RACE/ETHNIC DIFFERENCES IN INFANT MORTALITY FOR LEADING CAUSES OF DEATH THAT HAD NO REDUCTION IN RATES *

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ABSTRACT

The core research objective is to determine whether a substantial expansion of the race/ethnic gap in infant mortality that occurred for the three specific causes of death for which major reductions in cause-specific IMRs were achieved also occurred in the case of the other two leading causes for which no (or very small) declines in infant death were observed in the 1990s. There were no notable advances in perinatal care and technology specific to the two specific causes on which we focus, short gestation and unspecified low birth weight (SG/LBW) and maternal complications. But, in general, perinatal care has shown more or less continuous incremental improvements over time, and an important question is how race/ethnic minorities fared, as compared to non-Hispanic whites, in regard to infant mortality from the latter two causes. Using data linked birth/infant death cohort files for 1989-91 and 1995-98, we show that for infants born to non-Hispanic black and Mexican American women, the mortality gap tended to widen in both relative and absolute terms, while infants of Mexican immigrants typically recorded changes that made for a higher degree of survivorship and a widening of their already extent advantage as compared to non-Hispanic whites.

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BACKGROUND

In recent years, enormous strides in perinatal health care and technology have resulted in resuscitation of infants that previously would have been stillborn (Hollander 1995; Sowards 1997), and presumably a more than offsetting capacity to preserve the life of these high-risk newborns—given the overall decline in infant mortality rates. 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), the continuing relative disparities in infant mortality between the non-Hispanic white (NHW) majority and non-Hispanic black (NHB) populations are of growing concern to scholars and policy makers alike (Frisbie et al. 2004; Gortmaker and Wise 1997; Wise 2003). In fact, 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 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). It is also disturbing that the most recent publicly available data show that there was a nationwide increase in the infant mortality rate (IMR) between 2001 and 2002—the first such increase since 1957-58 (Kochanek and Martin 2004).

One likely (and rarely exploited) place to search for an explanation for these trends might be found in an examination of cause-specific infant mortality. The five leading causes of infant death throughout the 1990s into the first decade of the 21st century were congenital anomalies, disorders of short gestation and unspecified low birth weight (SG/LBW), sudden infant death syndrome (SIDS), respiratory distress syndrome (RDS), and maternal complications. In fact, recent research (using

nationwide data from the NCHS Linked Birth/Infant Death Files) on race/ethnic differentials in cause-specific infant mortality among infants born to NHW, NHB, Mexican American, and Mexican immigrant mothers leads to even greater concern. For three of the five leading causes of infant death in which had large declines in mortality were recorded in the past decade (congenital anomalies, sudden infant death syndrome [SIDS], and respiratory distress syndrome (RDS)], it is disturbing that the relative disparity in risk of infant death between the NHW and NHB populations increased for all three causes (Frisbie et al. 2004; Frisbie et al. 2005). The relative gap also increased between the NHW majority population and infants born to Mexican American women for two of these three causes. Furthermore, absolute disparities also increased between the NHW majority population and the two minority groups. Interestingly, these patterns were not observed in comparisons of NHW infants and infants born of Mexican immigrants (Frisbie et al. 2004; Frisbie et al. 2005).

Major advances in perinatal care and technology were accomplished in regard to each of these three causes in the late 1980s and early-to-mid-1990s. Major declines in infant mortality from RDS have been attributed to pulmonary surfactant therapy after this intervention was approved for general use by the Food and Drug Administration (FDA) in August 1989 (Ferrara et al. 1994; Halliday 1997; Hamvas et al. 1996; Malloy and Freeman 2000; Ranganathan et al. 2000). 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, the SIDS rate dropped substantially in the U.S. between 1989 and 1997 (Gibson et al. 2000; Pollack and Frohna 2001, 2002; Willinger et al. 1998). Finally, a number of remarkable medical innovations have also played a key 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 in the fetus, selective termination of pregnancies when lethal anomalies are detected, and intensive care interventions designed to preserve the life of infants born with congenital malformations (Lee et al. 2001).

Lack of Progress in Reducing Infant Mortality from Two Leading Causes

Conversely, little or no progress has been made in regard to the other two leading causes of death in the U.S. The infant mortality rate (IMR) for other two leading causes, SG/LBW and maternal complications, remained virtually unchanged over the 1900s.

Short Gestation and Unspecified Low Birth Weight (SG/LBW*)*

Disorders relating to short gestation and unspecified low birth weight which, for ease of exposition, we refer to as SG/LBW causes, were by the mid-1990s, second only to congenital anomalies as a leading cause of infant death. In 1980, the SG/LBW rate ranked fourth behind congenital anomalies, SIDS, and RDS rates in descending order (U.S. Census Bureau 2001). Moreover, SG/LBW is currently the number one cause of death among black infants, followed by congenital anomalies (Mathews et al. 2002). Creasy and Merkatz noted that spontaneous preterm delivery was "the most significant problem facing clinicians as we enter the 1990s" (1990:25), a conclusion that now seems even more relevant inasmuch as rates of adverse birth outcomes increased during the 1990s for the general population and for all large race/ethnic groups, except blacks (Demissie et al 2001; Frisbie and Song 2003). One partial explanation 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).

Spontaneous preterm birth occurs for reasons not completely understood, and interventions designed to prevent preterm labor have, in the past, not been particularly successful (Moore and Freda 1998). Pharmacological intervention, e.g., the administration of a tocolytic agent to arrest

uterine contractions, may delay, but does not prevent, preterm labor (Viamontes 1996). Also, while "intravenous hydration is a commonly used first clinical effort to reduce preterm labor contractions," as late as the mid-1990s, there was "no published evidence that pregnancies have been prolonged through use of hydration" (Freda and DeVore 1996: 385). It has been suggested that vaginal and intrauterine infections increase the risk of preterm labor so that antimicrobial therapy may act as a preventative (Goldenberg et al. 1996; Hauth et al. 1995). The results of recent clinical trials that assessed the effects of a form of progesterone (commonly referred to as 17P) make for some optimism. Although this particular intervention was employed at least two decades ago (Yemini et al. 1885), more recent work indicates that administration of 17P, either by weekly injections or daily vaginal suppository, lowers the likelihood of spontaneous preterm delivery (da Fonseca et al. 2003; Meis et al. 2003).

Maternal Complications: Maternal complications include a very wide range of conditions such as incompetent cervix, premature rupture of membranes, ectopic pregnancies, breech or other malpresentations, death of mother, etc. Few publications dealing with the general category of maternal complications that were comparative by race/ethnicity were found in our search of Popline and Medline. We do know, however, that this condition was the fifth leading cause of death from 1980 to the late 1990s (U.S. Census Bureau 2001: Table 103). We also know that, currently, the black infant mortality rate from maternal complications is two and one-half times that of whites, while the rates for the Mexican origin population are between 30% and 40% lower than the white rate (Mathews et al. 2001; Muhuri et al. 2004). Finally, maternal complications registered an overall rate increase among singleton births during the last decade for all groups except American Indians (Muhuri et al. 2004).

RESEARCH OBJECTIVES

The core research objective is to determine whether a substantial expansion of the race/ethnic gap in infant mortality that occurred for the three specific causes of death for which major reductions in cause-specific IMRs were achieved also occurred in the case of the other two leading causes for which no (or very small) declines in infant death were observed in the 1990s. The present research follows the approach of two previous studies (Frisbie et al. 2004; Frisbie et al. 2005). However, in this paper the focus is on the other two leading causes of infant death, short gestation and unspecified low birth weight (SG/LBW) and maternal complications, for which little or no reduction in IMRs occurred as shown in the following tabulation.

IMRs* for 5 LEADING CAUSES OF INFANT DEATH

	YEAR	
Causes of Infant Death	**1990**	**1998**
Congenital Anomalies	9.2	7.2
SIDS	1.3	0.7
RDS	0.7	0.3
SG/LBW	1.0	1.0
Maternal Complications	0.4	0.3

SOURCE: Statistical Abstract of the U.S. 2001, Table 103. Per 1000 live births.

As these values demonstrate, the IMR for congenital anomalies was substantially reduced, and the IMRs for SID and RDS were essentially cut in half. For the two causes of infant death on which we now concentrate attention, reductions were either very small or nonexistent.

The contrast is between infants born to non-Hispanic white (NHW), non-Hispanic black (NHB), U.S.-born Mexican-American, and Mexican immigrant women from the 1989-91 and 1995-98 cohorts. The time frame is important inasmuch as it has been 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).

Specifically, we (1) document race/ethnic differentials in four pregnancy outcomes: death due to SG/LBW, death due to maternal complications, deaths due to all other causes, and survival (the reference category); (2) model the extent to which social risk factors are associated with relative disparities in race/ethnic cause-specific infant mortality. That is, the aim is to determine if, and why, the direction and magnitude of change by specific cause is different for different race/ethnic populations; and (3) model the extent to which social risk factors are associated with absolute disparities in cause-specific infant mortality across race/ethnic populations. Just as in Aim 2, we will explore whether, and why, the direction and magnitude of change is different for different race/ethnic groups.

In all analyses, the four race/ethnic groups will be contrasted. 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) [1] infant mortality, but very few studies have addressed changing differentials by specific cause of death across multiple race/ethnic groups (for a partial exception, see Muhuri et al. 2004).

CONCEPTUAL MODEL

General Conceptual Framework

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

[1] The term "Mexican Origin" is used here to refer to both U.S.-born Mexican Americans and Mexican immigrants.

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). Even more directly relevant, Gortmaker and Wise (1997) warn that greater race/ethnic 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 minority 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 minorities 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.

The results of the research on causes of death for which substantial decreases in the IMR were recorded following improvements in perinatal care and technology showed a widening majority-minority disparity, allowing the inference of, but of course not proving (Moffitt 2004), lesser accessibility by minorities to these improvements. In the case the other two leading causes for which there was little or no reduction in the cause-specific IMRs, one might hypothesize that changes over time in race/ethnic differentials would be comparatively small—inasmuch as, overall, there was virtual stasis in rates. On the other hand, if accessibility to health care is generally more limited for minorities, one could easily argue that majority/minority disparities in risk of infant death due to SG/LBW and maternal complications should widen also. In any event, since there was neither one prominent intervention (such as surfactant therapy in the case of RDS) nor a series of well-documented innovations (as in the case of congenital anomalies) that emerged in the case of

either SG/LBW or maternal complications, it is important to shed light on the issue of whether increasing majority-minority disparities occurred in these cases as well. [2]

Risk Factors Included in the Analysis

Basic demographic covariates for this analysis include maternal age, marital status, 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), though some authors ascribe the immigrant advantage to cultural differences (Cobas et al. 1996; Scribner 1996) and others to the return of Mexican immigrant women with their infants who may die outside the U.S. thereby introducing a downward bias (Palloni and Arias 2004). However, research has just emerged (Hummer et al. 2005) that sheds considerable doubt on the validity of the "data artifact" interpretation. Our aims do not include assessment of the applicability of these interpretations.

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:

[2] This is not to say that no advances occurred that improved the survivability chances of infants at risk due to the latter two conditions. Obviously, there has been a consistent upgrading of the ability to preserve the life of high-risk newborns as can easily be seen by comparing the facilities and procedures that were available at the beginning of the 1990s with those available by the end of the decade, particularly at tertiary care faculties with well equipped Neonatal Intensive Care Units (NICUs).

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 (included referrals to service agencies) that is 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.

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 3 to 4 million 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). Note that no linked birth/infant death cohort files were produced by NCHS for 1992-1994. Causes of death codes are consistent over the two time periods; the 9th Revision of the International Classification of Diseases (ICD-9) is used throughout.

Methods

Our analysis closely follows the approach found in recent work by Frisbie et al. (2004; 2005). The methodological strategy consists of multivariate modeling of specific causes of death

between the 1989-91 and 1995-98 and includes an examination of both relative and absolute differentials over time.

Although studies of infant mortality are often limited to singleton births, our core data set includes plural births, as well as singletons. Recent studies 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 (< 37 weeks) and low weight births (< 2500 grams) and a rather substantial proportion of infant deaths. Moreover, the plural birth rate has notably increased over the past decade (Blondel et al. 2002). Finally, in almost every comparison, companion tables were generated showing the results for infants born at all weights and for high-risk infants—i.e., those born at low weights (< 2500 grams).

We exclude infants weighing ≤ 500 grams at birth for the usual reasons. Such cases are often misclassified stillbirths or reflect errors in recording of birth weight. Missing data was in general not a problem, except in the case of maternal smoking and weight gains. A problem arises with these two variables because items asking about these characteristics are not included on the birth certificates of some states, notably California. Where information was missing on smoking and weight gain, we adopted the conventional strategy of assigning a missing category for these covariates (Frisbie et al. 1998; Singh and Yu 1996).

We examine race/ethnic differentials in four infant outcomes: death due to SG/LBW, death due to maternal complications, deaths due to all other causes, and survival (the reference category). In addition to descriptive tabulations, we model outcomes using multinomial logistic regression, with results reported as odds ratios. We progressively adjust for a large number of risk factors to yield a sequence of models. The operationalization and distribution of risk factors by race/ethnicity appear below as Table 1. In a number of instances, the final model of a set includes birth weight

and gestational age as covariates. Some readers may view the latter type of model as involving excessive overlap with the LBW/SG outcome. We do not believe this to be the case for reasons discussed immediately below. However, those unconvinced by our reasoning, may simply wish to ignore "final models" and, instead focus on the immediately preceding model that, in every case, includes all controls except for LBW and short gestation. However, a strong rationale for the inclusion of birth weight and gestational age as covariates in models of causes of infant mortality can be made. First, the inclusion of birth weight and gestational age is consistent with recent research (e.g., Muhuri et al. 2004). These birth outcomes are the strongest predictors of infant mortality in general and also mediate the effects of a wide range of other risk factors (Cramer 1987; Hummer et al. 1999; Kline et al. 1989). Although we know, *a priori*, that the connection between adverse birth outcomes and death due to SG/LBW causes will be very strong, it is neither redundant, nor illogical, to include adverse birth outcomes as predictors of infant mortality risk from SG/LBW causes. Clearly, a large and growing proportion of low weight and preterm infants survive. Moreover, given the tremendous advances recently made in treatment of such high-risk infants, as epitomized by modern NICUs, it is reasonable to hypothesize that the relationship will be weaker in later time periods. On the other hand, in light of the sort of social inequalities described in earlier sections, it may be more reasonable to expect that race/ethnic disparities in both relative and absolute infant mortality to increase. Finally, as will be demonstrated below, even though rates of LBW and preterm births declined slightly among blacks and rose slightly among whites in the 1990s, the ratio of black to white rates of death from this cause remains high.

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 Mexican origin infants than among black infants. Further, infants born to Mexican immigrant women have lower IMRs than the rates for either white infants or for infants born to Mexican American mothers. (Frisbie et al. 2004; Hummer et al. 1999; Scribner 1996).

As delineated in the discussion of specific aims, are goal is 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). We have modeled 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 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 specific causes of infant death over time by pooling over the individual data over 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. We then fit two multinomial logistic regression models for each race/ethnic group. 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, as shown by comparing our estimates to the empirical IMRs actually observed (Frisbie et al. 2004). To assess change over time, we evaluate estimated yearly differences in the odds, interpreted as approximate rates and changes in rates. Significance tests of differences are carried out based on large sample properties of functions of maximum likelihood estimators. The estimated odds from a multinomial logit model have a normal sampling distribution in large samples, and the variance of the odds can be obtained using the delta method (Rao 1973). The standard errors of the yearly odds are then used to compute the standard errors of the difference between odds. (For a full discussion of the method, see Frisbie et al. 2004).

We present results only for second differences and only for low weight births. This is necessary to conserve space and because of the extremely high mortality observed among these high-risk births. That is, models begin with the odds estimates of rates for each race/ethnic groups and then evaluate how these differences changed from year to year. Then, we analyze second differences in which the NHW change is compared with the change for each minority groups separately for each specific cause of death. It must be borne in mind that the changes for non-Hispanic whites were subtracted from each race/ethnic group change, so that an estimate with a

positive sign represents a greater improvement for minority groups and a negative sign represents a greater improvement for non-Hispanic whites.

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).

DESCRIPTIVE RESULTS

Risk Factor Distributions

The distribution of risk factors by race/ethnicity and time period appears in Table 1. Inasmuch as the results are consistent with all known previous research showing (for the most part) a NHW advantage relative to minorities, we discuss only selected portions of the distribution that highlight race/ethnic differences. Births to unwed and teenage mothers are substantially higher among NHBs and Mexican Americans. However, it is notable that the percentage out-of-wedlock births increased by a much greater amount than for Mexican Americans than for any other group, although NHB women continue to have the highest proportion (over two-thirds of mothers) in this category. Women of Mexican origin are the most disadvantaged in terms of years of education completed; approximately 41% and over 73%% of Mexican American and Mexican immigrant women, respectively, had less than a high school education in 1989-91. By 1995-98, approximately 38% and 70% of these two Mexican origin groups still lacked a high school diploma. In regard to prenatal care (PNC), the percentage of minority group women receiving adequate care increased (especially among Mexican immigrants) between 1989-91 and 1995-98, but in every instance, they

continue to trail NHW women. [3] The relative number of mothers presenting with medical risks increased by 5% to 7% for every race/ethnic group, but immigrant women continue to be the most advantaged group in this regard. The proportion of women who smoked declined for all groups, but in both time periods, NHW women were more likely to smoke, while this behavior was virtually nonexistent among Mexican immigrant women. NHB women are far more likely to given birth to LBW and/or premature infants. It interesting to note, however, that as reported in other research based on vital statistics (Demissie et al. 2001; Frisbie and Song 2003), there was a small decrease in these adverse outcomes among NHB women, while all the other race/ethnic groups recorded small increases

--Table 1 About Here--

Changes in Cause-Specific IMRs by Race/Ethnicity

Births at All Weights

Table 2a presents IMRs for infants born at all weights (except those weighing ≤ 500 grams). Percentages in the IMRs for the SG/LBW cause category increased in absolute terms for NHW infants (8.3%) and Mexican Americans infants (approximately 4%). There was no change for Mexican immigrant infants, while the SG/LBW IMR declined by 14.0% for NHB infants. Turning to relative comparisons (rate ratios in the final six columns of Table 2a), all minority groups improved slightly relative to NHWs. In the case of infant death attributable to maternal complications, all groups recorded declines, except Mexican American infants among whom there was a percentage increase of 11%. Mexican immigrants had by far the largest absolute decline (-28.6%). Rate ratios show a very minor narrowing of the relative gap for babies born to NHB women, while there was an

[3] Note that the measure of PNC is the Kotelchuck APNCU index which is based on number and timing of visits for care. The "adequate plus" category denotes women who record more visits than the number recommended by the American College of Gynecologists, and is often viewed as evidence of negative selectivity (regarding problem pregnancies). Also, the Kleinman-Kessel (1987) index is used to take into account the interaction between maternal age and parity. Our diagnostics show no collinearity between the Klineman-Kessel measure and maternal age.

increase in the NHW/Mexican American rate ratio from 0.75 in 1989-91 to 1.0 in 1995-98. The decrease for immigrants was small, but note that the maternal complications IMR for infants of Mexican immigrants is only about half that of the NHW counterparts. For all other causes, there is considerable consistency in the patterns. All race/ethnic groups had proportional declines of 23% or more, and the rate/ratios demonstrate that all the minorities improved slightly relative to NHWs.

--Table 2a About Here--

Low Weight Births

Among LBW infants, the story is somewhat different (Table 2b). In the SG/LBW category, every group showed an absolute percentage decline. Thus, in every relative (rate ratio) comparison, the relative advantage of NHW infants increased. In regard to rate/ratios, there were very small improvements for every group of minority infants. The percentage decline for LBW Mexican immigrant infants with respect to maternal complications was quite large (approximately 38%), just as was the case for infants born at all weights. In sharp contrast, the IMR for the same cause declined by only about 3%. Immigrant infants also registered a decline in the rate ratio, but the maternal complications advantage for other two race/ethnic groups eroded somewhat. Just as in Table 2a, percentage drop in infant mortality from other causes was notable, ranging from 25% for NHB and Mexican American infants to approximately 30% for infants born to NHWs and Mexican immigrants. The relative NHW survival advantage increased in every race/ethnic comparison.

--Table 2b About Here--

Multivariate Analyses

Tables 3a and 3b present models of mortality for births occurring in 1989-91 at all weights and at low weights, respectively. The effects of risk factors used as controls are quite similar to those estimated by previous research. Inasmuch as these effects are consistent as to direction over time, 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 and time period. However, we do show how the risk of infant death varies by these variables as different blocks of risk factors are sequentially controlled. The control variables for each model are listed in the notes at the bottom of the tables.

Births at All Weights

Bivariate relationships (Model 1, Table 3a) show that for births at all weights in 1989-91, the risk of death for NHB infants due to SG/LBW is over four times higher than that of NHW infants, and that the odds ratio for maternal complications is about twice as high compared to that of NHW infants. The situation is decidedly different for Mexican Origin infants during this time period. The babies of Mexican Americans and Mexican immigrants have odds ratios (ORs) below 1.0, or have statistically equivalent risks compared to NHWs for all cause of death categories, except two, in each of the first three models. The only exception to this survival advantage is for Mexican Americans where the risk is about 12% greater for all other causes.

In Models 2 and 3 of Table 3a, which add controls for sociodemographic, behavioral, and medical risk factors, the ORs for NHB infants are sharply reduced. Nevertheless, in Model 3 (which includes all covariates except birth weight and gestational age), the NHB risk remains twice that for NHWs for SG/LBW and about 30% greater for all other causes, while the risk from maternal complications is reduced to statistical parity with NHW infants. However in Model 4, which adds birth outcomes as a control, the NHB risk due to SG/LBW exceeds that of NHWs by only 19%. Two findings stand out in the analysis of the Mexican origin population. First, in almost every comparison in every model in 1989-91, both Mexican origin groups continue to have lower ORs than do NHWs. The only exception is in Model 4 (in which birth weight and gestational age are controlled) where infants of Mexican immigrants are at a small and non-significant disadvantage in the SG/LBW category.

--Table 3a About Here--

The bivariate relationships as seen in the second panel of Table 3a pertain to the period 1995-98. At this later time period, survivorship improved somewhat for NHB infants for each cause of death. The bivariate pattern for Mexican Origin infants is rather similar to that observed for 1989-91. In 1995-98, while there was some deterioration in the survival advantage for Mexican American infants compared to 1989-91, but the situation consistently improved for immigrant infants. In Models that adjust for covariates, the Mexican Origin population had a survival advantage, compared to NHWs, However, taking nativity into account, the Mexican American advantage, although still evident, tended to erode, while the opposite was true of infants of Mexican immigrant mothers in most comparisons.

Low Weight Births

Comparisons among LBW infants appear in Table 3b. [4] For infants in the 1989-91 cohort (Panel 1 of Table 3b), in every single comparison across models and in both time periods, the odds of NHB infant death among low weight infants are lower than the corresponding odds for infants born at all weights, and in several instances, a reversal is in direction of the relationship is observed. The lower rates for LBW infants, of course, results from the inherently relative nature of logistic regression, as all estimates are relative to the reference group. One might still be interested in why, when the analysis is restricted to low weight births, the NHB infant risk of death is so much closer to the NHW risk, than was the case in the analysis of births at all weights. At least two explanations apply to this question. It is possible that NHB infants have a superior return to risk than do NHW infants. Second, in the estimates based on births of all weights, NHBs have a severe compositional disadvantage due to the much larger proportion of LBW infants. In Table 3b, this compositional

[4] 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

disadvantage is controlled in that only LBW infants are included. The second explanation receives considerable support when one compares the odds ratios for NHB infants in Table 3b with the odds ratios for this group in Model 4 of Table 3a which controls for LBW by including it as a covariate.

The results for the Mexican Origin population are mixed when only LBW infants are considered (Table 3b). Perhaps the most notable finding is that in 1989-91 Panel 1 of Table 3b), the risk of infant death from each of the causes in every model is lower for Mexican Americans than for Mexican immigrants. This is the only instance in our analysis where the U.S.-born consistently fare better than their Mexican immigrant counterparts. Indeed, this is the opposite of what has been demonstrated in virtually all previous studies. One plausible interpretation is that, as other research has shown, immigrants are less likely to be aware of, and integrated into, the formal health care system than are their U.S.-born counterparts (Frisbie et al. 2001; LeClere et al. 1994; Thamer et al. 1997)—a situation that could be especially deleterious for high-risk (LBW) infants.

--Table 3b About Here--

The results for LBW infants in the 1995-98 cohorts (Panel 2, Table 3b) are fairly similar to those seen in Panel 1 of this table for Models 1 and 2, but a few notable differences can be observed. The survival advantage of Mexican American infant tends to reappear, but in 1995-98, this phenomenon does not emerge for one of the specific causes, via., maternal complication. Also, in Model 3, the risk of death continued to be higher for each cause of death in 1995-98 than it was in 1989-91for NHBs and Mexican Americans. But for immigrant infants, pattern was reversed. That is, in Model 3 of Panel 2 of Table 3b, the risk of death for babies of Mexican immigrants was lower in 1995-98. We have no explanation for this divergence in pattern, though increased access to public assistance to immigrants that occurred between the early and late 1990s may play a role here.

Interaction of Race/Ethnicity and Time Period

We also pooled the data across time and then conducted separate regressions in which an interaction term, race x time period, was included This approach uses the same information as that on which Tables 3a and 3b are based, so that general conclusions are identical. Hence, we do not present the results of the interaction model in tabular form. Nevertheless, pooling the data with time period as a covariate allows estimation of the magnitude and significance of a race/ethnicity x time period interaction term. The results from this exercise are remarkably consistent. In every comparison, except one, the odds ratios for the interaction term were below 1.0 and typically statistically significant. This indicates that that the risk of death from all three categories of causes of infant death, was reduced in 1995-98 compared to 1989-91, thereby allowing the inference that advances in perinatal care improved for all race/ethnic groups over time for causes whose IMRs remained constant. (Of course, access to these advances may well differ by race/ethnicity, which is consonant with our results so far.) In more substantive terms, the race x time period interaction either partially offset the higher mortality risk of race/ethnic groups or further increased their advantage of surviving. This finding is perfectly consistent for low weight births. The one minor exception occurs in Model 3 for births at all weights, (recall that Model 3 adjusts for all risk factors except birth weight and short gestation), where the odds ratio associated with deaths due to SG/LBW was positive (OR = 1.068), thereby indicating a very small deleterious offset.

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. <u>However, only the results of estimates of absolute second differences for high-risk (LBW) infants are presented because of space limitations, and because it is this growing population of high-risk infants that cause the greatest concern.</u> Results from both bivariate and full models are shown in Table 4. Note that

second differences compare the change in approximated rates for each minority with the change for the NHW majority population for each specific cause of death. Note also 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. Significance of differences is shown in the Z scores that accompany the rate estimates. (A Z value ≥ 1.96 is needed to achieve the .05 level of significance with a two-tailed test.)

Table 4 is organized in three row panels—one for each the specific causes of death on which we focus. We begin by discussing the findings for SG/LBW.

In the case of infant death due to SG/LBW, the bivariate model shows that NHB infants had a slight absolute gain in survivorship compared to NHWs, but none of the differences was significant. [5] The same pattern is observed in the fully adjusted models, and no difference even remotely approaches statistical significance. (Z ≥ 1.96 is needed to achieve statistical significance with a two-tailed test.) The slight NHB advantage might be taken as a minor piece of evidence of the maturity of black fetuses at lower weights and shorter gestations—as several authors argue is the case (Kline et al. 1989; Wilcox and Russell 1986, 1990). The findings for the Mexican Origin population are almost completely different as to direction of the changes. For infants of both Mexican American and Mexican immigrant mothers, the absolute decline in mortality due to SG/LBW was greater for NHW infants—as indicated by the negative signs for each annual change except the first (1989-91). This seems to us to be an important difference between Mexican Origin population and NHB high-risk infants—if for no other reason than the consistency of the pattern, but in neither the bivariate model nor the full model do the greater absolute increases in survivorship of NHW infants attain conventional levels of statistical significance.

--Table 4 About Here--

[5] It warrants reiteration that we are working with the entire population, not a sample, so tests of significance of less interest than would otherwise be the case.

By contrast, absolute changes in the approximated rates of death from maternal complications are much more consistent across race/ethnic groups. The bivariate analysis demonstrates that the improvement in rate changes for this cause of death are, in the vast majority of comparisons, in a direction that is beneficial for NHWs compared to each of the three minorities—although infants of Mexican immigrants fare somewhat better than do infants of the other two race/ethnic groups. In the case of the full model, the second differences uniformly favor NHW infants.

As would be expected of a residual category, for all other causes of death, the patterns are much more variable, yet the findings regarding all other causes may be among the most important in our consideration of absolute change. Beginning with the bivariate model, the absolute changes reflect a non-Hispanic white advantage in every comparison with non-Hispanic black and Mexican American infants. Further, in almost every instance, the change scores comparing NHWs and NHBs are significant, but the same is true of only the 1995 to 1996 change for Mexican Americans. On the other hand, infants of Mexican immigrants consistently show an advantageous pattern f change.

Notably, in models with full controls, all minority infants tend to fare worse with respect to all other causes of death, again as shown by the negative signs. The majority of the change estimates continue to be significant for NHBs in the full model, as do the disadvantages of Mexican American infants. Even in the case Mexican immigrants, the signs are reversed from positive (an immigrant advantage) to negative (a NHW advantage).

CONCLUSIONS

Our general research goal was to determine whether there was any substantial expansion of the race/ethnic gap in infant mortality in the case of two of the five leading specific causes of infant death in the U.S., viz., short gestation and unspecified low birth weight and maternal complications, for which there was essentially no decline in their cause-specific IMRs over the past decade. In the

case of the other three leading causes of infant mortality (congenital anomalies, SIDS, and RDS), notable innovations in perinatal care and technology occurred in the timeframe of interest that, in turn, led to quite major decreases in the rates of infant death. Unfortunately, both relative and absolute race/ethnic disparities increased for NHB and Mexican American infants for these three causes. It has been argued that this increase in health inequality was due in some large measure to the smaller degree of access of minority groups to advances in perinatal care and technology (Gortmaker and Wise 1997; Link and Phelan 1995, 2002; Wise 2003). Thus, the general substantive issue was whether the changes in race/ethnic differentials in regard to SG/LBW and maternal complications would be similar or dissimilar to the changes documented for the other three causes just mentioned.

At one level, the answer to this question is easy. Our descriptive comparison shows that, for infants born at all weights, in almost all cases, race/ethnic minorities had lower rate ratios (relative to NHWs), but the changes were quite small. Only in the case of Mexican American infant deaths from maternal complication did the rate ratio increase which is consistent with the finding that this group had the only increase in rate percentage. One discouraging finding here is that the IMR for NHB infants seems to stuck at a bit more than twice the NHW IMR, and even by the later time period (1995-98), the NHB rate ratio for deaths from short gestation and unspecified low birth weight (SG/LBW) showed that NHB IMR from that cause was more than three times the rate for NHWs. When we look at only low weight births, the findings are less consistent, but essentially what we find is that large absolute percentage increases translate into either small increments or decrements in the position of minority infants relative to NHW infants.

At a more specific and more analytic level, based on multinomial logistic regression models, the answer is more complicated. If one focuses on the model which adjusts for all covariates (Model 3 of Tables 3a and 3b, with NHW infants as the reference), one finds that for infants born at all

weights, the net NHB disparity (expressed as odds ratios) is higher for every cause of death, and that, more often than not, the NHB risk of infant death increased between 1989-91 and 1995-98. Infants of both Mexican American and Mexican immigrant women have a lower risk of death, relative to NHW infants at both periods of time. However, immigrant infants increased their survival advantage, the Mexican American advantage eroded over time for every specific cause of death. In Table 3b, which includes only LBW infants, NHBs have odds ratios lower than one for both maternal causes and all other causes. However, the net survival advantage became smaller for both NHBs and Mexican Americans over time. Further, while the NHB risk of infant death from SB/LBW in 1989-91 exceeded that of their non-Hispanic white counterparts by 21%, the NHB risk had become 32% greater by 1995-98. The Mexican American survival advantage also eroded among LBW infant. Only in the case of infants of immigrant women was the change in odds ratios in a direction beneficial for the minority group.

Finally, we used exponentiated logits corresponding to year provide micro-data based approximations of the observed annual mortality rates (rather than falling back on ecological or aggregate data) for high-risk births. We presented second differences that compare the change in approximated rates for each minority with the change for the NHW majority population for each specific cause of death. The results of this analysis results (Table 4) are rather discouraging. Based on these estimated rates, in only two comparisons were absolute changes in survival more advantageous for minority infants. First, the chances of survival with respect to SG/LBW improved for NHB infants in both the bivariate and full models. Second, immigrant infants gained a bit of ground on NHW infants as shown in the bivariate model for the residual all other cause category.

In summary, the three race/ethnic groups did not fare particularly well in their infant mortality risk compared to NHWs. Overall, the picture is one of growing disparity for NHB and Mexican American infants in both relative and absolute terms. Overall, the disadvantageous

changes for SG/LBW and maternal complications are not as dramatic as those found for congenital anomalies, SIDS, or RDS—leading causes of death for which large declines in infant mortality were achieved in the 1990's decade due to rather specific, well-documented and highly effective advances in perinatal health care and technology. Nevertheless, other less spectacular and more incremental improvements in infant health care occurred. However, none of the advances seems to have had a beneficial effect in lowering the overall IMRs for SG/LBW and maternal complications. The fact that, in general the majority/minority gap widened for each race/ethnic group, with the notable exception infants born to Mexican immigrants is an unwelcome finding. The question of why immigrant infants often improved their survivorship vis a vis NHWs remains a topic of debate and will no doubt continue to spawn additional research.

To close on a more positive note, in addressing the specific aims of estimating the association of a wide range of demographic, social, behavioral, and biomedical risk factors with race/ethnic differentials in infant mortality, we clearly show that when negative characteristics are controlled, race/ethnic disparity decreases substantially. Thus, interventions designed to improve the social and behavioral profiles of all race/ethnic groups through an equitable expenditure of resources is clearly an important means of reducing race/ethnic inequality in health and mortality.

Table 1. 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.**
Maternal Age								
Under 18	5.79	16.31	16.77	8.52	5.88	15.82	19.18	7.65
18 or Older	94.21	83.69	83.23	91.48	94.12	84.18	80.82	92.35
Marital Status								
Unwed	16.71	66.72	35.31	33.63	21.23	69.25	45.39	33.78
Married	83.29	33.28	64.69	66.37	78.77	30.75	54.61	66.22
Parity								
First Birth	46.37	38.24	38.83	39.85	42.81	38.38	38.23	44.20
Low	42.48	37.56	38.67	35.67	46.11	39.04	42.19	33.96
High	11.15	24.20	22.50	24.48	11.08	22.58	19.58	21.84
Sex								
Male	51.31	50.72	51.01	50.99	51.28	50.74	51.00	50.92
Female	48.69	49.28	48.99	49.01	48.72	49.26	49.00	49.08
Education								
<11 years	15.00	29.84	40.95	73.56	12.86	27.48	37.70	68.94
12 years	39.32	43.19	39.65	17.96	33.13	39.25	38.09	21.25
13+ years	45.68	26.97	19.40	8.48	54.01	33.27	24.21	9.81
Prenatal Care								
Inadequate	10.81	29.47	26.05	37.86	7.83	20.42	16.84	22.81
Intermediate	15.09	14.92	16.60	20.64	13.75	13.63	14.47	17.33
Adequate	48.12	30.20	35.12	27.26	47.53	34.86	39.24	36.53
Adequate Plus	25.98	25.41	22.23	14.24	30.89	31.09	29.45	23.33
Previous Loss								
Yes	24.95	26.65	18.65	13.27	26.32	28.82	18.56	14.16
No	75.05	73.35	81.35	86.73	73.68	71.18	81.44	85.84
Medical Risks								
Yes	20.61	24.28	18.85	12.92	27.02	30.73	21.46	17.62
No	79.39	75.72	81.15	87.08	72.98	69.27	78.54	82.38
Plurality								
Yes	2.33	2.63	1.81	1.74	2.95	2.94	1.92	1.80
No	97.67	97.37	98.19	98.26	97.05	97.06	98.08	98.20
Smoking								
Yes	17.15	12.89	4.68	0.80	13.98	8.79	3.32	0.54
No	64.89	70.02	53.40	32.20	68.30	80.69	56.71	50.66
Missing	17.96	17.09	41.92	67.00	17.72	10.52	39.97	48.80
Weight Gain								
<15 lbs	4.84	9.27	4.44	2.69	6.55	11.25	5.54	5.14
15-40 lbs	64.59	57.18	37.30	20.27	65.00	59.74	39.55	31.92
40+ lbs	13.51	11.38	7.82	2.75	16.61	14.92	9.60	4.62
Missing	17.06	22.17	50.44	74.29	11.84	14.09	45.31	58.32
Gestational Age								
Preterm (<37 weeks)	8.27	18.22	11.01	9.81	9.46	16.90	11.34	9.98
Term (≥ 37 weeks)	91.27	81.78	88.99	90.19	90.54	83.10	88.66	90.02
Birth Weight								
Low (<2500 grams	5.40	12.72	6.01	4.80	6.11	12.42	6.45	5.16
Normal (≥2500 grams	94.60	87.28	93.99	95.20	93.89	87.58	93.55	94.84
N	**6853978**	**1682507**	**364179**	**608116**	**9033834**	**2116448**	**702276**	**1090639**

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

Table 2a. Infant Mortality Rates* among All Weight Births by Race/Ethnicity: United States, 1989-1991, and 1995-1998

	1989-1991				1995-1998				% Change 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	6.27	13.26	7.12	5.86	4.92	9.92	5.53	4.31	-21.53	-25.19	-22.33	-26.45	2.11	1.14	0.93	2.02	1.12	0.88
SG/LBW	0.24	1.00	0.26	0.24	0.26	0.86	0.27	0.24	8.33	-14.00	3.85	0.00	4.17	1.08	1.00	3.31	1.04	0.92
Maternal Comp.	0.12	0.22	0.09	0.07	0.10	0.18	0.10	0.05	-16.67	-18.18	11.11	-28.57	1.83	0.75	0.58	1.80	1.00	0.50
Other	5.91	12.05	6.77	5.54	4.56	8.88	5.17	4.02	-22.84	-26.31	-23.63	-27.44	2.04	1.15	0.94	1.95	1.13	0.88

Source: See Table 1.

Table 2b. Infant Mortality Rates* among Low Weight Births by Race/Ethnicity: United States, 1989-1991, and 1995-1998

	1989-1991				1995-1998				% Change 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	57.35	64.25	57.87	61.88	41.29	49.71	44.24	44.59	-28.00	-22.63	-23.55	-27.94	1.12	1.01	1.08	1.20	1.07	1.08
SG/LBW	4.15	7.37	4.00	4.68	3.96	6.60	3.75	4.40	-4.58	-10.45	-6.25	-5.98	1.78	0.96	1.13	1.67	0.95	1.11
Maternal Comp.	2.06	1.57	1.36	1.37	1.53	1.33	1.32	0.85	-25.73	-15.29	-2.94	-37.96	0.76	0.66	0.67	0.87	0.86	0.56
Other	51.14	55.31	52.52	55.84	35.79	41.78	39.17	39.34	-30.02	-24.46	-25.42	-29.55	1.08	1.03	1.09	1.17	1.09	1.10

Source: See Table 1.

Table 3a. Odds Ratios for Effects of Risk Factors on Infant Mortality among All Weight Births : United States, 1989-1991 and 1995-1998

	Model 1			Model 2			Model 3			Model 4		
	SG/LBW	**Maternal**	**Others**	**SG/LBW**	**Maternal**	**Others**	**SG/LBW**	**Maternal**	**Others**	**SG/LBW**	**Maternal**	**Others**
Ethnic Group [NHW]						**1989-1991**						
NHB	4.140***	1.910***	2.029***	2.741***	1.318***	1.337***	2.127***	1.096	1.293***	1.188***	0.711***	0.935***
MX-US	0.946	0.760	1.121***	0.777**	0.683**	0.840***	0.726***	0.724*	0.835***	0.712***	0.715*	0.827***
MX-IM.	1.038	0.627***	0.907***	0.904	0.664**	0.638***	0.953	0.742*	0.649***	1.091	0.817	0.698***
Intercept	-8.452***	-9.157***	-5.173***	-9.377***	-10.482***	-6.055***	-10.012***	-11.105***	-6.272***	-13.715***	-13.864***	-6.414***
-2LL		830396.4 ***			811904.4 ***			786668.1 ***			698871.8 ***	
Ethnic Group [NHW]						**1995-1998**						
NHB	3.475***	1.830***	1.952***	2.688***	1.620***	1.391***	2.123***	1.329***	1.359***	1.323***	0.869*	1.035***
MX-US	1.137	0.937	1.137***	0.950	0.874	0.860***	0.914	0.894	0.891***	0.902	0.881	0.882***
MX-IM.	0.901	0.488***	0.880***	0.809***	0.468***	0.645***	0.778***	0.484***	0.676***	0.873*	0.531***	0.718***
Intercept	-8.387***	-9.331***	-5.439***	-9.293***	-10.406***	-6.303***	-9.936***	-11.156***	-6.516***	-13.266	-13.713***	-6.673***
-2LL		904978.7 ***			885929.1 ***			858265.2 ***			767194.1 ***	

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 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, smoking and weight gain.

Model 4 includes the controls from model 3, plus gestational age and birth weight.

Table 3b. Odds Ratios for Effects of Risk Factors on Infant Mortality among Low Weight Births : United States, 1989-1991 and 1995-1998

	Model 1			Model 2			Model 3		
	SG/LBW	**Maternal**	**Others**	**SG/LBW**	**Maternal**	**Others**	**SG/LBW**	**Maternal**	**Others**
Ethnic Group [NHW]					**1989-1991**				
NHB	1.772***	0.808***	1.080***	1.618***	0.778***	0.956***	1.214***	0.716***	0.844***
MX-US	0.843	0.676**	1.012	0.826	0.729	0.948*	0.693***	0.706*	0.813***
MX-IM.	1.171*	0.708**	1.064**	1.239**	0.912	1.043	1.131	0.843	0.882***
Intercept	-5.505***	-6.214***	-2.966***	-5.898***	-6.907***	-3.464***	-6.330***	-7.750***	-3.674***
-2LL		306676.6 ***			304107.9 ***			296124.9 ***	
Ethnic Group [NHW]					**1995-1998**				
NHB	1.733***	0.911	1.180***	1.695***	1.039	1.104***	1.320***	0.874*	0.966**
MX-US	1.053	0.858	1.102***	1.015	0.945	1.026	0.855*	0.839	0.894***
MX-IM.	1.058	0.536***	1.093***	1.074	0.594***	1.035	0.862*	0.501***	0.878***
Intercept	-5.582***	-6.536***	-3.342***	-5.899***	-6.954***	-3.788***	-6.410***	-7.703***	-3.976***
-2LL		353339.9 ***			351304.6 ***			342515.0 ***	

SOURCE: See Table 1.

† Model Covariates: Model 1 is the bivriate relationship.

Model 2 controls 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, smoking and weight gain.

Table 4. Race/Ethnic Differences (Second Differences) in the Change in Infant Mortality 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 - SG/LBW												
1989	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.
1990	0.000448	0.82	0.00019	0.43	0.00001	0.01	-0.00002	-0.03	0.00009	0.09	0.00014	0.26
1991	0.000179	0.33	0.00008	0.18	-0.00078	-0.74	-0.00033	-0.51	-0.00196	-1.81	-0.00042	-0.66
1995	0.000309	0.57	0.00021	0.49	-0.00076	-0.79	-0.00027	-0.48	-0.00076	-0.78	-0.00012	-0.23
1996	0.000979	1.82	0.00049	1.17	-0.00047	-0.50	-0.00014	-0.24	-0.00013	-0.14	0.00008	0.15
1997	0.000498	0.93	0.00030	0.73	-0.00143	-1.49	-0.00050	-0.86	-0.00020	-0.22	-0.00001	-0.02
1998	0.000586	1.10	0.00031	0.75	-0.00170	-1.74	-0.00057	-0.94	0.00008	0.09	0.00009	0.20
Infant Mortality - Maternal Complications												
1989	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.
1990	-0.00002	-0.06	0.00000	-0.02	-0.00006	-0.09	-0.00005	-0.09	-0.00048	-0.76	-0.00008	-0.35
1991	0.00012	0.40	0.00005	0.26	-0.00018	-0.27	-0.00009	-0.16	0.00007	0.12	-0.00001	-0.05
1995	-0.00017	-0.59	-0.00003	-0.17	-0.00046	-0.73	-0.00022	-0.40	0.00009	0.17	-0.00010	-0.52
1996	-0.00022	-0.79	-0.00005	-0.27	-0.00042	-0.70	-0.00013	-0.28	0.00021	0.43	-0.00013	-0.74
1997	-0.00039	-1.40	-0.00008	-0.51	-0.00074	-1.23	-0.00027	-0.53	-0.00021	-0.41	-0.00021	-1.17
1998	-0.00009	-0.33	0.00001	0.03	-0.00050	-0.84	-0.00017	-0.35	-0.00028	-0.54	-0.00022	-1.21
Infant Mortality - All Other Causes												
1989	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.	ref.
1990	-0.00156	-0.92	-0.00122	-0.77	-0.00160	-0.38	-0.00185	-0.67	0.00305	0.77	0.00043	0.13
1991	-0.00355	-2.14	-0.00227	-1.48	-0.00474	-1.12	-0.00351	-1.28	0.00208	0.54	-0.00041	-0.13
1995	-0.00323	-2.01	-0.00300	-2.09	-0.00416	-1.08	-0.00555	-2.22	0.00086	0.24	-0.00245	-0.84
1996	-0.00503	-3.14	-0.00411	-2.90	-0.00776	-2.03	-0.00734	-2.94	0.00391	1.11	-0.00171	-0.61
1997	-0.00241	-1.52	-0.00275	-1.96	-0.00542	-1.44	-0.00658	-2.67	0.00160	0.45	-0.00251	-0.88
1998	-0.00528	-3.34	-0.00426	-3.02	-0.00340	-0.92	-0.00604	-2.50	0.00228	0.65	-0.00249	-0.88

SOURCE: See Table 1.

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

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