). For all these reasons, the most appropriate strategy for this analysis would seem to be to include multiple births and then control for plurality in our regression models.

We also control for sex. Male infants are less apt to be born at low birth weight, but are consistently more likely than females to die in the first year of life (Frisbie et al. 1998; Moss and Carver 1998).

Note that maternal age and marital status, although typically categorized as demographic variables, can be usefully 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 disadvantaged social conditions beginning when these young women were themselves children (Geronimus 1987; Geronimus and Korenman 1993). And, few would argue that existence of a marriage certificate in and of itself has any effect on health. Indeed, the higher mortality rate among infants born to unmarried women is generally considered to be a reflection of inadequacy of social and economic resources and/or life-style differences (Cramer 1987; Eberstein, Nam, and Hummer 1990; Hummer et al. 1999).

Other somewhat more direct indicators of access available in our data set are maternal education and prenatal care. In general, infant mortality risk decreases as maternal education rises. In addition to being an indicator of SES, maternal education may reflect knowledge of medical services and of how to circumvent obstacles to access. The education effect 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 (PNC) 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 based on 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, PNC is included in the present analysis because it represents a "package" of health related services highly relevant to pregnant women (Alexander et al.

1999; Shiono and Behrman 1995). If receipt of PNC 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 parturition.

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, previous pregnancy loss and presence of maternal medical risks (such as hypertension, anemia, diabetes [both chronic and pregnancy-related], eclampsia etc.) are included as covariates. The same is true of complications of labor and delivery (Hummer et al. 1999), and we also control for this risk factor (e.g., excessive meconium, premature rupture of membranes, placenta previa, etc.).

It has been established that 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 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 in general [8] and mediate the influence of many other risk factors (Cramer 1987; Hummer et al. 1999; Kline et al. 1989; McCormick 1985).

DATA

The data employed are the NCHS linked birth/infant death cohort files for the years 1989-1990 and 1995-1998, which include all infants born alive in the U.S. during those years. The data set consists of 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 (1989-90) and the post-surfactant period (1995-98). We include 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 showed only a slight difference in RDS mortality between 1989 and 1990.

Linked cohort files do exist for years prior to 1989, but they are far less rich than those from later years. For example, Hispanics cannot be distinguished prior to 1989, and no information at all 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.

The proportion of cases that had to be deleted due to missing data on covariates varies over time (17.7% in 1989-90 and 7.1% in 1995-98). We do not believe this leads to any serious distortion of our results. For example, following deletion of cases with missing data, the black-white IMR ratio based the linked files for 1995-98 stands at 2.3—identical to the 1997 ratio reported by Guyer et al. (1998). Other rates calculated from our data are also similar to those reported from vital statistics reports over the two time periods. Nevertheless, it is logical to suppose, and consistent with our diagnostics, that information is more apt to be missing for cases in which pregnancy outcomes are highly adverse and among minority groups who are less likely than whites to have adequate health care. This implies that errors in estimation will be "conservative"--i.e., the degree of racial inequality is likely to be even greater than our estimates are able to demonstrate. Information on smoking and weight gain is not available for a few states (e.g., California). Here, the conventional strategy of assigning a missing category for these covariates was adopted (Frisbie et al. 1998; Muhuri et al. 2004; Singh and Yu 1996).

METHODS

Measures

The outcome variable consists of three categories: infant death due to RDS, infant death from other causes, and infant survival. We focus primarily on non-Hispanic whites and non-Hispanic blacks and, to a more limited extent on the Mexican origin population. As recommended by NCHS, cases are categorized according to maternal race/ethnicity.

As will be seen in Table 2, most risk factors are measured in a conventional manner. However, the measures of prenatal care and parity warrant some elaboration. Prenatal care is 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 record more PNC visits than the standard 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 is measured via the Kleinman-Kessel index (1987) which combines maternal age and birth order to reflect the interaction of these two variables (see also Hummer et al. 1995). Our diagnostics indicate no problem of collinearity involving the Kleinman-Kessel measure and maternal age.

Models

Multinomial logistic regression is used to model the three-category outcome. Results are presented in the form of odds ratios. Because our data set consists of all vital events, the conventional reason for 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 estimated. Nonetheless, tests of significance retain utility "in order to rule out the simple 'chance processes' alternative" (Blalock 1979: 242).

Problems 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, including the present analysis, based on 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 lack of information on whether surfactant therapy and most other interventions were administered. [9] However, the linked NCHS files permit a reasonable "before and after" indirect test in that data (collected and coded with consistent protocols) exist on either side of the date at which surfactant therapy came into general use.

Two "basic" sets of models were constructed. They supplement each other in that both evaluate race/ethnic disparities over time, and each provides information not available in the other. The first set estimates the risk of infant death separately for the pre-surfactant (1989-90) and post-surfactant (1995-98) time periods. The second set pools the data over time and includes time period as a dummy variable. Evaluating the relationship separately for the two time periods allows an assessment of the race differences in risk without averaging of effects over time. Moreover, the results are more directly comparable to previous studies in which findings are often compared for separate years or groups of years. On the other hand, pooling the data over time allows the estimation of the magnitude and significance of the time period main effect and of a race x time period interaction term.

Inasmuch as we are interested in race disparities in infant mortality overall, it is necessary to analyze infants born at all weights. But because RDS is largely a condition that occurs among LBW or preterm infants, much previous research on RDS focuses either on low weight, or very low weight births. [10] Furthermore, LBW and short gestation are associated with higher risk from many other causes of infant death. Hence, we extend our analysis to, and present tabular results from, a second phase in which only LBW infants are included. These additional regressions parallel the "basic" models in that two sets of regressions were again performed: one for the separate time periods and

one with time period as a covariate. Models restricted to low weight infants do not include birth weight or gestational age as risk factors. [11]

DESCRIPTIVE RESULTS

Documenting Changes Over Time

The trends in non-Hispanic white and non-Hispanic black infant mortality rates between 1989-90 and 1995-98 are shown in Table 1 separately for all births (Table 1a) and low weight births (Table 1b). [12] As seen in Table 1a, the infant mortality rate per 1000 live births for both RDS and other causes dropped for both groups over time, with the decline in RDS rates being much the greater of the two. The percentage decline in rates was greater for whites than for blacks in every comparison, although differences are fairly small. The final two columns in the table show that the relative differential (rate ratio) increased, particularly for RDS mortality. The results are similar, but the race disparity is even more striking, when attention is limited to LBW infants (i.e., infants born weighing < 2500 grams). From Table 1b, we see that the race differential in the percentage decline in all rates was considerably greater among LBW infants. Moreover, not only did the black/white rate ratio increase for all causes, but also what was a very slight black advantage with respect to RDS mortality in 1989-90 had become a fairly substantial disadvantage by 1995-98.

--Table 1 about here--

The distribution of risk factors by race and time period appears 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 young teenage, unmarried, and high parity categories. Educational levels are lower among blacks. They are slightly more apt than whites to have had a previous pregnancy loss, to present with medical risks, to have complications of labor and delivery, and much more likely to gain only a small amount of weight during pregnancy and to receive prenatal care that is less than adequate. And, of course, the incidence of preterm and low weight

births is considerably greater among blacks. However, black women are less likely to smoke during pregnancy than are white women. In order to conserve space, tabulations of Mexican origin risk factors are not shown. However, it should be noted that, while this group, on average, is intermediate to whites and blacks with respect to most risk factors, the Mexican origin population has lower levels of education and is less apt to receive adequate prenatal care than are either whites or blacks.

--Table 2 about here--

RESULTS FROM REGRESSION MODELS: COMPARING BLACKS AND WHITES

We begin the discussion of multivariate results with comparisons of infants born at all weights and later compare these results with those obtained among LBW infants. Because so much of the literature on RDS differentials concentrates on black-white disparities, the largest portion of the analysis deals with only these two groups. The supplemental analysis that includes the Mexican origin population appears in later sections.

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

The Pre-Surfactant Period

The risk of black, as compared to white, infant mortality (at all birth weights) from RDS and all other causes is examined in Table 3 for the pre-surfactant period (1989-90) in terms of five models that progressively adjust for risk factors. The first two columns display the familiar bivariate relationship in which the odds ratios associated with death from both RDS and other causes are more than twice as high for black infants. With socio-demographic covariates controlled (Model 2), the black risk, is considerably reduced (ORs of about 1.6). The magnitude of the decline is not unexpected in that, e.g., black infants are much more likely to be born to teenage and unmarried mothers—clearly socially and economically disadvantaged populations. Following adjustment for education and prenatal care (Model 3 of Table 3), the risk of infant death among blacks from RDS and other causes 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 black odds, as ORs are now only 1.273 and 1.378 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 white risk. Moreover, the odds of a black infant dying from RDS become significantly lower than the white odds (OR = 0.796). In general, the results support the view that social and health conditions are influential determinants of black-white inequality in infant mortality. In particular, the results demonstrate that, once social and health covariates are controlled, black infants were less at risk than white infants from death due to RDS during the pre-surfactant period.

--Table 3 about here--

Table 3 also shows that the effects of most risk factors are consistent with virtually all previous research and the conceptual model discussed above, thereby rendering unnecessary a detailed discussion of these effects. However, a few of the relationships warrant at least brief mention. First, the odds ratios for race differences 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. The magnitudes of the effects of gestational age and birth weight are, of course, the largest. Further, their effects are much greater in the case of RDS than for other causes of death, thus reconfirming the strong association of RDS with early and light birth. The magnitude of the effects of most risk factors on infant death is substantially smaller with gestational age and birth weight controlled, as anticipated in light of the mediating role typically played by these birth outcomes. Thus, it is perhaps not surprising to find that the effects of PNC, smoking behavior, and weight gain are reduced and sometimes reversed because their relationship with infant mortality has often been shown to be indirect through gestational age and birth weight. It does seem anomalous that the smoking coefficient 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. In addition, recent research suggests this result may be a statistical artifact. Specifically, a similar finding has been reported in a twin study by Almond, Chay, and Lee who note 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" (2003: 33).

The Post-Surfactant Period

The patterns of risk factor effects are similar in both time periods. However, the estimates of racial disparity in the post-surfactant period 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 somewhat higher in 1995-98 (OR = 2.758) than in 1989-90 (OR = 2.361). In fact, in every model, the post-surfactant odds of death from RDS among blacks are considerably higher in 1995-98. To illustrate, controlling socio-demographic variables, the estimate of the risk of RDS death for blacks was about 65% higher than that for whites prior to the widespread use of pulmonary surfactant (Model 2 of Table 3). In the post-surfactant era, the analogous model estimates the relative odds to be over 120% greater (Model 2 of Table 4). In Model 4, which adjusts for all risk factors, except gestational age and birth weight, the black disadvantage was moderate in 1989-90 (OR = 1.273), but by 1995-98, the OR had increased to a value of 1.672. The same pattern of results obtains in regard to infant death from other causes, but the size of the differences in odds ratios between the two points in time is smaller compared to the risk associated with RDS. Finally, in the full model (Model 5), the black-white RDS differential 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 = 0.796). After the introduction of surfactant therapy, the analogous model shows that the risk for black infants became significantly higher than the risk for white infants (OR = 1.109). The odds of death among black infants from all

other causes, which were virtually identical to the white odds in 1989-90 (OR = 1.002), became significantly higher in 1995-98 (OR = 1.144).

--Table 4 about here--

The results of regression models constructed separately by time period for infants born at all weights provide some support for both the differential need for intervention and the differential access to intervention hypotheses. In Tables 3 and 4, we see that, while the bivariate estimate (Model 1) showed risk of black infant death to be roughly two and one-half times greater than the white risk, controls for social and maternal health factors (with the latter themselves quite likely to influenced by social processes) reduced the disparity considerably in both time periods—a finding consistent with the notion that social inequality restricts access to innovations in health care. On the other hand, the fact that the black-white risk differential was consistently greater in the post-surfactant period (especially for RDS mortality), as well as the reversal from a black RDS survival advantage before surfactant replacement became widespread to a survival disadvantage subsequent to that innovation, is consonant with, though it does not establish the validity of, the differential need for intervention explanation.

Multivariate Results: Low Birth Weight Infants in the Pre- and Post-Surfactant Periods

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

Inasmuch as previous research has consistently found a black survival advantage at very low birth weights, it is to be expected that the relative disparity between LBW black and white infants will be smaller than that observed among all infants. Indeed, this is exactly what we find. In Table 5, the odds ratios contrasting the mortality risk of LBW black infants with the risk for their white

counterparts are again shown separately by time 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), we see that, in every instance, the odds of black infant death are lower in Table 5. We also see that the change in racial disparity shifted more dramatically among LBW infants between 1989-90 and 1995-98 than was the case for all infants. In the earlier time period, the bivariate estimate (Model 1) showed that the risk for LBW black infants from RDS was statistically identical to the risk for LBW white infants. In each of the other models, the risk of a black death from RDS is lower than the white risk, with the full model (Model 4 of Table) showing that the odds for black infants were about 18% lower in 1989-90. The odds ratios associated with all other causes of death were modestly and significantly higher among black infants, except in Model 4 where there was no significant difference (OR = 0.991).

--Table 5 about here--

The findings for the post-surfactant period (1995-98) present a sharp contrast. Although the odds ratios decline in magnitude as controls are added, in every model, black infants, relative to whites, were significantly more likely to die from RDS and all other causes. Thus, it is clear that for low weight births, just as for births at all weights, over the decade of the 1990s, the former black advantage with respect to RDS at low birth weights was reversed, and the relative disparity in black-white infant mortality from all other causes increased.

Multivariate Results: Time Period as a Covariate

In order to quantify the time period effect, and to more precisely determine the extent to which the risk of death among black infants increased in the 1990s decade, we pooled the 1989-90 and 1995-98 data and applied the same regression models on which earlier tables are based. Table 6 displays the odds ratios for race, time period, and a race x time period interaction for all models, first for births of all weights (Table 6a) and then for low weight births only (Table 6b). In order to conserve space, and because, again, the pattern of other risk factor effects is very similar to those

estimated in the analogous models discussed earlier, only the odds ratios most relevant to the issue of changes in race disparities are included in Table 6.

All Birth Weights

Among all births, we once more find that the odds of dying in the first year of life from either RDS (OR = 2.559) or all other causes (OR = 2.261) are more than twice as great among blacks (Model 1 of Table 6a). In Model 2, we observe that the risk from RDS was approximately 55% lower in 1995-98 (post-surfactant) than in 1989-90 (pre-surfactant), while the odds were reduced by about 17% in the case of mortality from all other causes. This is as would be expected inasmuch as surfactant replacement is an innovation aimed, for the most part, at reducing RDS mortality. Inclusion of the time period variable has virtually no impact on the odds ratio for blacks in Model 2. However, when controls are imposed for all risk factors, including birth weight and gestational age, the disparity by race shifts dramatically to a black advantage with respect to RDS (OR = 0.937), along with a sharply reduced black risk from all other causes (OR = 1.088). The most striking findings in Table 6a emerge from Model 4 which adds the race x time period interaction. In this model, the odds of black infant death due to RDS are 20% lower than the odds for their white counterparts, and the race differential for all other causes becomes insignificant. Also, the odds of death in the later time period, compared to 1989-90, are reduced even further (OR = 0.345 for RDS and OR = .758 for other causes). Even more notable, the interaction term in Model 4 of Table 6a indicates that, for RDS-implicated mortality, the relative risk of being black was almost 40% greater in the post-surfactant period than in the pre-surfactant period (OR = 1.386). The direction of effect is the same for all other causes, but the effect is of smaller magnitude (OR = 1.129).

--Table 6 about here--

Low Weight Births

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, viz., the black-

white disparity is much less among infants born at low weight. In Table 6b, e.g., the risk of a LBW black infant dying from RDS is about one-fifth greater than the risk for LBW white infants (OR = 1.215), as contrasted with an odds ratio that is more than double the white risk among births at all weights (compare Model 1 in Tables 6a and 6b). Just as was the case among all infants, the relationship among LBW infants shifts to a significant black survival advantage with respect to RDS in the full model (Model 4, Table 6b), while for other causes, a small, but insignificant, black disadvantage is observed. Finally, for LBW infants, the interaction term also indicates that the relative risk for blacks was much greater in 1995-98 than in 1989-90.

ABSOLUTE CHANGE IN INFANT MORTALITY AMONG WHITES AND BLACKS

When one compares the absolute decline in infant mortality rates over time for births at all weights, the decline for blacks exceeds that for whites. This is unsurprising in that the much higher rates observed among blacks have much farther to fall. However, as shown in Table 1, the absolute drop in the empirical (raw) rates for LBW infants was actually greater for whites. To illustrate, the RDS rates among white LBW infants in 1989-91 stood at 9.98 per 1000 live births. By 1995-98, the rate had fallen to 3.66—an absolute decline of 6.32. Among LBW blacks, the reduction in rates over the same period was from 9.92 to 4.95—an absolute decline of only 4.97. Further, the extent to which absolute change patterns may vary with adjustment for risk factors is unknown.

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 differentials 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 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 time periods 1989-90 and 1995-98, 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 race 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 year dummies 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. [13] 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 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.

Note the analysis of absolute change shown here pertains only to singletons, while all analyses until now included both singletons and plural births. What is reported in this part of our paper draws on a complementary analysis limited to singletons. There is nothing in the procedure that would prevent its application to all births.

Again, these estimates are odds, not rates, but they serve as reasonable approximations of rates of change. To illustrate, in our analysis of singleton births, the observed rate of RDS mortality is 0.42 per 1000, and the mortality rate due to other causes is 5.75 per 1000, which translate into probabilities of 0.00042 and 0.00575 respectively. Let p_{RDS} and p_{OTH} denote the probabilities of death due to RDS and death from other causes respectively. The probability of survival is $p_{SURV} = 1$

$-(p_{RDS} + p_{OTH})$. The multinomial logit for mortality due to 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 1000) for RDS and 0.005786 (5.786 per 1000)—values more than close enough 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 from Logit Approximations of Absolute Change

The next set of tables below show absolute changes in approximated rates expressed per 1000 live births for each year compared to 1989 as the reference year for singleton births for both all weight and LBW infants based both on 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 ($p \leq .05$), a Z value of 1.96 is required; for $p \leq .01$, Z must be greater than, or equal to, 2.58.

Table A-1 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 all other cause mortality, the changes closely reflect what is observed in the distribution of empirical rates, including consistently greater absolute declines for blacks. Models with full controls present a different picture. In the case of RDS, the estimated absolute decline is greater for whites than for

blacks for every comparison except one, viz., for the year 1998 where the drop in rates is identical for both races. For deaths from other causes, the pattern is less uniform, with sometimes one race group, and sometimes the other, recording the greatest decrease.

--Table A-1 about here--

Table A-2 replicates the analysis for LBW infants. Two notable differences in the findings from Table A-2 demonstrate the utility of a separate consideration of infants born at low weight. For these high-risk infants, in the bivariate models for both RDS and other causes, the improvement for whites exceeded that for blacks—just as was true of the relative differentials 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 where the magnitudes of white reductions exceed those for blacks. The changes between 1989 and 1990 do not approach significance in three of the four comparisons.

--Table A-2 about here--

Tables A-1 and A-2 evaluated changes separately by race and tested to see if the differences were significant. By contrast, Tables A-3 and A-4 address the equally important (and, in this context, perhaps more important) question of the direction, magnitude, and significance of black-white differences in changes in the rate approximations. That is, models in the latter set of tables begin with the differences shown in Tables A-1 and A-2, and then evaluate how these differences changed from year to year. Specifically, Tables A-3 and A-4 analyze second differences in which the white change was subtracted from the black change so that 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 (Table A-3) reflects what is already known about trends in rates available from NCHS vital statistics reports, viz., 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.

--Table A-3 about here--

When the relationships of interest are modeled for the LBW infants (Table A-4), we see that, in the bivariate models for both RDS and other causes, mortality declines, in every instance, reflect a white advantage (as indicated by the negative signs). Thus, the trend among LBW infants seen in the raw rates in text Table 1 (and in our complementary analysis of singletons) is reflected in Table A-4. Controlling for risk factors, however, there are no significant race differences. The bivariate model for all other causes of death that occurred to LBW infants evidences the same pattern—i.e., whites had larger decreases than did blacks, most of which are significant. A slight white advantage remains, net of the effects of risk factors, but none of the differences is statistically significant.

--Table A-4 about here--

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 black rates surpass those of whites for infant mortality due to RDS and all other causes. Thus, it is important to recognize that, while 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 race/ethnic groups. However, when considers 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 effects are never statistically significant. In general, then, it appears that those who would shrug off the increase in the relative black-white infant mortality gap by pointing to the greater absolute improvements for blacks need to rethink their conclusions, at least with respect to those births at greatest risk, viz.,

LBW (or short gestation) infants. The need for additional attention is especially great because even with the modest rise in the rates of low weight and premature births among whites and the small decline in rates of these adverse birth outcomes among blacks, the latter continue to be characterized by an exceedingly high prevalence of low weight and preterm births—a compositional disadvantage that is offset only to a minor extent by the somewhat higher survival rates of LBW and short gestation black infants.

THE MEXICAN ORIGIN POPULATION: SELECTED COMPARISONS

In this section, we present a description of the change in RDS and other cause mortality rates of infants born to Mexican American and Mexican immigrant women in the U.S. between 1989-90 and 1995-98, plus selected models of the risk associated with race/ethnicity, time period, and their joint (interaction) effect. The data are the same as that used in the bulk of the analysis (NCHS linked birth/infant death cohort files for all births). We add the Mexican origin population to the analysis for two reasons. Interest in the epidemiologic paradox as it applies to Mexican American infants (including those born to Mexican immigrant mothers) has remained high. In addition, given the documented low level of access to the formal health care system of Mexican Americans (Frisbie et al. 1997), and immigrants in general (LeClere 1994), at least some additional insight into the role played by access to intervention in regard to risk of death from RDS and other causes should be gained by expanding the analysis in this manner.

The RDS death rate (per 1000 live births) among infants born to Mexican American women was similar, but in all cases, a bit higher than non-Hispanic white rates in both 1989-90 and 1995-98, and the relative gap increased from a ratio of 1.05 to 1.17 over this time period. Rates for all other causes followed a similar pattern (Table 7). However, infants of Mexican immigrant women recorded lower rates from both RDS and other causes in both time periods and showed a relative decrease in risk over time compared to non-Hispanic whites. These findings are clearly in line with what is by now a quite large literature reporting lower death rates in the Mexican origin

population (and especially among Mexican immigrants) than would be expected given the disadvantaged socioeconomic and health characteristics of this population. As seen in earlier tables (and previous literature), infant mortality, including that attributable to RDS, declined substantially over the 1990s.

--Table 7 About Here--

A series of logistic regression models of individual risk, presented as Table 8, compare the individual risk of death of infants born to U.S.-born and Mexican immigrant women with the risks for non-Hispanic blacks and non-Hispanic whites. For the most part, the models are analogous to the black-white comparisons shown in Table 6, with Table 8a examining births at all weights and Table 8b focusing on low weight births. In the bivariate model, the odds of a black infant dying in the first year of life are considerably more than double the odds for whites. Risks for infants of Mexican American women are higher than the white risk (significantly so in the case of other causes), but the magnitude of the difference is slight (ORs of 1.032 and 1.080 in the case of RDS deaths and deaths from other causes, respectively). In sharp contrast, risk of RDS death is almost 28% less for infants of Mexican immigrants as compared to infants born to non-Hispanic whites, and odds associated with other causes are 14% lower. Adding time period as a covariate shows that the odds of infant mortality were significantly lower in 1995-98 relative to 1989-90. The OR associated with RDS death is quite low, as would be expected in the post-surfactant period if this intervention had the kind of major beneficial effect indicated in the literature. Model 3 adds the race x time period interaction term, which shows that the risk of death for infants born to NHB or Mexican American women is slightly elevated over and above the main effects, while the odds for infants of Mexican immigrant women are slightly lowered. However, the magnitude of the interaction is small and usually not significant.

--Table 8a About Here--

Model 4 of Table 8a adjusts for all other covariates, including birth weight and gestational age, but does not include the interaction term. The net effect of time period is somewhat more pronounced in this model, but the most notable change is that, net of all other main effects, the odds of infant death are lower for minority infants, as compared to the reference group in every instance, save for the risk of death of black infants from other causes. When the interaction of race/ethnicity with time period is introduced (Model 5), the risk of RDS death for both black and Mexican American infants becomes about 20% lower, while the ORs are only slightly lower for all other causes. Only in the case of the babies of Mexican immigrants does the risk increase, but the OR remains less than unity (not significant). Once again, the interaction effect is rather striking. In the case of RDS mortality, being black in 1995-98, (as in Table 6a) was about 40 more risky than in the pre-surfactant period, thereby offsetting the main effects, while for infants of Mexican American women, the interaction adds 25% to the risk. By contrast, the interaction involving infants of Mexican immigrant women is small and not significant.

Turning to LBW infants (Table 8b), the pattern of effects is very similar to that seen in Table 8a, including the direction and magnitude of the time period effect. Also, just as in Table 6, the magnitude of the odds ratios for NHBs are much smaller for LBW infants compared to infants born at all weights. Looking at Models 2 and 3, in general and relative to NHWs, the ORs for LBW infants of Mexican American women are slightly more favorable, and those for infants of Mexican immigrants are somewhat less favorable when juxtaposed with the analogous odds shown for infants born at all weights. In Model 4 (controlling for main effects of all covariates) the risk of RDS mortality is significantly lower for each of the three minorities. Finally, the results of Model 5 in Table 8b are highly similar in direction, magnitude, and significance to the effects for infants born at all weights. Even the interaction terms are similar, with the risk being heightened among blacks and Mexican Americans by roughly the same amount. One exception is seen in the interaction term for

infants of immigrant women (OR = .811), which indicates that the risk for such infants from RDS is 19% (and significantly) lower in 1995-98.

CONCLUSIONS

Conclusions Based on comparisons of Non-Hispanic Blacks with Non-Hispanic Whites

The much greater percentage decline in the RDS-specific mortality rate and the more substantial rise in the black-white RDS rate ratio, in comparison to mortality from all other causes, leaves little room for doubt that a reduction in RDS mortality, associated with the advent of surfactant therapy, played a prominent role in both the nationwide decline in the IMR and in the widening of the relative racial gap in infant mortality (Table 1). Multivariate analyses conducted separately by time period and for LBW births, as well births at all weights, indicated a general reversal such that, in the case of RDS, what was a small black survival advantage in 1989-90 became a moderate disadvantage by 1995-98, while in regard to all other causes, statistical equality of risk changed to a significantly higher risk of death for black infants (see full models in Tables 3, 4, and 5). This conclusion was strongly affirmed by the demonstration that blacks were at much higher risk in 1995-98 than in 1989-90 (i.e., by the interaction term in Table 6).

The results provide some support for the conclusion that the differential need for intervention and the differential access to intervention hypotheses are most appropriately viewed as complementary. The theoretical framework emphasizing the importance of social determinants was supported in that controls for such factors reduced the black infant mortality disadvantage in regard to both RDS and all other causes both before and after the advances in perinatal care that occurred in the 1990s. Furthermore, regardless of the extent to which one is willing to conceive of differentials in maternal health endowments to be a function of social inequity, it seems obvious from the strong relationship between indicators of poor maternal health and infant mortality risk that one important means of reducing adverse pregnancy outcomes is to devote more attention and resources to the health of women (as also suggested by Gortmaker and Wise [1997]). Nevertheless, public health

and medical research has adduced strong (though not completely unambiguous) evidence that maturation occurs earlier for blacks than for whites in the process of gestation. Our findings showing a more dramatic change in the risk of black from RDS than from other causes, including the reversal of what was, formerly a black survival are consistent with this reasoning. Moreover, infants born less than fully developed as indicated by LBW or short gestation are almost surely at greater risk from most, if not all, causes of death. Even with respect to exogenous causes (those attributable to environmental or external causes after parturition, such as infections, accidents; see Bogue 1969), it is reasonable to suppose that LBW infants are less able to fight off infection and perhaps more susceptible to fatal accidents. If so, more rapid fetal maturation may connote at least some small degree of black survival advantage at LBW from virtually all causes of death. Thus, blacks, *ceteris paribus*, may be less apt than whites to need interventions designed to prevent or remedy a wide range of morbid conditions associated with preterm birth. However, there is every indication that things are <u>not</u> equal, and it seems quite likely that infants from a socially disadvantaged group will have greater difficulty in obtaining access to innovations in health care.

Our analysis based on logit approximations of rates unfortunately suggests that, even in the case of absolute change, blacks suffer some degree of disadvantage. Blacks did experienced a greater absolute decline in rates than whites for infants born at all weights. However, absolute gains in survivorship among low weight births were greater for whites than for blacks in the bivariate model. This is disturbing in that the absolute black-white disparity increased among the highest risk infants. The full model indicates that there would be little difference between whites and blacks if inequalities in risk factors could be eliminated.

Finally, we would be remiss if we did not elaborate on a point briefly raised in the Background section. Disparities in infant mortality (and other) rates are the result of differences in composition as well as differences in "return to risk." Thus, while the infant mortality risk at low birth weights may be less among blacks (i.e., a more favorable "return to risk" as compared to

whites), blacks are immensely disadvantaged in compositional terms due to the much greater proportion of black births that occur at low weights and short gestations—adverse birth outcomes which are the most powerful predictors of infant death from numerous causes. In this very important sense, then, the need of black infants for access to health care interventions must be at least as great, and likely much greater, than the need of white infants. Considered in this context, and further taking into account that blacks are far more disadvantaged in terms of their risk profile than are whites, and that controls for social factors (as broadly conceived by Link and Phelan) substantially diminish the race disparity in risk, we believe our results fairly strongly support the view that, whatever differences in need for intervention exist, technological advances are likely to give rise to even greater racial inequality in infant mortality because differential access to health interventions occurs in the presence of persistent social inequality.

Conclusions Regarding the Mexican Origin Population

Descriptive tabulations show that the difference between NHW and Mexican origin infant mortality did not change greatly over the period from 1989-90 to 1995-98. This was partly because the difference was small initially, and partly because the relative improvement (compared to NHWs) for infants born to Mexican immigrant women was partially offset by a modest expansion of the gap for infants born to Mexican American women. The most striking findings from the inclusion of the Mexican origin population in the logistic regression analysis were as follows: (1) The bivariate model showed that, while the odds of death for babies born to Mexican American mothers were slightly higher than the odds for their NHW counterparts, the risk for infants born to immigrant mothers was substantially lower. (2) In the full model, main effects indicate that the odds of death for all minority infants were statistically equivalent to, or lower than, the odds for the NHW reference category. However, the race/ethnicity x time period interaction term for infants of Mexican American women has an OR of 1.247, which represents an offset to the advantage suggested by the main effect (OR = .814). By contrast, the interaction term was not significant for

infants born to Mexican immigrants. In brief, the advantage in survivorship associated with Mexican immigrants, as compared to their U.S.-born counterparts, emerges in our results as well.

FOOTNOTES

[1] For example, from the 1980s into the 1990s, the white rate of preterm birth increased, while the black preterm rate declined (Demissie et al. 2001). However, preliminary data indicate that the low birth weight rate among blacks increased a bit between 2001 and 2002 (Hamilton, Martin, and Sutton 2003).

[2] 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 syndrome (Dorland's Illustrated Medical Dictionary, 27th Revision 1988).

[3] Some research indicates an increase over time in a black disadvantage in the crude relative risk (Malloy and Freeman 2000), and other research shows a decline, but not a reversal, in an initial black advantage.

[4] There is a large body of research that focuses on matters other than race/ethnic differentials, including evaluations of the benefits of surfactant therapy, the efficacy of natural (from animal lungs) as compared to synthetic surfactant, and the effectiveness of other interventions, e.g., administration of antenatal corticosteroids, as a means of enhancing fetal lung maturation (American Academy of Pediatrics 1999; Kresh and Clive 1998; Walfisch, Hallak, and Mazor 2001).

[5] An early study based on medical records from a South Carolina hospital did examine the effects of certain maternal characteristics on the incidence of RDS morbidity over the period 1982-1987 (Hulsey et al. 1993), but this useful study did not analyze infant risk of death, and the time period covered (1982-1987) antedated FDA approval of surfactant replacement by several years.

[6] However, maturational timing differences by race may not great enough to provide a full explanation (cf. Richardson and Torday 1994).

[7] To reiterate, while at times, we use the terms "white" and "black" for ease of exposition, our analysis is always of non-Hispanic whites (NHWs) and non-Hispanic blacks (NHBs).

[8] Recently, the causal significance of birth weight as a determinant of infant mortality has been questioned (Wilcox 2001). Regardless of the causal relationship, both birth weight and gestational age continue to be powerful predictors of infant survival and crucial risk factors in accounting for black-white inequality in infant mortality (Moss and Carver 1998; Hummer et al. 1999).

[9] Information regarding certain interventions (e.g., cesarean section, induction) can be found on birth certificates, but items eliciting information on many procedures, including surfactant therapy, are not included.

[10] A number of previous studies of RDS infant mortality have been limited to data on VLBW infants (weight < 1500 grams). Although this group is at high risk and certainly worthy of such attention, we compare results for normal weight and LBW infants because recent evidence shows that the greatest reduction in RDS mortality has occurred among infants weighing 2000-2499 grams and with gestational age of 33-36 weeks (Malloy and Freeman 2000)—i.e., in birth weight and gestational age categories that are near the normal cutpoints of 2500 grams and 37 weeks gestation.

[11] In addition, we repeated the analysis for singletons only, for neonatal mortality only, and also estimated models separately for blacks and whites. Results from these efforts do not change the general conclusions based on the analyses shown herein.

[12] The rates in our analysis will tend to be higher than those reported in research that restricts the data in certain ways—e.g., to singletons, to births occurring at 22 weeks gestation or greater, and/or to infants weighing 500 grams or more at birth, etc. Plurality is of interest for reasons delineated above. Further, as computed from the NCHS linked files employed in this analysis, in both the early and the mid-to-late 1990s, approximately 20% of infants born under 22 weeks gestation survived the first year of life. The same is true for 10%-13% of infants born at weights under 500 grams. Thus, the advantages of a more inclusive data set (i.e., the inclusion of infants most susceptible to RDS and

other morbid conditions) appear to outweigh the disadvantages (e.g., the possible inclusion of misclassified stillbirths or misreporting of birth weight or gestational age).

[13] In this context, the cause-specific estimates from the multivariate logit model are essentially identical to the observed rates.

Table 1. Infant Mortality Rates by Race: United States, 1989-1989 and 1995-1998

Table 1a. Infant Mortality Rates* (All Birthweights) by Race: United States, 1989-1990 and 1995-1998

	1989-1990		1995-1998		% Reduction in Rates between 1989-90 and 1995-98		Rate Ratio	
	Non-Hispanic White	Non-Hispanic Black	Non-Hispanic White	Non-Hispanic Black	Non-Hispanic White	Non-Hispanic Black	1989-1990	1995-1998
IMR per 1000	6.83	15.28	5.41	12.28	-20.8%	-19.6%	2.24	2.27
RDS per 1000	0.56	1.31	0.24	0.65	-57.1%	-50.4%	2.34	2.71
Other per 1000	6.27	13.96	5.17	11.63	-17.5%	-16.7%	2.23	2.25

* Total infant mortality (IMR)

SOURCE: NCHS Linked Birth/Infant Death Files. 1989-1990 and 1995-1998.

Table 1b. Infant Mortality Rates* among Low Birth Weight Infants† by Race: United States, 1989-1990 and 1995-1998

	1989-1990		1995-1998		% Reduction in Rates between 1989-90 and 1995-98		Rate Ratio	
	Non-Hispanic White	Non-Hispanic Black	Non-Hispanic White	Non-Hispanic Black	Non-Hispanic White	Non-Hispanic Black	1989-1990	1995-1998
IMR per 1000	69.03	81.00	50.97	69.27	-26.2%	-14.5%	1.17	1.36
RDS per 1000	9.98	9.92	3.66	4.95	-63.3%	-50.1%	0.99	1.35
Other per 1000	59.05	71.08	47.31	64.32	-19.9%	-9.5%	1.20	1.36

* Total infant mortality (IMR)

† Infants born weighing < 2500 grams.

SOURCE: NCHS Linked Birth/Infant Death Files. 1989-1990 and 1995-1998.

Table 2. Percent Distributions of Risk Factors for Infant Mortality by Race: United States, 1989-90 and 1995-98

	1989-1990		1995-1998	
RISK FACTORS	**NHW**	**NHB**	**NHW**	**NHB**
Place of Birth				
U.S.	95.9	93.6	95.3	91.2
Other	4.1	6.5	4.8	8.8
Maternal Age				
Under 18	5.8	16.4	5.9	15.8
18 or Older	94.2	83.6	94.1	84.2
Marital Status				
Unmarried	16.2	66.1	21.2	69.2
Married	83.8	34.0	78.8	30.8
Parity				
First Birth	42.6	37.8	42.1	39.1
Low	46.4	38.4	46.8	38.4
High	11.1	23.8	11.1	22.6
Sex				
Male	51.4	50.7	51.3	50.7
Female	48.7	49.3	48.7	49.3
Education				
<12 years	15.1	29.7	12.9	27.5
12 years	39.7	43.4	33.1	39.3
13+ years	45.2	27.0	54.0	33.3
Previous Loss				
Yes	24.6	26.4	26.3	28.9
No	75.4	73.6	73.7	71.1
Plurality				
Single	97.7	97.3	97.0	97.0
Plural	2.3	2.7	3.0	3.0
Medical Risks				
Yes	20.4	24.1	27.0	30.8
No	79.6	75.9	73.0	69.2
Labor/Delivery Comp.				
Yes	31.9	33.8	34.0	34.8
No	68.2	66.2	66.0	65.2
Prenatal Care				
Inadequate	11.1	30.0	7.8	20.4
Intermediate	15.2	14.9	13.7	13.6
Adequate	48.1	29.9	47.5	34.8
Adequate Plus	25.7	25.1	30.9	31.2
Smoking				
Yes	17.6	13.5	14.0	8.8
No	65.5	69.9	70.3	80.7
Missing	16.9	16.7	15.7	10.5
Weight Gain				
<15 lbs	4.8	9.2	6.6	11.4
15-40 lbs	64.5	56.7	65.0	59.6
> 40 lbs	13.1	11.2	16.6	14.9
Missing	17.6	22.9	11.8	14.1
Gestational Age				
Preterm (<37 weeks)	8.3	18.5	9.5	17.2
Term (≥ 37 weeks)	91.7	81.5	90.5	82.8
Birth Weight				
Low (<2500 grams)	5.4	12.9	6.2	12.7
Normal (≥2500 grams)	94.6	87.1	93.8	87.3
N	**4452941**	**1086318**	**9021404**	**2120234**

Table 3. Models of Risk Factor Effects on Infant Mortality: United States, 1989-1990

	Infant Mortality [Survive]									
	Model 1		Model 2		Model 3		Model 4		Model 5	
	RDS	Others	RDS	Others	RDS	Others	RDS	Others	RDS	Others
Ethnic Group [Non-Hispanic White]										
Non-Hispanic Black	2.361***	2.245***	1.654***	1.576***	1.525***	1.499***	1.273***	1.378***	0.796***	1.002
Place of Birth [U.S.]										
Foreign Born			0.861*	0.875***	0.889	0.882***	0.951	0.925***	0.984	0.966
Age [18 or Older]										
Under 18 years			1.312***	1.220***	1.217***	1.026	1.419***	1.188***	1.226***	1.125***
Marital Status [Married]										
Unwed			1.692***	1.700***	1.599***	1.517***	1.488***	1.412***	1.181***	1.243***
Parity [First Birth]										
Low			0.950	1.125***	0.951	1.101***	0.841***	1.060***	0.962	1.133***
High			1.453***	1.647***	1.399***	1.481***	0.973	1.267***	1.078	1.312***
Sex [Female]										
Male			1.551***	1.246***	1.529***	1.236***	1.561***	1.248***	1.649***	1.313***
Education [13+ Years]										
< 12 years					1.216***	1.418***	1.219***	1.335***	1.047	1.208***
12 years					1.159***	1.162***	1.189***	1.151***	1.100**	1.098***
Prenatal Care [Adequate]										
Inadequate					2.283***	1.766***	1.725***	1.502***	0.860***	1.103***
Intermediate					0.930	1.146***	0.939	1.149***	0.948	1.151***
Adequate Plus					4.805***	2.469***	2.979***	1.932***	0.806***	0.992
Previous Loss [None]										
Yes							1.277***	1.228***	1.112***	1.145***
Plurality [Singleton]										
Plural							7.160***	3.252***	1.509***	0.995
Medical Risks [None]										
Yes							1.955***	1.634***	1.177***	1.212***
Labor/Delivery Comp. [None]										
Yes							4.467***	2.255***	1.916***	1.513***
Smoking [Nonsmoker]										
Missing							0.694***	0.849***	0.783***	0.884***
Smoker							0.993	1.195***	0.752***	0.974**
Weight Gain [15-40 lbs.]										
Missing							2.675***	1.840***	1.885***	1.527***
<15 lbs.							6.210***	3.330***	2.975***	2.213***
40+ lbs							0.215***	0.578***	0.445***	0.835***
Gestational Age [Term]										
Preterm									24.503***	2.938***
Birth Weight [Normal]										
Low									55.274***	7.106***
Intercept	-7.48***	-5.07***	-7.88***	-5.42***	-8.69***	-5.88***	-9.92***	-6.52***	-12.33***	-6.50***
-2LL	562798.1***		557885.0***		549263.5***		516246.9***		451212.7***	

SOURCE: See Table 1.

Note: Brackets [ ] indicate reference groups.

*** $p \leq 0.01$. ** $p \leq 0.05$. * $p \leq 0.10$.

Table 4. Models of Risk Factor Effects on Infant Mortality: United States, 1995-1998

	Infant Mortality [Survive]									
	Model 1		Model 2		Model 3		Model 4		Model 5	
	RDS	Others	RDS	Others	RDS	Others	RDS	Others	RDS	Others
Ethnic Group [Non-Hispanic White]										
Non-Hispanic Black	2.758***	2.264***	2.208***	1.756***	2.061***	1.668***	1.672***	1.536***	1.109**	1.144***
Place of Birth [U.S.]										
Foreign Born			0.893	0.829***	0.940	0.846***	0.949	0.867***	1.000	0.908***
Age [18 or Older]										
Under 18 years			1.175***	1.254***	1.126*	1.037***	1.383***	1.233***	1.214***	1.170***
Marital Status [Married]										
Unwed			1.465***	1.470***	1.399***	1.298***	1.428***	1.252***	1.192***	1.125***
Parity [First Birth]										
Low			0.864***	1.039***	0.858***	1.010	0.701***	0.908***	0.897***	1.049***
High			1.112**	1.513***	1.076	1.340***	0.704***	1.062***	0.860***	1.176***
Sex [Female]										
Male			1.477***	1.226***	1.458***	1.216***	1.503***	1.236***	1.560***	1.291***
Education [13+ Years]										
< 12 years					1.189***	1.501***	1.226***	1.414***	1.070	1.275***
12 years					1.217***	1.259***	1.255***	1.235***	1.182***	1.180***
Prenatal Care [Adequate]										
Inadequate					2.223***	1.942***	1.607***	1.585***	0.753***	1.089***
Intermediate					1.016	1.140***	0.973	1.111***	0.989	1.116***
Adequate Plus					4.608***	2.660***	2.949***	2.082***	0.750***	0.968***
Previous Loss [None]										
Yes							1.312***	1.198***	1.179***	1.131***
Plurality [Singleton]										
Plural							7.412***	3.583***	1.418***	0.941***
Medical Risks[None]										
Yes							1.744***	1.559***	1.040	1.123***
Labor/Delivery Comp. [None]										
Yes							3.795***	2.089***	2.016***	1.481***
Smoking [Nonsmoker]										
Missing							0.548***	0.766***	0.679***	0.850***
Smoker							0.979	1.290***	0.754***	1.047***
Weight Gain [15-40 lbs.]										
Missing							2.897***	2.085***	1.998***	1.691***
<15 lbs.							5.664***	3.467***	3.041***	2.404***
40+ lbs							0.145***	0.547***	0.269***	0.786***
Gestational Age [Term]										
Preterm									19.729***	3.092***
Birth Weight [Normal]										
Low									44.652***	7.767***
Intercept	-8.35***	-5.26***	-8.62***	-5.55***	-9.48***	-6.09***	-10.57***	-6.70***	-12.82***	-6.77***
-2LL	915771.0***		910164.6***		893846.5***		83933.6***		732762.2***	

SOURCE: See Table 1.

Note: Brackets [ ] indicate reference groups.

*** $p \leq 0.01$. ** $p \leq 0.05$. * $p \leq 0.10$.

Table 5. Models of Infant Mortality among Low Birth Weight Infants by Time Period: United States, 1989-1990 and 1995-1998

Risk of Infant Death for Non-Hispanic Blacks [Non-Hispanic Whites]	Infant Mortality [Survive]							
	Model 1†		Model 2†		Model 3†		Model 4†	
	RDS	Other	RDS	Other	RDS	Other	RDS	Other
1989-1990								
Non-Hispanic Black	1.007	1.219***	0.910**	1.117***	0.916**	1.121***	0.818***	0.991
Intercept	-4.54***	-2.76***	-4.98***	-3.03***	-5.18***	-3.15***	-6.09***	-3.84***
-2LL	223068.8***		221871.3***		221494.3***		210771.7***	
1995-1998								
Non-Hispanic Black	1.377***	1.386***	1.321***	1.355***	1.326***	1.354***	1.128***	1.169***
Intercept	-5.56***	-3.00***	-5.89***	-3.20***	-6.08***	-3.35***	-6.95***	-4.01***
-2LL	385114.1***		383682.7***		383397.2***		363280.0***	

SOURCE: See Table 1.

Note: Brackets [ ] indicate reference groups.

† Model Covariates Are As Follows:

Model 1 is the bivariate relationship.

Model 2 controls place of birth, maternal age, maternal 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, plurality, medical risks, labor and delivery complications, smoking, and weight gain.

*** $p \leq 0.01$. ** $p \leq 0.05$. * $p \leq 0.10$.

Table 6. Models of Infant Mortality with Time Period as a Covariate: United States, 1989-1990 and 1995-1998

Table 6a. Selected Models of Infant Mortality for Births at All Weights: United States, 1989-1990 and 1995-1998

Odds Ratios for: Race, Time Period, and Race x Time Interaction	Infant Mortality [Survive]							
	Model 1†		Model 2†		Model 3†		Model 4†	
	RDS	Other	RDS	Other	RDS	Other	RDS	Other
Race [NHW]								
Non-Hispanic Black	2.559***	2.261***	2.541***	2.257***	0.937**	1.088***	0.800***	1.008
Year [1989-1990]								
1995-1998			0.445***	0.826***	0.389***	0.790***	0.345***	0.758***
Interaction								
NHB*1995-1998							1.386***	1.129***
Intercept	-7.97***	-5.19***	-7.51***	-5.07***	-12.12***	-6.52***	-12.06***	-6.50***
-2LL	1480731.5***		1478580.0***		1184456.6***		1184331.0***	

SOURCE: See Table 1.

Note: Brackets [ ] indicate reference groups.

† Model Covariates Are As Follows:

Model 1 is the bivariate relationship.

Model 2 adds time period.

Model 3 adds controls for all other risk factors, including gestational age and birth weight.

Model 4 includes all risk factors, plus the race x time period interaction.

*** $p \leq 0.01$. ** $p \leq 0.05$. * $p \leq 0.10$.

Table 6b. Selected Models of Infant Mortality for Low Weight Births : United States, 1989-1990 and 1995-1998

Odds Ratios for: Race, Time Period, and Race x Time Interaction	Infant Mortality [Survive]							
	Model 1†		Model 2†		Model 3†		Model 4†	
	RDS	Other	RDS	Other	RDS	Other	RDS	Other
Race [NHW]								
Non-Hispanic Black	1.215***	1.334***	1.169***	1.324***	0.958	1.102***	0.825***	1.019
Year [1989-1990]								
1995-1998			0.405***	0.828***	0.390***	0.818***	0.347***	0.779***
Interaction								
NHB*1995-1998							1.362***	1.129***
Intercept	-5.13***	-2.92***	-4.59***	-2.79***	-6.05***	-3.82***	-6.00***	-3.79***
-2LL	610188.7***		608279.9***		574342.8***		574256.4***	

SOURCE: See Table 1.

Note: Brackets [ ] indicate reference groups.

† Model Covariates Are As Follows:

Model 1 is the bivariate relationship.

Model 2 adds time period.

Model 3 adds controls for all other risk factors, except gestational age and birth weight.

Model 4 includes all risk factors, plus the race x time period interaction.

*** $p \leq 0.01$. ** $p \leq 0.05$. * $p \leq 0.10$.

Table A-1. Births All Weights: Reduction in Logit Approximations of Rates* Contrasted to 1989 by Race

Infant Mortality--Respiratory Distress Syndrome

	Bivariate Model					Full Model			
	NHW		NHB			NHW		NHB	
Year	Estimate	Z	Estimate	Z	Year	Estimate	Z	Estimate	Z
1989	ref.	ref.	ref.	ref.	1989	ref.	ref.	ref.	ref.
1990	0.130	6.51	0.222	3.37	1990	0.002	1.94	0.001	0.70
1995	0.301	16.72	0.663	11.00	1995	0.004	5.56	0.003	2.70
1996	0.320	17.97	0.652	10.76	1996	0.004	5.99	0.003	2.64
1997	0.329	18.62	0.695	11.62	1997	0.004	6.20	0.004	2.88
1998	0.321	18.02	0.760	12.98	1998	0.004	6.01	0.004	3.19

Infant Mortality--All Other Causes

	Bivariate Model					Full Model			
	NHW		NHB			NHW		NHB	
Year	Estimate	Z	Estimate	Z	Year	Estimate	Z	Estimate	Z
1989	ref.	ref.	ref.	ref.	1989	ref.	ref.	ref.	ref.
1990	0.355	4.84	0.654	2.90	1990	0.073	2.69	0.068	0.77
1995	1.155	16.23	2.361	10.64	1995	0.295	11.58	0.335	3.94
1996	1.355	19.16	2.306	10.34	1996	0.348	13.85	0.314	3.67
1997	1.347	19.02	2.950	13.40	1997	0.356	14.22	0.426	5.61
1998	1.486	21.09	2.789	12.67	1998	0.384	15.49	0.383	4.53

SOURCE: See Table 1.

* Approximation of rates per 1,000.

Significance Levels: 1.96 for $p \leq .05$; 2.58 for $p \leq .01$. All tests are two-tailed.

Table A-2. Low Weight Births: Reduction in Logit Approximations of Rates* Contrasted to 1989 by Race

Infant Mortality--Respiratory Distress Syndrome

	Bivariate Model					Full Model			
	NHW		NHB			NHW		NHB	
Year	Estimate	Z	Estimate	Z	Year	Estimate	Z	Estimate	Z
1989	ref.	ref.	ref.	ref.	1989	ref.	ref.	ref.	ref.
1990	2.920	6.71	1.924	3.19	1990	0.520	2.73	0.452	1.22
1995	7.228	17.32	5.814	10.51	1995	1.341	8.23	1.515	4.78
1996	7.692	18.68	5.667	10.16	1996	1.433	8.92	1.492	4.69
1997	7.884	19.25	6.085	11.06	1997	1.470	9.19	1.606	5.13
1998	7.809	19.02	6.712	12.48	1998	1.449	9.03	1.750	5.69

Infant Mortality--All Other Causes

	Bivariate Model					Full Model			
	NHW		NHB			NHW		NHB	
Year	Estimate	Z	Estimate	Z	Year	Estimate	Z	Estimate	Z
1989	ref.	ref.	ref.	ref.	1989	ref.	ref.	ref.	ref.
1990	3.144	0.07	2.314	1.38	1990	0.779	1.17	0.399	0.31
1995	13.018	11.05	7.025	4.13	1995	4.125	6.72	2.912	2.31
1996	14.957	12.78	6.873	4.01	1996	4.913	8.13	3.026	2.40
1997	15.071	12.88	9.492	5.60	1997	5.010	8.30	4.124	3.33
1998	14.864	12.69	7.121	4.17	1998	4.864	8.03	3.008	2.39

SOURCE: See Table 1.

* Approximation of rates per 1,000.

Significance Levels: 1.96 for $p \leq .05$; 2.58 for $p \leq .01$. All tests are two-tailed.

Table A-3. Births All Weights: Black-White Differences in Reduction of Approximations of Rates*

	Infant Mortality Respiratory Distress Syndrome					Infant Mortality All Other Causes			
	Bivariate Model		Full Model			Bivariate Model		Full Model	
	Difference in Estimate		Difference in Estimate			Difference in Estimate		Difference in Estimate	
Year	NHB - NHW	Z	NHB - NHW	Z	Year	NHB - NHW	Z	NHB - NHW	Z
1989	ref.	ref.	ref.	ref.	1989	ref.	ref.	ref.	ref.
1990	0.092	1.34	-0.000	0.29	1990	0.299	1.26	-0.006	0.06
1995	0.362	5.76	-0.000	0.16	1995	1.205	5.18	0.040	0.45
1996	0.333	5.27	-0.001	0.36	1996	0.951	4.06	-0.034	0.38
1997	0.366	5.87	-0.000	0.27	1997	1.603	6.93	0.070	0.80
1998	0.439	7.18	0.000	0.03	1998	1.303	5.64	-0.002	0.02

SOURCE: See Table 1.

* Approximation of rates per 1,000.

Significance Levels: 1.96 for $p \leq .05$; 2.58 for $p \leq .01$. All tests are two-tailed.

Table A-4. Low Weight Births: Black-White Differences in Reduction of Approximations of Rates*

	Infant Mortality Respiratory Distress Syndrome					Infant Mortality All Other Causes			
	Bivariate Model		Full Model			Bivariate Model		Full Model	
	Difference in Estimate		Difference in Estimate			Difference in Estimate		Difference in Estimate	
Year	NHB - NHW	Z	NHB - NHW	Z	Year	NHB - NHW	Z	NHB - NHW	Z
1989	ref.	ref.	ref.	ref.	1989	ref.	ref.	ref.	ref.
1990	-0.996	1.30	-0.068	0.16	1990	-0.830	0.02	-0.379	0.26
1995	-1.413	2.04	0.174	0.49	1995	-5.993	2.90	-1.213	0.87
1996	-2.025	2.92	0.059	0.17	1996	-8.085	3.90	-1.887	1.35
1997	-1.799	2.62	0.136	0.39	1997	-5.578	2.71	-0.886	0.64
1998	-1.097	1.62	0.301	0.87	1998	-7.743	3.74	-1.856	1.33

SOURCE: See Table 1.

* Approximation of rates per 1,000.

Significance Levels: 1.96 for $p \leq .05$; 2.58 for $p \leq .01$. All tests are two-tailed.

Table 7. Infant Mortality Rates* by Race: United States, 1989-1990 and 1995-1998

Table 7a. Infant Mortality Rates* (All Birthweights) by Race: United States, 1989-1990 and 1995-1998

	1989-1990				1995-1998			
	NHW	**NHB**	**MXG-U.S.**	**MXG-Imm.**	**NHW**	**NHB**	**MXG-U.S.**	**MXG-Imm.**
IMR per 1000	6.83	15.28	7.30	6.09	5.41	12.28	6.01	4.65
RDS rate per 1000	0.56	1.31	0.59	0.47	0.24	0.65	0.28	0.17
Other Cause Rate per 1000	6.27	13.96	6.71	5.62	5.17	11.63	5.74	4.48

* Total Infant Mortality (IMR).

SOURCE: See Table 1.

Table 7b. Ratios of Infant Mortality Rates to Non-Hispanic Whites: United States, 1989-1990 and 1995-1998

	1989-1990				1995-1998			
	NHW	**NHB**	**MXG-U.S.**	**MXG-Imm.**	**NHW**	**NHB**	**MXG-U.S.**	**MXG-Imm.**
IMR per 1000	1	2.24	1.07	0.89	1	2.27	1.11	0.86
RDS rate per 1000	1	2.34	1.05	0.84	1	2.71	1.17	0.71
Other Cause Rate per 1000	1	2.23	1.07	0.90	1	2.25	1.11	0.87

SOURCE: See Table 1.

Table 8. Models of Infant Mortality with Time Period as a Covariate: United States, 1989-1990 and 1995-1998

Table 8a. Selected Models of Infant Mortality for Births at All Weights

Race, Time, and Race x Time Interaction	Infant Mortality [Survive]									
	Model 1†		Model 2†		Model 3†		Model 4†		Model 5†	
	RDS	Other	RDS	Other	RDS	Other	RDS	Other	RDS	Other
Race [NHW]										
NHB	2.559***	2.261***	2.541***	2.257***	2.361***	2.245***	0.931***	1.083***	0.796***	1.004
Mexican-U.S.	1.032	1.080***	1.114*	1.098***	1.054	1.070***	0.917	0.872***	0.814**	0.809***
Mexican-Imm.	0.724***	0.861***	0.773***	0.873***	0.839**	0.895***	0.817***	0.748***	0.892	0.746***
Year [1989-1990]										
1995-1998			0.442***	0.826***	0.420***	0.824***	0.386***	0.788***	0.345***	0.755***
Interaction										
NHB*95-98					1.168***	1.008			1.383***	1.128***
MX-U.S.*95-98					1.111	1.037			1.247*	1.116***
MX-Imm.*95-98					0.863	0.966			0.862	1.007
Intercept	-7.97***	-5.19***	-7.50***	-5.07***	-7.48***	-5.06***	-12.09***	-6.53***	-12.04***	-6.51***
-2LL	1651668.8***		1649287.9***		1649269.8***		1323071.3***		1322933.5***	

SOURCE: See Table 1.

Note: Brackets [ ] indicate reference groups.

† Model Covariates Are As Follows:

Model 1 is the bivariate relationship.

Model 2 adds time period.

Model 3 adds the race x time period interaction.

Model 4 adds controls for all other risk factors, including gestational age and birth weight.

Model 5 includes all risk factors, plus the race x time period interaction.

*** $p \leq$ 0.01. ** $p \leq$ 0.05. * $p \leq$ 0.10.

Table 8. Models of Infant Mortality with Time Period as a Covariate: United States, 1989-1990 and 1995-1998

Table 8b. Selected Models of Infant Mortality for Low Weight Births

Race, Time, and Race x Time Interaction	Infant Mortality [Survive]									
	Model 1†		Model 2†		Model 3†		Model 4†		Model 5†	
	RDS	Other	RDS	Other	RDS	Other	RDS	Other	RDS	Other
Race [NHW]										
NHB	1.215***	1.334***	1.168***	1.324***	1.007	1.219***	0.951*	1.100***	0.800***	1.017
Mexican-U.S.	0.929	0.998	0.994	1.011	0.903	0.925**	0.898*	0.891***	0.803**	0.804***
Mexican-Imm.	0.829***	1.029*	0.883**	1.041**	0.952	1.026	0.838***	0.917***	0.945	0.935**
Year [1989-1990]										
1995-1998			0.402***	0.829***	0.360***	0.786***	0.385***	0.818***	0.347***	0.779***
Interaction										
NHB*95-98					1.368***	1.137***			1.358***	1.129***
MX-U.S.*95-98					1.202	1.134***			1.233*	1.157***
MX-Imm.*95-98					0.877	1.026			0.811*	0.978
Intercept	-5.13***	-2.92***	-4.59***	-2.79***	-4.54***	-2.76***	-6.06***	-3.84***	-6.01***	-3.81***
-2LL	673826.9***		671729.5***		671623.5***		635477.8***		635373.3***	

SOURCE: See Table 1.

Note: Brackets [ ] indicate reference groups.

† Model Covariates Are As Follows:

Model 1 is the bivariate relationship.

Model 2 adds time period.

Model 3 adds the race x time period interaction.

Model 4 adds controls for all other risk factors, except gestational age and birth weight.

Model 5 includes all risk factors, plus the race x time period interaction.

*** $p \leq 0.01$. ** $p \leq 0.05$. * $p \leq 0.10$.

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