ADVANCES IN PERINATAL CARE AND INCREASING RACE/ETHNIC DISPARITY IN INFANT MORTALITY *

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Abstract

Although substantial declines in infant mortality rates have occurred across race/ethnic groups, there has been a marked increase in relative disparity in risk of infant death. The objective is to gain insight into the reasons for growing race/ethnic inequality based on cause-of-death specific data from linked birth/infant death cohort files for 1989-1991 and 1995-1998. We are particularly interested in widening disparities which followed in the wake of advances in perinatal care. We find that the relative gap between non-Hispanic white (NHW) infants and infants born to both non-Hispanic black (NHB) and Mexican American mothers increased when innovations in health care occur in a continuing context of social inequality. Models of absolute change demonstrate that, among low weight births, absolute declines in mortality from some leading causes were greater for NHW infants than for infants born to NHB and Mexican American women. However, these findings do not hold for infants born to Mexican immigrants.

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INTRODUCTION

In recent years, tremendous advances in perinatal health care and technology have resulted in resuscitation of infants born at ever lower weights and ever shorter gestations (Hollander 1995; Sowards 1997), and presumably a more than off-setting capacity to preserve the life of these high-risk newborns—given the consistent temporal decline in infant mortality rates. (Guyer et al. 1998). [1] In certain quarters, the general reduction in infant death rates may have created the impression that race/ethnic differentials in survival chances would become an issue of declining substantive interest. Just the opposite has proven to be the case.

As is quite clear from the Healthy People 2010 report (U.S. Department of Health and Human Services [U.S. DHHS] 2000), despite the emergence of new and enhanced preventative and curative perinatal health care interventions, large relative differentials in infant mortality between the non-Hispanic white majority and certain minority populations have persisted and are of growing concern to scholars and policy makers alike (Frisbie et al. 2004, in press; Gortmaker and Wise 1997; Wise 2003). Even more disturbing is the finding that, while substantial absolute declines in infant mortality have occurred across virtually all race/ethnic groups, the relative disparity between blacks and whites has not only persisted, but has actually increased. 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. By the early 1990s, both ratios had risen to approximately 2.3, where they plateaued until 1997 (Guyer et al. 1998). Since then, however, widening of the gap has resumed. For example, between 1998 and 1999, there was a decline of 3.8% in the death rate for white infants, as compared

[1] Just reported by Kochanek and Martin (2004) is evidence that a nationwide increase in the IMR occurred between 2001 and 2002—the first such increase since 1957-58. This increase occurred too recently to allow determination of whether this is a short-lived inflection or a longer-term alteration in the secular trend.

to a decline of only 0.3% among black infants (Hoyert et al. 2001: 5), and by the year 2000, the black/white rate ratio stood at 2.4-2.5 (Hoyert et al. 2001; Mathews et al. 2002; Minino et al. 2002). Now there is evidence that, among high risk (i.e., low birth weight) infants, the absolute gap in black-white infant mortality has widened, at least in the case of one leading specific cause of death (Frisbie et al. 2004, in press).

A considerably expanded research agenda which focuses on race/ethnic disparities by specific causes of infant death is needed. As put by Mathews et al., "(a)n examination of cause-specific differences in infant mortality rates between race and Hispanic origin groups can help the researcher to understand overall differences between these groups" (2002: 7; emphasis added). There, of course, continues to be considerable interest in Hispanic (especially Mexican origin) infant mortality, but few studies have attempted to explain changing differentials by specific cause of death across multiple race/ethnic groups (for an exception, see Muhuri et al. 2004). Further, over the past decade, the Mexican Origin/non-Hispanic white infant mortality rate ratio remained little changed, [2] but, as we will demonstrate, this relative stability resulted from the higher rates for infants of U.S.-born Mexican women being offset by lower rates among infants born to Mexican immigrants.

Objectives

The general substantive issue addressed by the present research can be posed as follows: "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). In this paper, we document and attempt to explain the variation observed nationwide in infant mortality risk from three leading causes of death among the non-Hispanic white (NHW), non-Hispanic black (NHB), and the Mexican origin populations over the period 1989-1998. The time frame is important inasmuch as it has been

[2] Throughout, we use the term "Mexican origin" to refer to both U.S.-born Mexican Americans and immigrants from Mexico.

concluded that "the past two decades have witnessed the most profound alterations ever recorded in the structure of infant mortality patterns in the United States" (Gortmaker and Wise 1997: 152). [3]

CONCEPTUAL MODEL

General Model

How does it happen that greater race/ethnic disparities in health follow in the wake of improvements in health care and technology? A prominent theory of health inequalities among race/ethnic groups is based on the premise that 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; see also Link and Phelan 1995, 1996). In brief, the Link and Phelan theory of fundamental social causes proposes that "as health-related situations change, those with the most resources are best able to avoid diseases and their consequences" (Link and Phelan 1996: 472).

"Middle-Range" Theory

Consistent with this general model is recent literature on the widening relative gap in black/white infant mortality. Gortmaker and Wise (1997) 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. This does not imply that a high-risk black infant will be denied therapeutic intervention or be accorded lower quality of care. Rather, we understand the Gortmaker and Wise argument to be similar to that advanced by Link and Phelan (1995, 2002); viz., socially disadvantaged groups are less likely to have the information, the social networks, and/or the socioeconomic wherewithal to acquire knowledge of, and/or acquire access to, innovations in health care.

[3] The analysis begins with 1989 because that is the first year in which identification of Hispanic groups was sufficiently comprehensive to support the analysis.

Our aim is to examine the extent to which disparity in the risk of infant mortality increased following the introduction of advances in perinatal care and technology and to estimate the direction and magnitude of the effects of social, demographic, behavioral, and biomedical risk factors that may influence changing race/ethnic differentials. Specifically, we will examine the change in risk from three leading causes of infant mortality: Sudden Infant Death Syndrome (SIDS), Respiratory Distress Syndrome (RDS), and congenital anomalies for the period 1989-1998. These three conditions were among the five leading causes of infant mortality over the 1990's decade, and each allows somewhat different, but interrelated and complementary, insights regarding race/ethnic infant mortality differentials. [4]

Comparing the risk of infant death for the Mexican origin population with the risk for NHWs and NHBs provides analytical leverage with respect to the degree to which differential access to health care interventions is responsible for race/ethnic disparities. As has been consistently documented, the Mexican origin population, even compared to blacks, is characterized by lower average levels of education, greater proportions of women lacking adequate prenatal care, and a higher percentage without insurance of any kind to pay for delivery (Frisbie et al. 1997; Moss and Carver 1998)—all risk factors that impede access to care. Further, Mexican immigrant women (indeed, immigrants in general), are less apt to have health insurance and more likely to have restricted access to the formal health care system than are their U.S.-born counterparts (Frisbie et al. 2001; LeClere et al. 1994; Thamer et al. 1997). Thus, if differential access to health care advances is a root cause of race/ethnic variation in infant mortality, over time, the relative risk of adverse

[4] The other two leading causes of infant mortality are disorders relating to short gestation and unspecified low birth weight (SG/LBW) and maternal complications (e.g., incompetent cervix, premature rupture of membranes, ectopic pregnancies, and multiple pregnancy. The IMR for SG/LBW has actually increased in recent years if one considers all births, rather than just singletons, and the same is true for infant mortality related to maternal complications (Muhuri et al. 2004). Clearly, additional research on these causes is sorely needed. They are not included here, however, because of our focus on causes for which notable declines in infant mortality, associated with innovations in health care, occurred.

pregnancy outcomes among Mexican origin infants, especially those born to immigrant mothers, should have increased.

However, a large body of research reports that the risk of Mexican origin infant mortality is very similar to, (and sometimes lower than) that observed among non-Hispanic whites, and mortality rates have been found to be lower among infants born to Mexican immigrant women than are the rates for either NHW infants or for infants born to U.S.-born Mexican American mothers (Frisbie et al. 1998; Hummer et al. 1999; Scribner 1996; Singh and Yu 1996). Hence, we do not expect that risk of Mexican origin infant mortality will have become notably (or even modestly) higher than the NHW risk over the 1990s, and support for the theory does not depend on such results. The theory would be supported, however, by the finding that the relative prevalence of positive pregnancy outcomes among Mexican origin infants eroded following the introduction of innovations in perinatal care.

To summarize, we first will determine whether, and the degree to which race/ethnic variation in three leading cause of infant death changed in the wake of major innovations in perinatal care**.** Second, we will estimate the degree to which social inequities, which can be expected to limit access to advances in perinatal care, or other alternative explanations, can account for variation over time in the causes of death. Third, we want to model the risk of three leading causes of infant death for high risk infants. The third aim is important for several reasons. Inasmuch as medical technology is credited with an increasing ability to resuscitate high risk (low birth weight) many of whom would have been stillborn in previous times, it is of crucial importance to determine the extent to which perinatal care and technology has been able to improve the viability of these high-risk births. The matter is further complicated by the fact that, from the 1980s into the 1990s, rates of preterm birth and low weight births increased among whites and many other race/ethnic groups, while rates of these adverse birth outcomes declined slightly among blacks (Demissie et al. 2001; Frisbie and Song

2003). One interpretation of the upward inflection in the rates of low weight and preterm births among white women is that fertility enhancement procedures have resulted in an increased rate of multiple births to white women—a phenomenon not observed among black women (Demissie et al 2001; see also Blondel et al. 2002). Plural births will of course be, on average, earlier and lighter than singletons.

Finally, although the relative disparity in infant mortality between race/ethnic minorities and the NHW majority has increased, a number of authors have taken some comfort from data showing that the absolute gap has narrowed. The latter finding characterizes overall changes in rates, and while encouraging, it is also unsurprising inasmuch as the absolute rates for a number of minority groups, especially blacks, are high and "have much more room to fall." However, recent research suggests there may be reason to qualify optimism concerning absolute rates. Frisbie et al. (2004, in press) report the disturbing finding that among high-risk (i.e., low weight) births at least for RDS, the greatest absolute improvement in survival for RDS-related deaths occurred among whites, not blacks. Hence, another of our aims is to determine whether such an unexpected finding applies in regard to SIDS and congenital anomalies.

TRENDS IN SPECIFIC CAUSES OF INFANT DEATH

The highest rate of infant mortality in 1989-91 was for congenital anomalies, followed by SIDS, SG/LBW, and RDS in descending order. In 1995-98, the rank order was the same, except that SIDS and SG/LBW switched places in the ranking. We begin the discussion of changes in cause-specific rates with RDS because of the wealth of clinical evidence linking the fall in rates from that condition to a particular techno-medical innovation, and because of the relative lack of population based research on race/ethnic differentials in the decline in RDS for the nation as a whole.

RDS*:* There is convincing evidence that pulmonary surfactant replacement therapy has been extremely effective in reducing RDS mortality (Ferrara et al. 1994; Halliday 1997; Hamvas et al.

1996; Malloy and Freeman 2000; Ranganathan et al. 2000). RDS results from a deficiency of naturally occurring surfactant in the lungs of the fetus such that the functioning of the alveoli may be compromised and gas exchange may fail. 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). The availability of data on variation in risk of death from RDS in the periods before and after the approval of surfactant therapy by the U.S. Food and Drug Administration (FDA) for general use in August 1990 offers an opportunity to gain insight into how variation in the impact of advances in perinatal health care can be produced or mediated by social factors. In addition to the clinical research, a few descriptive studies, found mainly in the public health literature, have taken advantage of this natural experiment by observing changes in the black-white RDS differentials in the "pre-surfactant" and "post-surfactant" periods (Frisbie et al. 2004, in press; Malloy and Freeman 2000; Ranganathan et al. 2000).

Following approval of surfactant replacement, RDS mortality dropped substantially among both blacks and whites. If one considers the IMR for all live births, absolute declines were greater for blacks than for whites, but in relative terms, the black/white gap increased substantially as indicated by rate ratios (Halliday 1997; Hamvas et al. 1996; Malloy and Freeman 2000; Ranganathan et al. 2000). Thus, the decrease in infant death associated with RDS appears to have contributed both to the overall decline in infant mortality in the 1990s and to the widening black-white RDS relative risk differential (Ranganathan et al. 2000). At least one study, based on data from hospitals in the St. Louis area, indicates a reversal from a black RDS survival advantage to a white survival advantage among very low birth weight (500-1500 grams) infants after introduction of surfactant therapy. More recent research indicates that a reversal also occurred nationwide and

that, among low weight births (< 2500 grams), the absolute improvement for white infants was greater than that for black infants (Frisbie et al. 2004, in press).

SIDS: The prominence of SIDS as a leading cause of infant death in general, and the fact that it was the number one cause of postneonatal mortality in both the 1980s and 1990s (Black et al. 1986; Pollack and Frohna 2001, 2002) represent part of the rationale for including this condition in this analysis. Importantly for present purposes, SIDS, like RDS, is a condition for which a specific advance in perinatal health care apparently had a large and beneficial impact on infant survivorship. Following the recommendation by the American Academy of Pediatrics (AAP) in 1992 that infants not be put to sleep in the prone position and the educational campaign promoting "back to sleep" that was mounted nationwide in 1994 (Gibson et al. 2000; Pollack and Frohna 2001, 2002; Willinger et al. 1998), the SIDS rate dropped by more than one-third among singleton births in the U.S. between 1989 and 1997 (Pollack and Frohna 2001). The utility of a "before and after" analysis is strongly supported by the fact that prior to 1992, the SIDS mortality rate changed little, even though infant death rates from many other causes were on the decline (Willinger et al. 1998).

SIDS is a condition that is included in the International Classification of Diseases (ICD) under the rubric "Symptoms, Signs, and Ill-Defined Conditions," with the specific definition being "the sudden and unexpected death of an apparently healthy baby" (Davies 1994: 139). The etiology of SIDS remains to be firmly established, and "the mechanism by which the prone sleeping position should confer an increased risk of death to a young infant remains unclear" (Jeffery et al. 1999: 263). Research begun on animals and extended to human infants suggests that "airway protection is compromised in the prone sleeping position" (and improved in the supine position) because when infants sleep on their stomachs, "the swallowing rate is reduced significantly" (with) "no compensatory increase in arousal" (Jeffrey et al. 1999: 263). Ambient smoke appears to contribute to

risk of death from SIDS because the normal stimulation to ventilation that occurs through laryngeal chemoreflex is depressed by nicotine (Jeffery et al. 1999).

Descriptive studies indicate that the disparity between black and white risk of death from SIDS (as indicated by the ratio of black-to-white cause-specific IMRs), widened substantially following the "back-to-sleep" initiative (Moon et al. 2002; Willinger et al.1998). Indeed, "the SIDS rate has declined twice as fast among Caucasians than among African Americans (AA) from 1989 to 1995" (Gibson et al. 2000: 286). Inclusion of other minority groups is important to this research because descriptions of rate changes indicate substantial variability by race/ethnicity. For example, the incidence of SIDS mortality per 1000 live births was lower among Mexican Americans than among non-Hispanic whites both before and after the back to sleep campaign (Black et al. 1986; Pollack and Frohna 2001). Relative survival gains were smallest for African Americans (Pollack and Frohna 2001). One early study, prior to the "back to sleep" initiative, based on California vital records found the incidence of SIDS to be lowest among infants of mothers born in Mexico, followed by infants born to Mexican American, white, and black mothers in ascending order (Grether and Schulman 1989: 563).

Congenital Anomalies

From 1980 to the present, congenital anomalies was the leading cause of infant death in the United States (U.S. Census Bureau 2001: 77). Another major reason for including this cause of death is that it represents one of the few conditions for which the white infant mortality rate exceeded the non-white rate in the not-too-distant past. In the early 1970s, the white IMR from congenital malformations stood at 3.1 per 1000 live births as compared to a nonwhite rate of 2.6 per 1000 (Lee et al. 2001). While the vast majority of all nonwhites are African Americans, the presence of other groups (notably Asians) in the nonwhite category no doubt contributed to the nonwhite survival advantage. From 1970 to 1980, the white rate of death from congenital anomalies declined more

rapidly than that for nonwhites. Still, even with this relatively greater improvement for whites, when data became available that allowed a specific comparison of blacks with whites in the early 1980s, white and black rates were quite similar (Lee et al. 2001). But, in the mid-1980s, the infant death rate for whites from this condition continued to decrease faster than did the rate among blacks.

Importantly, these trends support the conclusion that genetic differences are not a cause of black- white disparities in infant mortality. Thirty years ago, the white death rate from congenital anomalies was higher than the nonwhite rate (Lee et al. 2001), and 20 years ago, the rates for blacks and whites were quite similar. Since that time, a black disadvantage has emerged, but genetic changes of consequence do not occur over a few decades. It is simply not plausible that in the past two or three decades the formerly superior (or at least similar, if the earlier need to rely on nonwhite as opposed to black rates casts some doubt) survivorship of blacks from congenital malformations was erased or reversed due to changes in genetic makeup. If a biological (i.e., genetic) explanation is not applicable, what can explain the emergence of a white survival advantage? A likely explanation is that social inequity has led to race differentials in benefits from advances in health care.

In recent years, a number of remarkable medical innovations have occurred that have played a role in the reduction in infant mortality from congenital defects, including preventative measures (e.g., consumption of folic acid prior to conception and in the early stages of pregnancy apparently reduces risk of neural tube defects), the growing ability to detect congenital malformations in the course of prenatal care, antenatal surgical procedures to correct malformations detected in the fetus, selective termination of pregnancies when lethal anomalies are detected, and interventions designed to preserve the life of infants born with congenital malformations (Lee et al. 2001). Access to these interventions appears to be much more limited for "low-income women and women of color" (Nsiah-Jefferson 1993:308). Among the limitations for both NHB and Mexican origin access to preventive and curative interventions are a lower percentage who receive adequate prenatal care and

screening during which congenital defects might be discovered (and, at least in recent years, ameliorated or remedied), lower levels of education and knowledge of the health care system, and insufficient economic resources and health insurance. Each of these can also be seen as barriers to genetic counseling that can help reduce the incidence of conception of infants with congenital malformations (Nsiah-Jefferson 1993). Unlike the case with SIDS and RDS, perinatal care innovations aimed at reducing infant morality from congenital malformations appear to have been more incremental in nature. However, note that while surfactant replacement and the back-to-sleep program have been credited with a large proportion of the reduction in infant death from RDS and SIDS, respectively, other perinatal care advances have had beneficial effects as well. [5]

DIFFERENTIAL MECHANISMS

Mechanisms Associated with Fundamental Social Causes Theory

The core conceptual model underlying this analysis proposes that race/ethnic differentials in access to interventions—interventions made possible by recent notable advances in health care technology—will eventuate in a higher degree of relative minority disadvantage vis a vis the NHW majority. However, the precise nature of the mechanisms through which social inequality can lead to a widening race/ethnic gap in infant mortality may differ somewhat according to the nature of the health care intervention and by specific cause of death. For example, the beneficial effects of surfactant therapy on RDS can be expected to vary with economic resources, having adequate health insurance, and the level and quality of diagnostics and medical care available at parturition. This is because surfactant replacement is a medical/technological advance that is administered in a clinical setting, with the likelihood of successful intervention being greatest in tertiary care hospitals with a top-flight neonatal intensive care unit (NICU). As just discussed above, very similar mechanisms

[5] For example, antenatal corticosteroids are employed to improve fetal lung maturation and thereby reduce risk of death from RDS and other respiratory ailments (Morales et al. 1989).

seem likely to play a significant role in the smaller benefits obtained by minority groups in the reduction of infant mortality attributable to congenital anomalies.

By contrast, the risk of SIDS death has been dramatically reduced by the simple procedure of putting infants to sleep on their back. Hence, any growth in disparity between minority and majority infants from this condition is much less likely to be due to lack of social and economic resources, and much more apt to be a result of "outreach failures." That is, it is certainly plausible that the "back-to-sleep" campaign of the early 1990s did not adequately penetrate segregated inner-city neighborhoods in which the population is more isolated, less educated on average, and therefore generally less likely to cognizant of recommendations for achieving improved health. Lack of adequate prenatal and postnatal ("well baby") care would exacerbate the problem in that advice and counseling regarding healthy behaviors is less likely to be received.

An Alternative Mechanism

Public health studies comparing black and white changes in infant mortality due to RDS have suggested an alternative mechanism through which innovations in perinatal care may lead to a widening majority-minority gap in infant mortality. Specifically with respect to RDS, it has been concluded that a substantial portion of the increase in disparity between blacks and whites in the risk of infant mortality is due, not to differential access to intervention, but rather to differential need for intervention. The putative link between surfactant therapy and the increasing black RDS infant mortality disadvantage is that "Fetal pulmonary surfactant matures more slowly in white than in black fetuses, and therefore RDS is more prevalent among whites than among blacks" (Hamvas 1996: 1635). 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). This interpretation is consistent with earlier research which concluded that, in

general, black infants mature at shorter gestations and lower birth weights than white infants (Kline et al. 1989; Wilcox and Russell 1986, 1990).

In a later commentary, Hamvas (2000) maintains that the widening black-white gap "may result from biologic differences," especially as these are related to "genetic and developmental mechanisms that determine the response to environmental influences" (2000: 428). Hamvas also explicitly acknowledges that the widening relative disparity between black and white risk of infant mortality from RDS could be due to other mechanisms, including differences in access to intervention.

At this juncture, it will be useful to consider whether the "differential need for access to intervention" hypothesis can be applied to SIDS and congenital anomalies—the other two leading causes on which we focus. Although the literature may not as directly link racial variation in speed of fetal maturation to the latter two causes, it is nonetheless the case that infant mortality rates from SIDS and congenital anomalies (and most other causes) are much higher among infants born at low weights and short gestations. Our calculations for the period 1995-98 show that, depending on race/ethnic group, LBW infants were from 7 to 18 times more likely to die of RDS, as compared to all infant births. For SIDS and congenital anomalies, the excess risk among low weight births ranged from 2 to 3 times higher for SIDS and from 5 to 10 times higher for congenital anomalies. Had low weight births been compared to normal weight births, the excess would be much greater. It seems logical to expect that infants born early and light will be less robust in their capacity to survive morbid conditions of any nature or origin. That fetal development of blacks occurs more rapidly than is the case for white infants has long been a tenet in public health literature used to explain well-documented fact of a higher rate of survival among LBW black infants that among their LBW white counterparts (Hamvas et al. 1996; Wilcox and Russell1990). Thus, the differential need for intervention hypothesis might be plausibly entertained for conditions other than RDS.

Conversely, a more recent analysis that examined changes in both relative and absolute racial disparity in RDS-related infant mortality for the period 1989-90 to 1995-98 concluded that "…taking into account that blacks are far more disadvantaged in terms of their risk profile than are whites, and that controls for social factors substantially diminish the race disparity in risk, we believe our results support the view that, whatever differences in need for intervention exist, social inequality remains a fundamental cause of disparities in health care, morbidity, and mortality" (Frisbie et al. 2004, in press). Finally, there is no indication of any notable differences between the Mexican origin and NHW populations in the rate of fetal maturation. Thus, the inclusion of the Mexican origin population (by nativity) in the current research should allow additional insight into whether the "social model" that lies at the core our research or the "medical" model has the greater explanatory potential.

RISK FACTORS

Basic demographic covariates for this analysis include maternal age, marital status, and nativity, along with parity and sex of infant. We control for sex because 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). Infant mortality risk is higher for infants born to teenagers and unmarried mothers (Cramer 1987; Hummer et al. 1999; Moss and Carver 1998). 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). 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 2005, forthcoming), though some authors ascribe the immigrant advantage to cultural differences (Cobas et al. 1996; Scribner 1996).

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

Other somewhat more direct indicators of access to health care available in our data set are maternal education and prenatal care. Infant mortality risk decreases as maternal education rises. In addition to being an indicator of SES, maternal education may reflect knowledge of available medical services and of strategies for circumventing obstacles to access (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). Adequate PNC is one mechanism through which the woman and medical personnel can become aware of existing maternal morbidities and/or problems in fetal development well before the onset of labor. 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 childbirth.

Maternal health endowments have a powerful impact on pregnancy outcomes (Eberstein et al. 1990; Frisbie et al. 1998; Kallan 1993; Moss and Carver 1998). Hence, 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.

It has been established that smoking, particularly through its negative effect on birth weight, heightens the risk of infant mortality (Chomitz et al. 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 have long been considered the strongest proximate predictors of infant mortality and mediate the influence of many other risk factors (Cramer 1987; Hummer et al. 1999; Kline et al. 1989; McCormick 1985).

DATA AND METHOD

Data

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. The data employed are the NCHS linked birth/infant death cohort files for the years 1989-1991 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 1989-1991 and those occurring in 1995-1998. (Note that no linked birth/infant death cohort files were produced by NCHS for 1992-1994.) The

temporal subdivision we use corresponds generally to the time periods before and after the widespread use of pulmonary surfactant therapy and the mounting of the back-to-sleep initiative. As noted above, surfactant replacement did not receive FDA approval until August 1990, and the 1992 American Academy of Pediatricians' recommendation that infants be put to sleep on their back gave rise to the nationwide initiative of 1994. In the case of surfactant replacement, a "cleaner" cut-point would likely result from selection of the two-year 1989-90 period to denote the "pre-surfactant" era (Frisbie et al. 2004, in press), but this would have required deletion of the millions of births that occurred in 1991 and would have been disadvantageous with respect to the analysis of SIDS or congenital malformations. [6] Recent research on changes in infant mortality by cause has, in fact, used 1989-91 as the "before" interval (Muhuri et al. 2004). Furthermore, we estimated a large number of models of the black-white change in risk of infant death from RDS based on the 1989-90 and 1995-98 period. Conclusions were the same as those reported in the present paper, though the magnitude of the contrast in RDS mortality is dampened by including 1991 as part of the pre-surfactant era.

The data analyzed are limited to live births weighing 500 grams or more at birth, which occurred in the United States to women in the three race/ethnic groups. Infants weighing less than 500 grams at birth are excluded because of concern that many of these extremely low weight births are either misclassified stillbirths or result from errors in the recording of birth weight.

Although studies of infant mortality are often limited to singleton births, our core data set includes plural births, as well as singletons. Recent studies of RDS also include plural births (Hamvas et al. 1996; Malloy and Freeman 2000), presumably because, while the number and proportion of all births lost to the analysis when multiple births are excluded is small, this strategy leaves out an important subset of short gestation and low weight births and a rather substantial

[6] We believe that, for research that deals specifically with RDS, 1989-90 comes closest to circumscribing the pre-surfactant period.

proportion of infant deaths. Moreover, the plural birth rate has notably increased over the past decade (Blondel et al. 2002). Note that we also estimated a supplementary set of models that included only singletons. Conclusions derived from the two sets of models were the same, with the direction (and often the magnitude) of the coefficients being quite similar across the two data sets. Hence, the results of the supplementary analysis are not shown, but are available upon request.

We also attempt to shed additional light on whether differential access to intervention or differential need for intervention offers a more adequate explanation changes in race/ethnic disparities over time. We do this by expanding the model to include other causes of infant death, viz., SIDS and congenital anomalies. The utility of including SIDS inheres in the fact that, like RDS, infant mortality from SIDS dropped dramatically between the early and late 1990s after a specific innovation in perinatal care was introduced. The comparison is even more instructive because the changes RDS rates are strongly associated with a "high-tech" innovation, viz., surfactant replacement therapy, that must be administered by professionals in a clinical setting (Hamvas et al. 1996). By contrast, most of the decline in SIDS mortality is credited to the "low-tech" advance represented by putting infants to sleep on their back (Gibson et al. 2000; Pollack and Frohna 2001, 2002), a procedure that is essentially cost-free and does not require paying for the services of a trained professional, for medication, or other costs associated with a clinical setting.

Congenital malformations are a focus because this is the leading cause of infant death in the U.S., and because declines in death from this cause can also be attributed largely to advances in perinatal care, albeit of a more incremental nature. Improvements in prenatal care that can facilitate earlier detection of congenital anomalies and medical advances that, under appropriate conditions, allow fetal malformations to be treated prior to parturition are illustrations in point.

Importantly, comparing the risk of infant death for the Mexican origin population with the risk for blacks provides analytical leverage with respect to the degree to which differential access to

health care interventions is responsible for race/ethnic disparities. Inclusion of the Mexican origin population will make for a stringent test of the conceptual model, because, as has been consistently documented, the Mexican origin population, even compared to blacks, is characterized by lower average levels of education, greater proportions of women lacking adequate prenatal care, and a higher percentage without insurance of any kind to pay for delivery (Frisbie et al. 1997; Moss and Carver 1998). Nevertheless, the risk of infant mortality among Mexican Americans is very similar to that observed among non-Hispanic whites. Further, Mexican immigrant women (indeed immigrants in general), are less apt to have health insurance and more likely to have restricted access to the formal health care system than are their U.S.-born counterparts (Frisbie et al. 2001; LeClere et al. 1994; Thamer et al. 1997). Yet, mortality rates have been found to be lower among infants born to Mexican immigrant women than are the rates for either white infants or for infants born to Mexican American mothers. The debate continues over the extent to which such findings can be explained by positive selection of migration or cultural factors. A third interpretation is that the immigrant advantage is a data artifact attributable to return migration to Mexico.

Methods

We first document trends in race/ethnic disparity in cause-specific infant mortality using rate ratios to describe relative change and difference scores to describe absolute changes in rates. Then, we conduct a multivariate analysis of relative disparities through multinomial logistic regression, with results presented as 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).

As recommended by NCHS, cases are categorized according to maternal race/ethnicity. We distinguish infants of mothers who non-Hispanic whites, non-Hispanic blacks, Mexican Americans, and Mexican immigrants. Our analytic aims dictate that we distinguish Mexican origin infants born to immigrant women from those born to their Mexican American counterparts, rather than simply including nativity as a control variable. It is of course possible to subdivide NHW and NHB infants into groups whose mothers are U.S.-born and those whose mothers are foreign-born, but the number of foreign-born black mothers was rather small which would have made the stability of estimates of some of our more elaborate regressions suspect. In future research, we intend to perform diagnostics on models in which the infants of foreign-born black women, Island-born Puerto Rican women, etc., are distinguished in order to determine the number of risk factors whose effects can appropriately be estimated.

Race/ethnic variation in specific causes of infant death will be analyzed by regressing a five-category outcome: categories of infant death from each of the three leading causes discussed above, an "other cause" residual, and survivors (the referent) on relevant predictor variables. In each case, a series of models will progressively adjust for sets of risk factors. For the most part, after examining the bivariate relationship, we will progressively add blocks of control variables. As already alluded to above, various alternative models are estimated including models for (1) all infants born above 500 grams, (2) LBW infants, (3) singleton births, and various combinations of these—none of which led to differences in the general conclusions

We also conducted separate regressions for relevant time periods and regressions in which time period is a covariate. These two approaches use the same information, and general conclusions from each should be very similar, if not identical. Still, pooling the data with time period as a covariate allows examination of the magnitude and significance of a race x time period interaction term.

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 currently available national data sets of sufficient size to allow modeling the effects of a large number of factors on the individual risk of infant mortality is lack of information either on whether surfactant therapy was administered, [7] on infant sleep position, or on whether antenatal interventions were attempted in the case of congenital anomalies. Thus, a direct test of the differential impact of various innovations in perinatal care and technology cannot be conducted for the nation as whole. 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 dates at which surfactant therapy (late 1990) and the back-to-sleep program (1992-94) began nationwide. This allows a "natural experiment" by observing changes in the black-white infant mortality differentials on either side of these perinatal care thresholds periods. Thus, while no definitive conclusions can be reached, we can at least determine whether the results are consistent with the proposition (Gortmaker and Wise 1997; Wise 2003) that technological innovations in health care are associated with greater racial disparities in infant mortality because of persistent social inequality.

Our analysis is primarily motivated by a desire to respond to the recent call for an expanded research agenda focused on the growth of racial disparity in the face of absolute declines in infant mortality rates (Malloy and Freeman 2000; Wise 2003). However, for reasons delineated earlier, we also model the absolute change in infant mortality due to the several causes of death on a year-to-year basis. Except for our own recent work, we found no multivariate models of absolute change in nationwide race/ethnic differentials in infant mortality in the literature that would allow a direct

[7] An item inquiring as to the use of pulmonary surfactant therapy was added to U.S. birth certificates beginning in 2002. Given the time interval typically required for all (or most) states to adopt new items and the time-lag between the year in which records are reported and NCHS release of data for analysis, it seems unlikely to expect the 2002 linked cohort files to become available before 2006.

comparison with change in individual relative risk. The approach to modeling absolute change that comes most immediately to mind involves regressing mortality rates obtained for counties, cities, or other geographic units on aggregate characteristics of the spatial unit selected. Aggregate studies of this sort are of interest and proven utility, and a number are cited herein. But to generalize findings from such research to relationships at the individual level would be to engage in an obvious "ecological fallacy."

As an alternative to an ecological analysis (i.e., regressing rates on characteristics of some geographic aggregate), we investigated absolute change in the several causes of infant death over time by pooling over the individual data for the time periods 1989-91 and 1995-98, while including dummy variables for each of the seven 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 provide close approximations of the observed annual mortality rates (For a full discussion, see Frisbie et al. 2044, in press). Two dimensions of absolute change in infant mortality were examined: reductions within race/ethnic groups over time and differences in reductions (i.e., second differences) over time. We report only the latter because of space limitations, and more importantly, because second differences indicate whether the absolute decline among NHWs was greater than the absolute decline for minority groups.

DESCRIPTIVE RESULTS

Documenting Changes Over Time

The race/ethnic trends in IMRs per 1000 live births for all thee specific causes of death, and the "other causes" residual are shown in Table 1A for infants born at all weights between 1989-91

and 1995-98. The analogous information for low weight births is shown in Table 1B separately for low weight births.

Looking first at the overall IMRs by race/ethnicity for infants born at all weights (Table 1A), we find that the rank orderings are consistent at both time periods with all previous research. That is, black rates are much higher than rates for the other three groups. However, consonant with vital statistics publications, infant mortality rates fell over time for all groups and all causes shown in Table 1A. As the literature on perinatal care interventions would lead us to expect, the declines in infant death from SIDS and RDS were rather dramatic. The IMRs for NHW and Mexican origin infants are relatively similar, and as is typically observed, a survival advantage is associated with infants of Mexican immigrant mothers. The rate ratios (cause-specific IMRs for minority groups divided by the corresponding NHW rate—see final panels of Table 1A) show just how great the black disparity is in relative terms. With the exception of congenital anomalies, non-Hispanic black infant mortality rates are typically at least twice as high as those for the non-Hispanic whites. By contrast, Mexican American infant rates are only slightly higher, and rates for infants of Mexican immigrants are typically lower, than the NHW rates. The rate of SIDS deaths for immigrants is only half the NHW rate; but, in sharp contrast, the rate for congenital anomalies, like that for NHBs, has a rate ratio greater than one.

--Table 1A about here--

In regard to change in rates, the black relative disparity increased for each of the three specific causes of death between 1989-91 and 1995-98. It is worth noting that the overall black-white disparity declined from a ratio of 2.09 to a ratio of 2.01 (Table 1A). While some improvement is better than none, the overall rate is still double than for NHWs. [8] Although the disparity is <u>much</u> less pronounced, infants born to Mexican American mothers also recorded higher rate ratios for

[8] It is also worth noting that when blacks are compared to all whites (including those of Hispanic origin), the black-white ratio increased between the early and late 1990s.

congenital malformations and RDS. For infants of Mexican immigrants, their relative advantage increased over time; the only exception is in regard to their higher IMR from congenital anomalies where the rate ratio remained unchanged at 1.13.

It is not surprising that the IMRs for LBW infants are much higher than the rates for all births. What may be surprising is that for every cause of death, as well as all causes combined, the percent improvement in survival was greater for whites than for blacks (Table 1B, Panel 3) The contrast is particularly stark in the case of congenital anomalies where the NHW rate of decline was 23.5% as compared to the NHB decline of only 1.65%. Nevertheless, reinforcing the point that genetic factors have little impact is the fact that the black IMR for congenital anomalies remains less than the white IMR from that cause. Note further that among these high-risk infants, black-white rate ratios for both SIDS and RDS increased between 1989-91 and 1995-98; for SIDS, the black-white ratio changed from 1.41 to 1.64, while for RDS the change was from near parity (1.01) to a rate ratio of 1.28. [9]

--Table 1B about here--

Comparing the NHW white population with the Mexican origin population, we again see that, unlike the case for all births, LBW infants born to Mexiccan immigrants had a higher overall IMR in 1989-91, compared to either NHW infants or infants born to their Mexican American counterparts. However, by 1995-98, the overall IMR was lower among immigrants than for any group except NHWs. One of the most notable findings from Table 1B is that what was a LBW Mexican American survival advantage relative to their high-risk NHW counterparts in 1989-91 had become a survival disadvantage in regard to both congenital anomalies and RDS—thereby paralleling the findings for NHBs. The opposite occurred in regard to the rate of death from SIDS (rate-ratios = 1.17 and 0.92 in the earlier later time periods respectively). The rate ratio comparing

[9] If 1989-90 is taken as the pre-surfactant period, there is a reversal from a black RDS survival advantage to a black disadvantage.

infants of Mexican immigrants to NHW infants showed very little change overall between 1989-91 and 1995-98, but this "non-change" was the result of a rising rate ratio associated with congenital anomalies and a declining rate ratio for SIDS (last panel of Table 1B).

Risk Factor Distributions

The distribution of risk factors by race/ethnicity appears in Table 2. The distributions are consistent with all prior comparisons, including the finding that births to Mexican immigrants make up over 60% of all Mexican origin births. Thus, we highlight only those differentials that present the sharpest contrasts. Mexican Americans and NHBs are more likely to give birth as young (< 18) teenagers—though the percentage of teen births declined a bit for blacks and rose a bit for Mexican American women between 1989-91 and 1995-98. Well over 90% of NHW and Mexican immigrant mothers are over 18 years of age—a similarity that likely reflects marital preferences for the former group and selectivity of migration for the latter. About 80 % of NHW mothers are married, as compared to roughly 3/5th of Mexican origin women, and 1/3 of NHB women. However, the percent married among Mexican America mothers had dipped to 54.6 % by 1995-98. The Mexican origin population is especially disadvantaged with regard to education. About 40% of Mexican American mothers and roughly 70% of Mexican immigrant mothers had less than a high school education at both time periods. Mexican immigrant women are the most likely to have received inadequate prenatal care—though their situation in this regard improved substantially over time. The proportion with adequate PNC is highest for NHWs, followed by Mexican Americans, NHBs, and Mexican immigrants in descending order. It warrants mention that both groups of Mexican origin women are least likely to present with medical risks or labor/delivery complications. NHW women are the most apt to smoke, while smoking is low for Mexican American women, and close to nonexistent for Mexican immigrant women. Blacks, of course, have much higher rates of low weight and preterm births. Note, however, that these data are consistent with previous studies (Demissie et al. 2001;

Frisbie and Song 2003) that show small decline in rates of adverse birth outcomes among blacks, and small increases for most other race/ethnic groups.

--Table 2 about here--

RESULTS FROM REGRESSION MODELS

We begin the discussion of multivariate results with comparisons of infants born at all weights and then compare these results with those obtained among LBW infants. We are interested primarily in estimating, on a nationwide level, race/ethnic differentials and changes in differentials by specific cause of infant death. Specifically, we want to draw regarding the reasons for changes in race/ethnic differentials in risk of infant death that followed the rather dramatic innovations in perinatal care that emerged in the early- to mid-1990s. The effects of risk factors used as controls are quite similar to those estimated by previous research and inasmuch as these effects are consistent as to direction over time. Thus, any discussion or tabular presentation of these relationships would be largely redundant, and therefore we present odds ratios only for the effects of race/ethnicity, time period, and the race/ethnicity x time period interaction. However, we do show how the risk of infant death varies by these core variables as different blocks of risk factors are simultaneously controlled. (Estimates for all risk factor effects are available upon request.)

Multivariate Results: Comparison by Time Period

Table 3 consists of two panels—the first showing the relationships of interest for the 1989-91 time period and the second for the 1995-98 time period. Model 1 for 1989-91 presents the simple bivariate relationships which show that the risk of NHB infant mortality is 2 to almost 3 times higher than the NHW risk for all causes (relative to survival), except for congenital anomalies where the odds ratio (OR) is only 1.152. The bivariate risk for infants of Mexican American mothers is statistically identical to, or slightly greater than, the NHW risk for all causes. By contrast, infants of Mexican immigrants have a significant survival advantage for all causes, except congenital

malformations (OR = 1.13—very similar to the value for NHB infants). Model 2 adds controls for basic demographic variables, as well as for maternal education and prenatal care. (See the stubs of the Table for the list of controls.) In every case, the odds of infant mortality for the three minority groups are substantially reduced (compared to NHW infants), and the two Mexican origin groups show survival advantages for every specific cause in 1989-91. Model 3 adds controls for biomedical and behavioral risks, but adjustment for these latter risk factors has little effect. The story is quite different for NHB when birth weight and gestational age are controlled in Model 4. In fact, NHB infants have a survival advantage vis a vis NHW infants in regard to both congenital anomalies and RDS (OR = 0.627 and OR = 0.820). The NHB odds for SIDS are only slightly higher (OR = 1.048), but the difference is significant, and the black disparity for all other causes is also much reduced). Net of controls for birth outcomes, the risk for both of the Mexican origin populations increases slightly, but except for the odds ratio associated with congenital anomalies among infants of immigrants, the Mexican origin population has a large to moderate survival advantage during this early time period.

In 1995-98 (Panel 2 of Table 3), the bivariate model demonstrates that the NHB odds of infant mortality are higher than the odds estimated for the 1989-91 period. The odds for infants of Mexican origin are identical in direction and show only modest changes in magnitude when the risks for the later time period are compared with those for the earlier period. Note, however, that the odds of infant death from congenital anomalies are significantly greater for each of minorities in Model 1. Controlling for demographic and access-to-care variables (education and prenatal care) has the same sort of effect in the 1995-98 data as in the 1989-91 data. The disparity separating NHW and NHB is diminished, and in every comparison, the Mexican origin infants have a survival advantage—an advantage that is most pronounced for the immigrant population. Estimates based on Model 3 again are generally not greatly different. Controlling birth weight and gestational age in 1995-98 results in

a diminution of the NHB disparity. In fact, the odds ratio for congenital anomalies is reduced to 0.627. Little change occurs in the estimates for the Mexican origin population, but note that, following adjustment for birth weight and gestational age, the risk of death from congenital anomalies becomes statistically identical to the NHW risk.

--Table 3 about here--

The most striking difference observed in the full model (Model 4) in Panel 2 of Table 3 where the survival advantage for NHBs in regard to congenital anomalies is slightly eroded, while the NHB black survival advantage in risk of RDS death seen in 1989-91 (OR = 0.820, $p \leq 0.01$) is wiped out in the later time period (OR = 1.036, ns), thereby confirming the reversal observed by Hamvas et al. (1996) using clinical data. Further, the NHB odds of death from SIDS and all other causes are slightly to modestly higher in the period after the introduction of innovations in perinatal care and technology.

Table 4 presents the odds ratios for low weight births. Findings are similar in a broad sense to those shown for births at all weights, but several differences warrant mention. First, in every single comparison across models and in both time periods, the odds of NHB infant death among low weight infants (Table 4) are lower than the corresponding odds for infants born at all weights (Table 3). [10] This finding is consistent with, but of course does not prove, the proposition that LBW black infants mature at lower weights and shorter gestations than do their white counterparts. However, exactly the same pattern holds for infants born to Mexican immigrants. Since there have been no studies that examine the possibility of more rapid fetal maturity among babies of immigrants, the potential for some alternative explanation must be considered. No consistent pattern is seen in the comparison between 1989-91 and 1995-98 odds ratios for the Mexican American population. It

[10] Comparisons can be made only with the first three models of Tables 3 and 4 inasmuch as birth weight and gestational age are not controlled in the population of LBW infants.

does not seem likely to us that that fetal maturation could be greatly different for Mexican Americans as compared to Mexican immigrants, however.

--Table 4 about here--

Multivariate Results: Time Period as a Covariate

Table 5 includes time period as a covariate for infants born at all weights; Table 6 presents the analogous estimates for LBW infants.

The most notable finding from Table 5 is that odds of infant morality among infants born at all weights are significantly lower in 1995-98 than in 1989-91. This finding is unsurprising given the widely documented decline in infant mortality overall, and for most specific causes) over the past several decades. Note also that, as would be expected from the comparisons of estimates for 1989-91 and 1995-98 in Table 3, the control for time period in Table 5 results in a significantly lower risk of RDS for NHB infants, and the NHB disadvantage associated with SIDS becomes non-significant in Table 5. However, the race/ethnicity x time period interaction, the full model (Model 4 of Table 5) shows an offset to the main effects such that it was substantially more risky to be black in 1995-98, as compared to 1989-91, for the three specific causes of death: 14% greater risk in the case of congenital anomalies, 21% greater risk from SIDS, and 28% greater risk from RDS. The interaction offset was similar for infants of Mexican American mothers, with the exception of SIDS. In regard to congenital malformations and RDS, the risks associated with being an infant of a Mexican American mother were 17% greater and 22% greater in the period after the nationwide introduction of advances in perinatal care. The risk was also slightly greater for all other causes for this population. The story is different for infants born to Mexican immigrant women. In this case, there is a <u>reinforcement of decline</u> in risk for all causes, save for congenital anomalies, and here the interaction effect (OR = 1.013) is statistically nonexistent.

--Table 5 about here--

Table 6 presents the analogous estimates for low weight births (except, of course, birth weight and gestational age are not among the controls). For LBW infants, the patterns are very similar to those observed for all infants in Table 5. Infant mortality from each cause is reduced in 1995-98 as compared to 1989-91. Controlling for time period results in a significant survival advantage among NHBs, rather a survival disadvantage as seen in Panel 2 of Table 4. Just as in Table 5, however, the race/ethnicity x time period interaction among LBW infants shows that it was riskier to be a NHB infant in 1995-98 than in the earlier time period—again offsetting to some degree the advantageous main effect observed among NHB infants. Table 6 also reveals the same sort of offset among infants of Mexican American mothers. Just as in Table 5, the offset occurs among LBW infants in Table 6 for congenital malformations and all other causes. The interaction terms for SIDS and RDS are not statistically significant. Finally, the estimates for infants of Mexican immigrants are identical in pattern for LBW infants as compared to those for infants born at all weights. There is an additional decline in risk associated with the interaction term in the case of SIDS and all other causes, while the other two interaction terms (congenital anomalies and RDS) are not significant.

Models of Absolute Change in Infant Mortality

Two dimensions of absolute change in infant mortality by race were examined: reductions over time and differences in reductions (i.e., second differences) over time. Only the results of estimates of absolute second differences for low birth weight infants are presented in tabular form because of space limitations, and because it is for this population that the greatest departures from the wide-spread view that declines in infant mortality rates are greater for minority (most notably black) infants occur. However, results from both bivariate and full models are shown. Note that negative signs indicate that the decline in infant mortality was greater for non-Hispanic white infants than for the minority group with which the comparisons are drawn. Note also that second

differences compare the change in approximated rates for each minority with the change for the NHW majority population.

When the relationships of interest are modeled for the LBW infants (Table 7), we see that, in the bivariate models for both congenital anomalies and RDS, mortality declines, in every instance, reflect an advantage for NHW infants over NHB infants (as indicated by the negative signs), and that all relationships are statistically significant. Thus, the trend in among LBW infants seen in the raw rates (Table 1B) is also reflected in Table 7. The black-white changes in approximated rates (bivariate model) are mixed in the case of SIDS, but none of the second differences is significant. NHB infants did experience greater reductions in rates than did NHW infants in the residual category—i.e., all other causes, and from 1995 onward, these declines in disparity are significant in the bivariate model. Notably, in changes in rates for congenital anomalies, even with full controls, NHB infants lost ground relative to their NHW counterparts, and the widening disparity was significant from 1995 onward. The same is generally true of the narrowing of the NHB-NHW disparity with respect to all other causes. None of the approximations of changes for SIDS and RDS are significant in the full model for blacks and whites.

The second-differences show a heretofore unrecognized rising level of disadvantage for infants of Mexican American women as compared to NHW infants. For every cause of death, and for nearly every year-to-year comparison, the rate of decline for NHW infants was greater than the decline for Mexican American infants. Only a few of the change differences are significant, but recall that we are working with the entire population, not a sample.

The only major differences in changes in infant mortality rates for infants of Mexican immigrants occur in approximated rates for SIDS and RDS. The bivariate model shows that improvements in SIDS survivorship were consistently larger for the NHW population than for the Mexican immigrant population. The reverse is true for RDS. Again, only a few of the comparisons

reach conventional levels of significance in the bivariate model, and no significant effects are seen in the full model. However, for all the comparisons drawn, it is well to remember that the bivariate relationships show what exists in reality, while models with controls indicate what the difference would be if compositional equality existed in regard to control variables.

CONCLUSIONS

In regard to our first (descriptive) aim, we found considerable change in cause-specific rates of infant death in the wake of notable advances in perinatal care and technology that occurred in the early- to mid-1990s. Indeed, for all three specific causes of death (congenital anomalies, SIDS, and RDS), the relative disparity (rate ratios) between NHB and NHW rates increased between 1989-91 and 1995-98. The widening of the NHB/NHW gap was even greater when only LBW infants were considered (Table 1B). [11]

It was not unexpected that the IMRs for infants of Mexican American mothers would be modestly higher than the rates for NHW infants. However, we did not necessarily expect that the disparity would widen, but that, in fact, was the trend. The cause-specific rates for the Mexican American population taken as ratios to the NHW population increased for congenital anomalies and RDS, regardless of whether the comparison is for all births or for low weight births only. One bright spot was that the Mexican American survival disadvantage for SIDS among LBW infants observed in 1989-91 (rate ratio = 1.17) was actually reversed by 1995-98. That is, infants born to Mexican American women were slightly less likely to die of SIDS than were NHW infants (rate-ratio = .92) in the later time period.

By contrast, the rate changes were more favorable for infants of Mexican immigrant mothers than for infants of NHW mothers in virtually every comparison, including those for causes of death where the immigrant group already held a sizeable advantage. For example, infants of Mexican

[11] Among LBW infants, the infant mortality rate for congenital anomalies is lower for NHB infants than for NHW infants. However, the NHB advantage eroded to some degree between 1989-91 and 1995-98.

immigrants were only about half as apt to die of SIDS as NHW infants in 1989-91 (rate ratio about 0.5 in both Tables 1A and 1B). By 1995-98, the rate ratios had declined to approximately 0.4. Essentially, the only mortality disadvantage for immigrant infants was in regard to congenital anomalies—especially those born at low weights.

One primary analytic aim was to determine the extent to which race/ethnic differentials in cause-specific infant mortality could be explained by the sort of "fundamental social causes" described by Link and Phelan (1995, 1996, 2002). Here the conclusion is quite clear. Controlling for sociodemographic, SES, biomedical, and behavioral risk factors leads, in almost every instance, not only to a diminution of the NHW-minority group disparity that usually (but not always) is found in the bivariate relationships, but also to an actual reversal to a minority group survival advantage. Of course, infants of Mexican immigrants tend to have lower infant mortality to begin with, and then, with controls added, tend maintain or strengthen the survival advantage. In any case, we can safely conclude that social inequality accounts for a large portion of the large disparities in risk of infant death between the NHB and NHW populations and for the modest disadvantage we often observed among infants of Mexican American mothers.

Another analytic objective was two-pronged—(a) to determine to what extent minority populations became more disadvantaged with respect to infant mortality after great strides were made in perinatal care and technology and (b) to shed at least a little light on the issue of whether increasing disparities between the majority and minority populations can be more appropriately attributed to differential access to intervention or to differential need for intervention. The answer to question (a) is fairly straightforward. For specific causes of infant death regarding which one or more notable perinatal care advances occurred (and based on the fully adjusted regression models), the NHW population was more likely to enjoy greater benefits from the new (or improved) interventions than was the case for infants of NHB or Mexican American women. The conclusions

reached based on Tables 3 and 4 were reconfirmed by the finding that the race/ethnicity x time period interaction term typically had a deleterious effect for minority groups. That is, it was relatively more risk to be a NHB or Mexican American infant in 1995-98 than it was in the "pre-innovation" period. Surprisingly, the exact reverse was true of the change in risk over time for babies of Mexican immigrants. In almost every comparison on Tables 3 and 4, the risk for the Mexican immigrant population compared to NHW population reflected an overtime increase in what was already a Mexican immigrant advantage. The one exception is the slightly greater risk of infant mortality due to congenital anomalies, and for this condition, the NHW advantage was narrowed.

Can the deleterious changes that occurred among NHB and Mexican American infants between 1989-91 and 1995-98, compared to NHW infants, be more reasonably attributed to differential access to health care interventions or to differentials in the need for intervention? Although Mexican American women of childbearing age exhibit some behavioral and health endowment advantages (such as a lower prevalence of previous pregnancy loss and of medical complications) in regard to the variables that theoretically at least should be most strongly associated with obstacles to accessing health care (e.g., years of completed education and adequacy of prenatal care), all of the minority groups are highly disadvantaged. The main finding supporting the differential need for intervention explanation is that the risk of infant mortality among LBW minority infants is usually lower than the risk for infants born at all weights (above 500 grams). This finding is consistent with the conclusion reached by several scholars that LBW black infants mature at lower weights and shorter gestations than do LBW white infants (Kline et al. 1989; Wilcox and Russell 1986, 1990; Hamvas et al 1986). However, the only analysis we were able to find that bears on the issue among Mexican Americans (Overpeck et al. 1999) found some evidence that the birth weight for gestational age was slightly higher for infants of U.S.-born Mexican American women than was the case among NHW infants. However, these authors conclude that "the ability to

recognize (either) fetal growth retardation or excessive growth is questionable" in comparing these two groups (Overpeck et al. 1999: 943).

The picture becomes even more complex when the issue of changes in infant mortality risk for infants of Mexican immigrants is considered. If there is any group where obstacles to medical intervention can be expected to be large and numerous, it is this group. Mexican immigrant women have much lower levels of education, are much more apt to have inadequate prenatal care (though access to PNC seems to be improving somewhat), and, as other research has shown are much less likely to have medical insurance of any kind to help pay for delivery (Frisbie et al. 2001; LeClere et al. 1994; Thamer et al. 1997). Thus, the findings for Mexican immigrants undermines the differential need for intervention explanation—unless one is inclined to believe that migration is so highly and positively selective on health that this more than overrides obstacles to perinatal care access.

Finally, our analysis of absolute change (via logit approximated rates) leads to a mixed conclusion. At least among high-risk (LBW) infants born to NHB or Mexican American women, the absolute minority disadvantage in infant survival changes is widening in the case of congenital anomalies and RDS. However, in regard to SIDS and all other causes, the majority-minority gap is either closing or at least not significantly widening.

Why the difference in patterns across the specific causes of infant death? We suspect that the explanation is most likely to be found in the differential access to intervention hypothesis. For congenital anomalies and RDS, preventive and curative interventions are administered by health professional in a clinical setting—often in a Neonatal Intensive Care Unit (NICU). Such interventions are achieved only at considerable monetary cost—costs which many minority members may not be able to bear due to lack of money or medical insurance or both. By contrast, major

reductions in SIDS mortality have occurred by the cost-free innovation of placing infants to sleep on their backs.

All things considered (including the advantaged position of infants of Mexican immigrants—which is no doubt partly, if not largely, due to selectivity of migration), we believe our results lead to the conclusion that social inequality explains a very large proportion of race/ethnic differentials in infant mortality. We also believe that our findings make a stronger case for the differential access to intervention explanation for the increase in the infant mortality disadvantage among minorities that occurred in the wake of major advances in perinatal care and technology.

REFERENCES

Alexander, G.R., M.E. Tompkins, M.C. Allen, and T.C. Hulsey. 1999. "Trends and Racial Differences in Birth Weight and Related Survival." *Maternal and Child Health Journal* 3: 71-79.

Alexander, G.R., and M. Kotelchuck. 2001. "Assessing the Role and Effectiveness of Prenatal Care: History, Challenges, and Directions for Future Research." *Public Health Reports* 116: 306-316.

Black, L., R.J. David, R.T. Brouillette, and C.E. Hunt. 1986. "Effects of Birth Weight and Ethnicity on Incidence of Sudden Infant Death Syndrome." *The Journal of Pediatrics* 108: 209-214.

Blalock, H.M., Jr. 1979. *Social Statistics*. Rev. 2nd edition. New York: McGraw-Hill.

Blondel, B., M.D. Kogan, G.R. Alexander, N. Dattani, M.S. Kramer, A. Macfarlane, and S.W. Wen. 2002. "The Impact of the Increasing Number of Multiple Births on the Rate of Preterm Birth and Low Birthweight: An International Study." *American Journal of Public Health* 92: 1323-1330.

British Columbia Reproductive Care Program. 1993. "Surfactant Replacement Therapy" *Newborn Guideline* 6: 1-4.

Cabral, H., L.E. Fried, S. Levenson, H. Amaro, and B. Zuckerman. 1990. "Foreign-Born and U.S.-Born Black Women: Differences in Health Behaviors and Birth Outcomes." *American Journal of Public Health* 80: 70-72.

Chomitz, V.R., L.W.Y. Cheung, and E. Lieberman. 1995. "The Role of Lifestyle in Preventing Low Birth Weight." *The Future of Children* 5: 121-138.

Cobas, J.A., H. Balcazar, M. Benin, V.M. Keith, and Y. Chong. 1996. "Acculturation and Low-Birthweight Infants among Latino Women: A Reanalysis of HHANES Data with Structural Equation Models." *American Journal of Public Health* 86: 394-396.

Cramer, J.C. 1987. "Social Factors and Infant Mortality: Identifying High-Risk Groups and Proximate Causes." *Demography* 24: 299-322.

Davies, D.P. 1994. "Ethnicity and the Sudden Infant Death Syndrome: An Introduction." *Early Human Development* 38: 139-141.

Demissie, K., G.G. Rhoads, C.V. Anath, G.R. Alexander, M.S. Kramer, and K.S. Joseph. 2001. "Trends in Preterm Birth and Neonatal Mortality among Blacks and Whites in the United States from 1989 to 1997." *American Journal of Epidemiology* 54: 307-315.

Eberstein, I.W., C. Nam, and R. A. Hummer. 1990. "Infant Mortality by Cause of Death: Main and Interaction Effects." *Demography* 27: 413-430.

Ferrara, B.T., F.E. Hoekstra, R.J. Couser, E.P. Gaziana, S.E. Calvin, N.R. Payne, and J.J. Fangman. 1994. "Survival and Follow-Up of Infants Born at 23 to 26 Weeks of Gestational Age: Effects of Surfactant Therapy." *The Journal of Pediatrics* 124: 119-124.

Frisbie, W.P. 2005. "Infant Mortality." Chapter 9 in D.L. Poston, Jr., and M. Micklin (eds.) *Handbook of Population*. New York: Kluwer/Plenum (Forthcoming).

Frisbie, W.P., M. Biegler, P.B. de Turk, D. Forbes, and S. G. Pullum. 1997. "Racial and Ethnic Differences in Determinants of Intrauterine Growth Retardation and Other Compromised Birth Outcomes." *American Journal of Public Health* 87: 1977-1983.

Frisbie, W.P., D. Forbes, and R.A. Hummer. 1998. "Hispanic Pregnancy Outcomes: Additional Evidence." *Social Science Quarterly* 79: 149-169.

Frisbie, W.P., Y. Cho, and R.A. Hummer. 2001. "Immigration and the Health of Asian and Pacific Islander Adults in the United States" *American Journal of Epidemiology* 153:372-380.

Frisbie, W.P., and S.E. Song. 2003. "Hispanic Pregnancy Outcomes: Differences Over Time and Current Risks." *The Policy Studies Journal* 31: 237-252.

Frisbie, W.P., S.E. Song, D.A. Powers, and J.A. Street. 2004. "Increasing Racial Disparity in

Infant Mortality: Respiratory Distress Syndrome and Other Causes." *Demography* (In Press).
Geronimus, A.T. 1987. "On Teenage Childbearing and Neonatal Mortality in the United States." *Population and Development Review* 13: 245-279.
Geronimus, A.T., and S.D. Korenman. 1993. "Maternal Youth or Family Background?: On the Health Disadvantages of Infants with Teenage Mothers." *American Journal of Epidemiology* 137: 213-225.
Gibson, E., C.A. Dembofsky, S. Rubin, and J.S. Greenspan. 2000. "Infant Sleep Position 2 Years into the 'Back to Sleep' Program." *Clinical Pediatrics* May: 285-289.
Gortmaker, S.L., and P.H. Wise. 1997. "The First Injustice: Socioeconomic Disparities, Health Services Technology, and Infant Mortality." *Annual Review of Sociology* 23: 147-170.
Grether, J.K., and J. Schulman. 1989. "Sudden Infant Death Syndrome and Birth Weight." *The Journal of Pediatrics* 114: 561-567.
Guyer, B., M.F. MacDorman, J.A. Martin, K.D. Peters, and D.M. Strobino. 1998. "Annual Summary of Vital Statistics-1997." *Pediatrics* 102: 1333-1349.
Halliday, H.L. 1997. "Surfactant Therapy: Questions and Answers." *Journal of Neonatal Nursing* 3: 28-36.
Hamvas, A. 2000. "Disparate Outcomes for Very Low Birth Weight Infants: Genetics, Environment or Both?" *Journal of Pediatrics* 136: 427-428.
Hamvas, A., P.H. Wise, R.K. Yang, N.S. Wampler, A. Noguchi, M.M. Maurer, C.A. Walentik, W.F. Schramm, and F.S. Cole. 1996. "The Influence of the Wider Use of Surfactant Therapy on Neonatal Mortality among Blacks and Whites." *The New England Journal of Medicine* 334: 1635-1640.
Hollander, D. 1995. "Improvements on Neonatal Care Have Increased Survival Rates for Very Low Birth Weight Infants." *Family Planning Perspectives* 27: 182-183.
Hoyert, D.L., E. Arias, B.L. Smith, S.L. Murphy, and K.D. Kochanek. 2001. "Deaths: Final Data for 1999." *National Vital Statistics Reports* 49, No. 8. Hyattsville, MD: National Center for Health Statistics.
Hummer, R.A., M. Biegler, P.B. de Turk, D. Forbes, W.P. Frisbie, Y. Hong, and S.G. Pullum. 1999. "Race/Ethnicity, Nativity, and Infant Mortality in the United States." *Social Forces* 7: 1083-1118.
Institute of Medicine. 1988. *Reaching Mothers, Reaching Infants*. Washington DC: National Academy Press.
Jeffery, H.E., A. Megevand, and M. Page. 1999. "Why the Prone Position Is a Risk Factor for Sudden Infant Death Syndrome." *Pediatrics* 104: 263-269.
Kallan, J.E. 1993. "Race, Intervening Variables, and Two Components of Low Birth Weight." *Demography* 30: 489-506.
Kline, J., Z. Stein, and M. Susser. 1989. *Conception to Birth: Epidemiology of Prenatal Development*. New York: Oxford.
Kleinman, J.C., and S.S. Kessel. 1987. "Racial Differences in Low Birth Weight: Trends and Risk Factors." *The New England Journal of Medicine* 317: 749-753.
Kochanek D.K., and J.A. Martin. 2004. *Supplemental Analyses of Recent Trends in Infant Mortality*. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Health Statistics. Available at http://www.cdc.gov/nchs/data/nvsr/nvsr52/nvsr52_13.pdf.
LeClere, F.B., L. Jensen, and A.E. Biddlecom. 1994. "Health Care Utilization, Family context, and Adaptation among Immigrants to the United States." *Journal of Health and Social Behavior* 35: 370-384.
Lee, C.S., B. Khoshnood, L. Chen, S.N. Wall, W.J. Cromie, and R.L. Mittendorf. 2001. "Infant

Mortality from Congenital Malformations in the United States." *Obstetrics and Gynecology* 98: 620-627.
Link, B.G., and J. Phelan. 1995. "Social Conditions as Fundamental Causes of Disease." *Journal of Health and Social Behavior* 35: 80-94.
_____. 1996. "Understanding Sociodemographic Differences in Health – the Role of Fundamental Social Causes." *American Journal of Public Health* 86: 471-472.
_____. 2002. "McKeown and the Idea That Social Conditions Are Fundamental Causes of Disease." *American Journal of Public Health* 92: 730-732.
Mathews, M.S., F. Menacker, and M.F. MacDorman. 2002. "Infant Mortality Statistics from the 2000 Period Linked Birth/Infant Death Data Set." *National Vital Statistics Reports* 50, No. 12. Hyattsville, MD. National Center for Health Statistics.
Malloy, M.H., and D.H. Freeman. 2000. "Respiratory Distress Syndrome Mortality in the United States, 1987-1997." *Journal of Perinatology* 20: 414-420.
McCormick, M.C. 1985. "The Contribution of Low Birth Weight to Infant Mortality and Childhood Mortality." *New England Journal of Medicine* 312: 82-90.
Minino, A.M., E. Arias, K.D. Kochanek, S.L. Murphy, and B.L. Smith. 2002. "Deaths: Final Data for 2000." *National Vital Statistics Reports* 50, No. 15. Hyattsville, MD: National Center for Health Statistics.
Moon, R.Y., J.L. Gingras, and R. Erwin. 2002. "Physician Beliefs and Practices Regarding SIDS and SIDS Risk Reduction." *Clinical Pediatrics* July/August: 391-395.
Morales, W.J., W.F. O'Brien, J.L. Angel, R.A. Knuppel, and S. Sawai. 1989. "Fetal Lung Maturation: The Combined Use of Corticosteroids and Thyrotroin-Resisting Hormone." *Obstetrics and Gynecology* 73: 111-116.
Moss, N.E., and K. Carver. 1998. "The Effect of WIC and Medicaid on Infant Mortality in the United States." *American Journal of Public Health* 88: 1354-1361.
Muhuri, P. K., M. F. MacDorman, and T. M. Ezzati-Rice. 2004. "Racial Differences in Leading Causes of Infant Death in the United States." *Paediatric and Perinatal Epidemiology* 18: 51-60.
Nsiah-Jefferson, L. 1993. "Access to Reproductive Genetic Services for Low-Income Women and Women of Color." *Fetal Diagnosis Therapy* 8: 107-127.
Overpect, M.D., M.L. Hediger, J. Zhang, A.C. Trumble, and M.A. Klebanoff. 1999. "Birth Weight for Gestational Age of Mexican American Infant Born in the United States." *Obstetrics and Gynecology* 93: 943-947.
Palloni, A., and J.D. Morenoff. 2001. "Interpreting the Paradoxical in the Hispanic Paradox: Demographic and Epidemiologic Approaches." *Annals of the New York Academy of Sciences* 954: 140-174.
Pollack, H.A., and J.G. Frohna. 2001. "A Competing Risk Model of SIDS Incidence in Two Birth Cohorts." *The Journal of Pediatrics* 138: 661-667.
Pollack, H.A., and J.G. Frohna. 2002. "Infant Sleep Position After the Back to Sleep Campaign." *Pediatrics* 109: 608- 614.
Ranganathan, D., S. Wall, B. Khoshnood, J.K. Singh, and K.S. Lee. 2000. "Racial Differences in Respiratory-Related Neonatal Mortality among Very Low Birth Weight Infants." *The Journal of Pediatrics* 136: 454-459.
Scribner, R. 1996. "Editorial: Paradox as Paradigm - The Health Outcomes of Mexican Americans." *American Journal of Public Health* 86: 303-305.
Shiono, P.H., and R.E. Behrman. 1995. "Low Weight Birth: Analysis and Recommendations." *The Future of Children* 5: 4-18.
Singh, G.K., and S.M. Yu. 1996. "Adverse Pregnancy Outcomes: Difference between US- and

Foreign-Born Women in Major US Racial and Ethnic Groups." *American Journal of Public Health* 86: 837-843.
Sowards, K.A. 1997. "Premature Birth and the Changing Composition of Newborn Infectious Disease Mortality: Reconsidering "Exogenous" Mortality." *Demography* 34: 399-409.
Thamer, M., C. Richard, A.W. Casebeer, and N.F. Ray 1997. "Health Insurance Coverage among Foreign-Born U.S. Residents: The Impact of Race, Ethnicity, and Length of Residence." *American Journal of Public Health* 87: 96-102.
U.S. Bureau of the Census. 2001. *Statistical Abstract of the United States: 2001.* Washington, DC.
U.S. Department of Health and Human Services. 1995. "Linked Birth/Infant Death Data Set: 1989 Cohort." *Public Use Data File Documentation*. Hyattsville: National Center for Health Statistics.
U.S. Department of Health and Human Services. 2000. "With Understanding and Improving Health and Objectives for Improving Health." *Healthy People 2010.* 2nd ed. Washington DC: U.S. Government Printing Office.
Wilcox, A.J., and I.T. Russell. 1986. "Birthweight and Perinatal Mortality: III. Towards a New Method of Analysis." *International Journal of Epidemiology* 15: 188-196.
_____. 1990. "Why Small Black Infants Have a Lower Mortality Rate than Small White Infants: The Case for Population-Specific Standards for Birth Weight." *Journal of Pediatrics* 116: 7-10.
Willinger, M., H.J. Hoffman, K.T. Wu, J.R. Hou, R.C. Kessler, S.L. Ward, T.G. Keens, and M.J. Corwin. 1998. "Factors Associated with the Transition to Nonprone Sleep Positions of Infants in the United States." *Journal of the American Medical Association* 280: 329-335.
Wise, P.H. 2003. "The Anatomy of a Disparity in Infant Mortality." *Annual Review of Public Health* 24: 341-362.

Table 1A. Infant Mortality Rates* by Race/Ethnicity: United States, 1989-1991, and 1995-1998

	1989-1991				1995-1998				% Reduction in Rates between 1989-91 and 1995-98				1989-1991			1995-1998		
	NHW	NHB	MX-US	MX-IM.	NHW	NHB	MX-US	MX-IM.	NHW	NHB	MX-US	MX-IM.	NHB /NHW	MX-US /NHW	MX-IM. /NHW	NHB /NHW	MX-US /NHW	MX-IM. /NHW
IMR per 1000	5.94	12.41	6.56	5.38	4.64	9.34	5.25	4.05	-21.89	-24.74	-19.97	-24.72	2.09	1.10	0.91	2.01	1.13	0.87
Con. Anom.	1.68	1.92	1.67	1.90	1.36	1.65	1.49	1.53	-19.05	-14.06	-10.78	-19.47	1.14	0.99	1.13	1.21	1.10	1.13
SIDS	1.15	2.20	1.27	0.58	0.67	1.43	0.68	0.27	-41.74	-35.00	-46.46	-53.45	1.91	1.10	0.50	2.13	1.01	0.40
RDS	0.48	1.14	0.50	0.39	0.21	0.55	0.25	0.16	-56.25	-51.75	-50.00	-58.97	2.38	1.04	0.81	2.62	1.19	0.76
Other	2.62	7.15	3.11	2.52	2.39	5.72	2.84	2.09	-8.78	-20.00	-8.68	-17.06	2.73	1.19	0.96	2.39	1.19	0.87

* Rates per 1000 live births.

SOURCE: NCHS Linked Birth/Infant Death Files. 1989-1991, and 1995-1998.

Table 1B. Infant Mortality Rates* among Low Weight Births by Race/Ethnicity: United States, 1989-1991, and 1995-1998

	1989-1991				1995-1998				% Reduction in Rates between 1989-91 and 1995-98				1989-1991			1995-1998		
	NHW	NHB	MX-US	MX-IM.	NHW	NHB	MX-US	MX-IM.	NHW	NHB	MX-US	MX-IM.	NHB /NHW	MX-US /NHW	MX-IM. /NHW	NHB /NHW	MX-US /NHW	MX-IM. /NHW
IMR per 1000	54.43	60.55	53.61	57.57	38.98	47.23	42.28	41.62	-28.39	-22.00	-21.13	-27.71	1.11	0.98	1.06	1.21	1.08	1.07
Con. Anom.	14.91	7.86	12.54	18.68	11.41	7.73	11.60	15.37	-23.47	-1.65	-7.50	-17.72	0.53	0.84	1.25	0.68	1.02	1.35
SIDS	3.25	4.57	3.79	1.76	1.91	3.14	1.75	0.66	-41.23	-31.29	-53.83	-62.50	1.41	1.17	0.54	1.64	0.92	0.35
RDS	8.65	8.75	8.05	7.65	3.34	4.26	3.60	2.88	-61.39	-51.31	-55.28	-62.35	1.01	0.93	0.88	1.28	1.08	0.86
Other	27.62	39.37	29.23	29.48	22.32	32.09	25.33	22.71	-19.19	-18.49	-13.34	-22.96	1.43	1.06	1.07	1.44	1.13	1.02

* Rates per 1000 live births.

SOURCE: NCHS Linked Birth/Infant Death Files. 1989-1991, and 1995-1998.

Table 2. Percent Distributions on Risk Factors of Infant Mortality by Race/Ethnicity : United States, 1989-1991, and 1995-1998

	1989-1991				1995-1998			
Risk Factors	**NHW**	**NHB**	**MX-US**	**MX-IM.**	**NHW**	**NHB**	**MX-US**	**MX-IM.**
Place of Birth								
U.S.	95.92	93.49	37.38		95.26	91.20	39.17	
Other	4.08	6.51		62.62	4.74	8.80		60.83
Maternal Age								
Under 18	5.79	16.32	16.77	8.52	5.88	15.82	19.18	7.65
18 or Older	94.21	83.68	83.23	91.48	94.12	84.18	80.82	92.35
Marital Status								
Unwed	16.72	66.73	35.43	33.71	21.23	69.25	45.39	33.77
Married	83.28	33.27	64.57	66.29	78.77	30.75	54.61	66.23
Parity								
First Birth	42.47	37.53	38.68	35.66	42.11	39.04	42.19	33.96
Low	46.38	38.25	38.82	39.85	46.81	38.38	38.23	44.20
High	11.15	24.22	22.50	24.49	11.08	22.58	19.58	21.84
Sex								
Male	51.31	50.72	51.01	50.97	51.27	50.74	51.00	50.91
Female	48.69	49.28	48.99	49.03	48.73	49.26	49.00	49.09
Education								
<12 years	15.01	29.84	40.92	73.57	12.86	27.48	37.70	68.94
12 years	39.33	43.20	39.67	17.96	33.13	39.25	38.09	21.25
13+ years	45.66	26.96	19.41	8.47	54.01	33.27	24.21	9.81
Previous Loss								
Yes	24.94	26.63	18.67	13.29	26.33	28.81	18.56	14.16
No	75.06	73.37	81.33	86.71	73.67	71.19	81.44	85.84
Plurality								
Single	97.67	97.37	98.19	98.27	97.05	97.06	98.08	98.20
Plural	2.33	2.63	1.81	1.73	2.95	2.94	1.92	1.80
Medical Risks								
Yes	20.48	24.17	18.61	12.80	27.00	30.71	21.45	17.61
No	79.52	75.83	81.39	87.20	73.00	69.29	78.55	82.39
Labor/Delivery Comp.								
Yes	32.05	33.57	27.15	23.41	33.97	34.69	25.17	25.49
No	67.95	66.43	72.85	76.59	66.03	65.31	74.83	74.51
Prenatal Care								
Inadequate	10.81	29.45	25.98	37.80	7.83	20.42	16.84	22.80
Intermediate	15.10	14.91	16.62	20.66	13.75	13.63	14.47	17.33
Adequate	48.14	30.24	35.17	27.30	47.53	34.86	39.24	36.54
Adequate Plus	25.95	25.40	22.23	14.24	30.89	31.09	29.45	23.33
Smoking								
Yes	17.16	12.91	4.67	0.79	13.97	8.78	3.32	0.54
No	64.90	70.17	53.10	31.86	70.30	80.70	56.69	50.64
Missing	17.94	16.92	42.23	67.35	15.73	10.52	39.99	48.82
Weight Gain								
<15 lbs	4.84	9.29	4.42	2.67	6.55	11.25	5.54	5.13
15-40 lbs	64.62	57.28	37.26	20.18	65.02	59.75	39.54	31.92
40+ lbs	13.51	11.39	7.80	2.73	16.61	14.92	9.60	4.62
Missing	17.03	22.04	50.52	74.41	11.81	14.08	45.32	58.33
Gestational Age								
Preterm (<37 weeks)	8.25	18.21	10.98	9.79	9.45	16.90	11.33	9.98
Term (≥ 37 weeks)	91.75	81.79	89.02	90.21	90.55	83.10	88.67	90.02
Birth Weight								
Low (<2500 grams)	5.38	12.70	5.99	4.78	6.11	12.42	6.45	5.16
Normal (≥2500 grams)	94.62	87.30	94.01	95.22	93.89	87.58	93.55	94.84
N	**6813486**	**1669571**	**360703**	**604275**	**9011429**	**2112566**	**701901**	**1090057**

SOURCE: See Table 1.

Table 3. Models of Infant Mortality for All Birth Weight Infants by Time Period: United States, 1989-1991 and 1995-1998

	Infant Mortality [Survive]															
	Model 1†				Model 2†				Model 3†				Model 4†			
	Con.	SIDS	RDS	Others	Con.	SIDS	RDS	Others	Con.	SIDS	RDS	Others	Con.	SIDS	RDS	Others
Ethnic Group								**1989-**	**1991**							
NHB	1.151***	1.923***	2.373***	2.742***	0.929***	1.006	1.565***	1.789***	0.858***	1.139***	1.319***	1.621***	0.627***	1.048**	0.820***	1.146***
MX-US	0.997	1.103**	1.045	1.187***	0.873***	0.652***	0.834**	0.908***	0.847***	0.758***	0.820**	0.883***	0.837***	0.756***	0.828**	0.882***
MX-IM.	1.130***	0.499***	0.812***	0.959***	0.986	0.247***	0.698***	0.705***	0.993	0.299***	0.734***	0.708***	1.070*	0.304***	0.847**	0.772***
Intercept	-6.38***	-6.76***	-7.63***	-5.94***	-6.97***	-8.10***	-8.88***	-6.81***	-7.44***	-8.26***	-10.06***	-7.55***	-7.48***	-8.23***	-12.33***	-7.56***
-2LL		948260 ***				926717 ***				887671 ***				800522 ***		
Ethnic Group								**1995-**	**1998**							
NHB	1.216***	2.133***	2.572***	2.406***	1.049**	1.091***	1.896***	1.732***	0.974	1.309***	1.552***	1.600***	0.723***	1.216***	1.036	1.198***
MX-US	1.095***	1.006	1.170**	1.187***	0.958	0.571***	0.950	0.916***	0.966	0.700***	1.022	0.975	0.936**	0.698***	1.008	0.957*
MX-IM.	1.127***	0.401***	0.753***	0.874***	0.949*	0.207***	0.693***	0.672***	0.933**	0.269***	0.704***	0.703***	0.993	0.273***	0.797***	0.753***
Intercept	-6.60***	-7.30***	-8.45***	-6.03***	-7.23***	-8.69***	-9.63***	-6.90***	-7.68***	-8.80***	-10.71***	-7.58***	-7.78***	-8.79***	-12.74***	-7.65***
-2LL		1012352 ***				990299 ***				951001 ***				803028 ***		

Source: See Table 1.

Note: Brackets [ ] indicate reference groups. *** $p \leq 0.01$. ** $p \leq 0.05$. * $p \leq 0.10$.

† Model Covariates: Model 1 is the bivriate relationship. Model 2 controls place of birth, maternal age, marital status, parity, sex of infant, education, and prenatal care. Model 3 includes the controls from Model 2, plus previous loss, plurality, medical risks, labor and delivery complications, smoking and weight gain. Model 4 includes the controls from model 3, plus gestational age and birth weight.

Table 4. Models of Infant Mortality for Low Weight Infants by Time Period: United States, 1989-1991 and 1995-1998

	Infant Mortality [Survive]											
	Model 1†				Model 2†				Model 3†			
	Con.	SIDS	RDS	Others	Con.	SIDS	RDS	Others	Con.	SIDS	RDS	Others
Ethnic Group						**1989-**	**1991**					
NHB	0.531***	1.415***	1.017	1.435***	0.521***	0.854***	0.934**	1.276***	0.453***	0.944	0.838***	1.137***
MX-US	0.840***	1.167	0.929	1.058	0.842***	0.780**	0.892	1.012	0.722***	0.867	0.819**	0.895***
MX-IM.	1.257***	0.545***	0.888*	1.071*	1.334***	0.339***	0.944	1.084**	1.165***	0.379***	0.857**	0.947
Intercept	-4.15***	-5.67***	-4.69***	-3.53***	-4.37***	-6.99***	-5.39***	-4.03***	-4.57***	-7.15***	-6.24***	-4.80***
-2LL		352481 ***				349006 ***				336866 ***		
Ethnic Group						**1995-**	**1998**					
NHB	0.683***	1.663***	1.286***	1.450***	0.704***	0.964	1.216***	1.381***	0.604***	1.105*	1.039	1.218***
MX-US	1.020	0.919	1.082	1.139***	1.019	0.592***	1.030	1.076**	0.870***	0.696***	0.980	0.994
MX-IM.	1.351***	0.348***	0.864*	1.020	1.311***	0.218***	0.914	1.000	1.084*	0.272***	0.791***	0.868***
Intercept	-4.43***	-6.22***	-5.66***	-3.76***	-4.60***	-7.55***	-6.24***	-4.24***	-4.76***	-7.75***	-7.08***	-4.97***
-2LL		392959 ***				389812 ***				376061 ***		

Source: See Table 1.

Note: Brackets [ ] indicate reference groups. *** $p \leq 0.01$. ** $p \leq 0.05$. * $p \leq 0.10$.

† Model Covariates: Model 1 is the bivariate relationhip. Model 2 controls place of birth, maternal age, marital status, parity, sex of infant, education, and prennatal care. Model 3 includes the controls from Model 2, plus plurality, medical risks, labor and delivery complications, smoking, and weight gain.

Table 5. Models of Infant Mortality for All Birth Weight Infants with Time Period as a Covariate: United States, 1989-1991, and 1995-1998

	Infant Mortality [Survive]															
	Model 1†				Model 2†				Model 3†				Model 4†			
	Con.	SIDS	RDS	Others	Con.	SIDS	RDS	Others	Con.	SIDS	RDS	Others	Con.	SIDS	RDS	Others
Ethnic Group [NHW]																
NHB	1.187***	2.024***	2.465***	2.564***	1.184***	2.013***	2.445***	2.560***	1.151***	1.923***	2.374***	2.742***	0.631***	1.025	0.817***	1.180***
MX-US	1.036	0.999	1.021	1.177***	1.055**	1.050	1.100*	1.193***	0.997	1.104**	1.045	1.187***	0.818***	0.756***	0.829**	0.889***
MX-IM.	1.110***	0.431***	0.738***	0.900***	1.127***	0.448***	0.784***	0.909***	1.130***	0.499***	0.812***	0.959	1.022	0.308***	0.850**	0.776***
Year [1989-91]																
1995-98					0.821***	0.594***	0.453***	0.865***	0.810***	0.581***	0.441***	0.910***	0.755***	0.611***	0.369***	0.833***
Interaction																
1995-98*NHB									1.056**	1.109***	1.084*	0.877***	1.140***	1.209***	1.281***	0.991
1995-98*MX-US									1.098*	0.911	1.120	1.000	1.166***	0.923	1.223*	1.068*
1995-98*MX-IM.									0.997	0.804***	0.927	0.911***	1.013	0.870*	0.935	0.965
Intercept	-6.50***	-7.03***	-8.01***	-5.99***	-6.39***	-6.77***	-7.64***	-5.91***	-6.38***	-6.76***	-7.63***	-5.94***	-7.49***	-8.26***	-12.10***	-7.51***
-2LL		1964270 ***				1960715 ***				1960612 ***				1663973 ***		

Source: See Table 1.

Note: Brackets [ ] indicate reference groups. *** $p \leq 0.01$. ** $p \leq 0.05$. * $p \leq 0.10$.

† Model Covariates: Model 1 is the bivariate relationship. Model 2 adds time period. Model 3 adds the race x time period interaction. Model 4 controls ALL risk factors, and includes the race x time period interaction.

Table 6. Models of Infant Mortality for Low Weight Infants with Time Period as a Covariate: United States, 1989-1991, and 1995-1998

	Infant Mortality [Survive]															
	Model 1†				Model 2†				Model 3†				Model 4†			
	Con.	SIDS	RDS	Others	Con.	SIDS	RDS	Others	Con.	SIDS	RDS	Others	Con.	SIDS	RDS	Others
Ethnic Group [NHW]																
NHB	0.613***	1.560***	1.156***	1.459***	0.607	1.524***	1.109***	1.443***	0.531***	1.415***	1.017	1.435***	0.466***	0.917*	0.836***	1.176***
MX-US	0.930**	0.986	0.923	1.089***	0.946	1.027	0.992	1.108***	0.840***	1.167	0.929	1.058	0.709***	0.871	0.820**	0.897**
MX-IM.	1.290***	0.424***	0.824***	1.025	1.308***	0.438***	0.872***	1.040*	1.257***	0.545***	0.886*	1.071*	1.098*	0.394***	0.862**	0.944
Year [1989-91]																
1995-98					0.805***	0.607***	0.413***	0.799***	0.753***	0.578***	0.380***	0.795***	0.766***	0.637***	0.368***	0.786***
Interaction																
1995-98*NHB									1.288***	1.175**	1.264***	1.011	1.267***	1.240***	1.255***	1.081
1995-98*MX-US									1.215**	0.787	1.164	1.077	1.242***	0.793	1.197	1.104*
1995-98*MX-IM.									1.075	0.634**	0.974	0.953	1.034	0.657*	0.915	0.923*
Intercept	-4.31***	-5.97***	-5.16***	-3.67***	-4.19***	-5.70***	-4.73***	-3.60***	-4.15***	-5.67***	-3.53***	-5.94***	-4.53***	-7.22***	-6.18***	-4.77***
-2LL		748103 ***				745530 ***				745440 ***				713195 ***		

Source: See Table 1.

Note: Brackets [ ] indicate reference groups. *** $p \leq 0.01$. ** $p \leq 0.05$. * $p \leq 0.10$.

† Model Covariates: Model 1 is the bivariate relationship. Model 2 adds time period. Model 3 adds the race x time period interaction. Model 4 controls ALL risk factors (except birth weight and gestational age) plus the race x time period interaction.

Table 7. Race/Ethnic Differences (Second Differences) in the Reduction in Approximations of Rates per 1,000 Live Births: Low Weight Births Only

	Difference in Estimate NHB - NHW				Difference in Estimate MX-US. - NHW				Difference in Estimate MX-IM. - NHW			
	Bivariate Model		Full Model		Bivariate Model		Full Model		Bivariate Model		Full Model	
Year	NHB - NHW	z	NHB - NHW	z	MX-US. - NHW	z	MX-US. - NHW	z	MX-IM. - NHW	z	MX-IM. - NHW	z
Infant Mortality - Congenital Anomalies												
1989	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.
1990	-1.414	-1.89	-0.923	-1.04	0.835	0.40	0.352	0.19	1.580	0.69	0.529	0.24
1991	-2.005	-2.75	-1.265	-1.47	-0.701	-0.33	-0.580	-0.32	-0.295	-0.13	-0.590	-0.26
1995	-4.304	-5.95	-2.819	-3.34	-1.369	-0.70	-1.494	-0.86	0.983	0.46	-0.513	-0.25
1996	-5.075	-7.05	-3.297	-3.95	-3.706	-1.87	-2.816	-1.57	0.876	0.42	-0.619	-0.30
1997	-4.783	-6.73	-3.037	-3.70	-3.207	-1.65	-2.557	-1.46	-1.435	-0.68	-1.995	-0.93
1998	-5.120	-7.19	-3.233	-3.90	-2.479	-1.29	-2.137	-1.24	1.149	0.56	-0.646	-0.32
Infant Mortality - Sudden Infant Death Syndrome												
1989	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.
1990	0.199	0.43	0.044	0.23	-0.051	-0.05	-0.022	-0.08	-0.231	-0.35	-0.035	-0.14
1991	-0.322	-0.70	-0.047	-0.24	-0.291	-0.26	-0.052	-0.19	-1.202	-1.72	-0.183	-0.64
1995	0.008	0.02	0.001	0.01	0.504	0.52	-0.028	-0.12	-0.689	-1.21	-0.100	-0.51
1996	0.306	0.72	0.069	0.40	0.691	0.73	-0.032	-0.14	-0.733	-1.35	-0.111	-0.60
1997	-0.005	-0.01	-0.006	-0.04	0.501	0.53	-0.044	-0.20	-1.050	-1.86	-0.163	-0.81
1998	-0.034	-0.08	-0.014	-0.08	0.731	0.79	-0.035	-0.16	-0.690	-1.32	-0.098	-0.56
Infant Mortality - Respiratory Distress Syndrome												
1989	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.
1990	-1.252	-1.77	-0.177	-0.49	-1.089	-0.64	-0.316	-0.54	1.578	0.96	0.042	0.07
1991	-1.807	-2.64	-0.262	-0.75	-2.814	-1.63	-0.629	-1.07	2.551	1.65	0.111	0.19
1995	-1.884	-2.98	-0.106	-0.34	-1.463	-1.00	-0.595	-1.23	1.657	1.15	-0.277	-0.52
1996	-2.076	-3.31	-0.140	-0.46	-2.159	-1.48	-0.734	-1.51	1.621	1.14	-0.329	-0.62
1997	-1.908	-3.09	-0.066	-0.22	-3.087	-2.10	-0.908	-1.84	0.911	0.64	-0.426	-0.80
1998	-1.643	-2.68	-0.010	-0.03	-2.216	-1.53	-0.768	-1.59	0.313	0.22	-0.491	-0.91
Infant Mortality - All Other Causes												
1989	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.
1990	1.086	0.78	0.352	0.42	-0.847	-0.26	-0.326	-0.25	0.152	0.05	0.173	0.13
1991	0.672	0.49	0.263	0.32	-1.813	-0.56	-0.437	-0.34	-0.684	-0.24	0.121	0.09
1995	2.925	2.18	1.396	1.82	-3.165	-1.06	-0.989	-0.80	-1.429	-0.53	-0.077	-0.06
1996	2.472	1.84	1.234	1.61	-3.520	-1.19	-1.093	-0.90	2.534	0.97	0.723	0.63
1997	4.226	3.19	1.941	2.58	-1.908	-0.66	-0.801	-0.67	3.009	1.16	0.881	0.77
1998	1.878	1.41	1.146	1.50	-1.477	-0.52	-0.768	-0.65	1.648	0.63	0.555	0.48

Source: NCHS Linked Birth and Infant Death Files, 1989-1991 and 1995-1998. Note: $z = 1.96$ at $p \leq 0.05$; $z = 2.58$ at $p \leq 0.01$ (two-tailed tests).