Racial and Ethnic Disparities in Infant Mortality

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Racial and ethnic disparities in infant mortality in the United States seem to defy all attempts at elimination. Despite national priorities to eliminate these disparities, black infants are 2.5 times more likely to die in infancy compared with non-Hispanic white infants. This disparity is largely related to the greater incidence among black infants of prematurity and low birth weight, congenital malformations, sudden infant death syndrome, and unintentional injuries. This greater incidence, in turn, is related to a complex interaction of behavioral, social, political, genetic, medical, and health care access factors. Thus, to influence the persistent racial disparity in infant mortality, a highly integrated approach is needed, with interventions adapted along a continuum from childhood through the periods of young adulthood, pregnancy, postpartum and beyond. The content and methodologies of these interventions need to be adapted to the underlying behaviors, social influences, and technology and access issues they are meant to address.
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Racial and ethnic disparities in infant mortality in the United States seem to defy all attempts at elimination. Despite national priorities to eliminate these disparities,[1] non-Hispanic black infants are 2.5 times more likely to die in infancy compared with non-Hispanic white infants.[2] American Indian or Alaskan Native and Puerto Rican infants continue to also be more likely to die in infancy (1.5 and 1.4 times more than non-Hispanic white infants, respectively), whereas other ethnic minorities, including other Hispanic groups and Asian and Pacific Islanders, have equivalent or lower infant mortality rates.[3]

The article in this supplement by MacDorman provides detailed data about this infant mortality gap, leading causes of infant mortality by race and ethnicity, and the factors that contribute to the gap, especially that of preterm birth.[4] In this article, we will further describe the causes of infant mortality by race and ethnicity in the neonatal and postneonatal periods, and seek to identify possible explanations for the persistent disparities, areas of research needed to better understand the disparities, and recommendations for future research.

Leading Causes of Infant Mortality in the Neonatal and Postneonatal Periods

Two-thirds of all infant deaths occur in the neonatal period (birth to 28 days).[2] There are clear patterns of causes of infant deaths depending on the time frame after birth. Infants who die in the neonatal period are affected primarily by congenital malformations and disorders associated with preterm birth, including disorders related to short gestation and low birth weight not elsewhere classified, maternal complications of pregnancy, respiratory distress of newborn, bacterial sepsis of newborn, and neonatal hemorrhage. Sudden infant death syndrome (SIDS) accounted for 5.9% of the neonatal deaths in 2006. This syndrome reaches its peak incidence at 2-4 months of infant age, and as will be described in a later section, is more common among infants who are preterm or low birth weight.

For non-Hispanic black infants, the 3 leading causes of neonatal death are disorders related to short gestation and low birth weight not otherwise classified (2.99/1,000 live births), congenital malformations (1.20/1000 lb), and newborn affected by maternal complications of pregnancy (0.90/1000 lb; Table 1).[3] For non-Hispanic white infants, the top 3 causes of death are congenital malformations (0.95/1000 lb), disorders related to short gestation and low birth weight (0.76/1000 lb), and newborn affected by maternal complications of pregnancy (0.32/1000 lb). Thus, the black-white gap

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Table 1 Causes of Neonatal Deaths by Race-Ethnicity for the 3 Leading Causes of Death

Causes of Neonatal Deaths	All Races				White, Non-Hispanic				Black, Non-Hispanic			
	Rank	Number	Percent of Total Deaths	Mortality Rate*	Rank	Number	Percent of Total Deaths	Mortality Rate*	Rank	Number	Percent of Total Deaths	Mortality Rate*
Disorders related to short gestation and low birth weight, not elsewhere classified	1	4718	24.8	1.11	2	1752	20.6	0.76	1	1844	32.8	2.99
Congenital malformations, deformations and chromosomal abnormalities	2	4202	22.1	0.99	1	2188	25.7	0.95	2	738	13.1	1.20
Newborn affected by maternal complications of pregnancy	3	1661	8.7	0.39	3	733	8.6	0.32	3	556	9.9	0.90
Newborn affected by complications of placenta, cord and membranes	—	—	—	—	—	—	—	—	—	—	—	—

Causes of Neonatal Deaths	Hispanic				American Indian or Alaska Native				Asian or Pacific Islander			
	Rank	Number	Percent of Total Deaths	Mortality Rate*	Rank	Number	Percent of Total Deaths	Mortality Rate*	Rank	Number	Percent of Total Deaths	Mortality Rate*
Disorders related to short gestation and low birth weight, not elsewhere classified	2	899	22.8	0.87	2	43	21.4	0.90	1	142	23.3	0.59
Congenital malformations, deformations and chromosomal abnormalities	1	1072	27.2	1.03	1	52	25.9	1.09	2	138	22.6	0.57
Newborn affected by maternal complications of pregnancy	3	296	7.5	0.29	—	—	—	—	3	46	7.5	0.19
Newborn affected by complications of placenta, cord and membranes	—	—	—	—	3	18	9.0	**	—	—	—	—

*Mortality rate is the number of deaths per 1000 live births.
**Figure does not meet standards of reliability or precision according to the National Vital Statistics Report.

for congenital malformations is 1.3, for short gestation and low birth weight (LBW) 3.9, and for maternal complications of pregnancy 2.8. The 3 leading causes of neonatal death for American Indians or Alaska Natives, Hispanic whites, and Asians and Pacific Islanders are shown in Table 1.

In the postneonatal period, the 3 leading causes of infant death among black and white infants are the same: SIDS, congenital malformations, and accidents (ie, unintentional injuries; Table 2).[3] However, the black-white disparities for SIDS, congenital malformations, and accidents are 2.1 (1.02 per 1000 lb vs 0.49 per 1000 lb), 1.8 (0.60/1000 vs 0.34/1000 lb), and 2.5 (0.52/1000 vs 0.21/1000 lb), respectively. Therefore, this black-white gap is present for each of these leading causes of death in similar magnitude. The 3 leading causes of postneonatal mortality for the other racial-ethnic groups are shown in Table 2.

Preterm-related Causes of Death

Black women are twice as likely as white women to deliver preterm infants. It is estimated that 54% of the black-white disparity in infant mortality is attributable to the greater incidence of preterm births among black women. The greater incidence of prematurity leads to both greater prevalences of LBW and very low birth weight (VLBW) infants and a greater incidence of mortality in the black community. Compared with normal birth weight infants, LBW infants are 25 more times likely to die, and VLBW infants are 100 times more likely to die.[5]

Several theories have been proposed to explain the excess deaths among black infants. (See the article by MacDorman in this supplement for a more in-depth discussion.)[4] College-educated black women are more likely to experience an infant death compared with college-educated white women, even after controlling for prenatal care, parity, maternal age, and marital status.[6-8]

As a result, one theory posits that black women experience greater lifetime exposure to acute and chronic stressors, including racism, which increases their susceptibility to preterm delivery. Maternal stress may affect immune, endocrine, and vascular functioning, which can affect uteroplacental function. There may also be an interaction between stress and infection, increasing the risk of preterm birth.[9] Hogue and colleagues propose that black women may age prematurely because of exposure to traumatic life events beginning early in life related to poverty or stressful life events, a phenomenon they call "stress age."[5] There may be differences in stress age related to genetic resistance to stressors or other gene-environment interactions. The effects of stress on the individual are further modified by other factors, such as harmful coping behaviors (such as smoking or drug use) and emotional disturbances (such as anxiety or depression).

The authors of other studies have also demonstrated an association between maternal stress levels and preterm infants.[10-13] In a study of low-income primiparous black women and women of Mexican origin, black women were 2.4 times more likely to give birth to a preterm infant and 3 times more likely to deliver an LBW infant, and prenatal stress was associated with unhealthy behaviors in pregnancy, such as smoking or substance abuse, and negative attitudes during pregnancy. Although poverty can increase stress,[11] poverty alone cannot explain the greater incidence of preterm births among black women because low-income women of Mexican origin do not experience a similar plight. Arguing against a genetic cause is the finding that infants of African-born black women have similar birth weights as infants of U.S.-born white women.[14,15] Maternal attitudes toward pregnancy are more predictive of preterm birth and LBW than poverty. Women with positive attitudes are more likely to engage in healthy behaviors during pregnancy and therefore experience better perinatal outcomes.[10]

Previously, preterm black infants had a survival advantage over white infants, but advances in perinatology, including improvements in mechanical ventilation and surfactant, have eliminated this advantage. For unknown reasons, the decrease in mortality of black infants since the introduction of these technologies has been smaller than the decrease in white infant mortality, despite equal access to tertiary care facilities.[16,17]

Howell et al[5] argue for a new framework to address infant mortality caused by prematurity. Wise[18] describes the mechanisms for black-white infant mortality disparities to be attributable to "differential underlying risk status" and "differential access to effective interventions." Research on the former has yet to improve the efficacy of primary prevention.[5] Thus, Howell proposes to focus on measuring and improving quality of care, particularly for infants who are VLBW.[5] "Adequacy" of care is most commonly expressed as the number of prenatal visits and when in pregnancy care began. Data from the Early Childhood Longitudinal Study—Birth Cohort found that among a large number of biological, sociodemographic, and pregnancy behavior variables, prenatal care adequacy, as measured by the Kessner Index, was the most important predictor of prematurity across all racial and ethnic groups.[19]

However, the Kessner index does not reflect the quality of care received during these prenatal visits. Indeed, few studies to date have measured quality of care received by VLBW infants and differences by race and ethnicity. It is possible that women of different ethnicities receive prenatal care in different hospitals and that the hospitals serving black mothers provide lower quality of care. Alexander and colleagues[20] found that hospitals in New York City that serve a high proportion of minority patients have higher than expected VLBW mortality. Black VLBW infants are served disproportionately by hospitals with greater rates than expected of VLBW mortality, which may be an indicator of poorer quality of care.

It has been difficult to identify the specific characteristics that comprise "high quality care" for LBW and VLBW infants. A review of infant death and injury conducted by the Joint Commission identified the most important factors in adverse neonatal outcomes as staff hierarchy and intimidation, inability to function as a team, and poor communication.[21]

Table 2 Causes of Postneonatal Deaths by Race-Ethnicity for the 3 Leading Causes of Death

Causes of Postneonatal Deaths	All Races				White, Non-Hispanic				Black, Non-Hispanic			
	Rank	Number	Percent of Total Deaths	Mortality Rate*	Rank	Number	Percent of Total Deaths	Mortality Rate*	Rank	Number	Percent of Total Deaths	Mortality Rate*
Sudden infant death syndrome	1	2116	22.2	0.50	1	1130	25.7	0.49	1	632	21.9	1.02
Congenital malformations, deformations and chromosomal abnormalities	2	1617	17	0.38	2	775	17.6	0.34	2	369	12.8	0.60
Accidents (unintentional injuries)	3	1031	10.8	0.24	3	489	11.1	0.21	3	320	11.1	0.52

Causes of Postneonatal Deaths	Hispanic				American Indian or Alaska Native				Asian or Pacific Islander			
	Rank	Number	Percent of Total Deaths	Mortality Rate*	Rank	Number	Percent of Total Deaths	Mortality Rate*	Rank	Number	Percent of Total Deaths	Mortality Rate*
Sudden infant death syndrome	2	266	14.8	0.26	1	49	25.5	1.03	2	37	14.1	0.15
Congenital malformations, deformations and chromosomal abnormalities	1	394	21.9	0.38	3	21	10.9	0.44	1	64	24.3	0.27
Accidents (unintentional injuries)	3	168	9.3	0.16	2	34	17.7	0.71	3	23	8.7	0.10

*Mortality rate is the number of deaths per 1000 live births.

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Gaps in Knowledge

Elimination of racial and ethnic disparities in preterm-related infant mortality requires identification of the factors causing differences in underlying risk status and the nature of the differences in access to effective interventions.[18] This will include expanding the paradigms that explain causation to include models of stress, such as the one proposed by Hogue and colleagues.[6] Developing reliable and valid measures of quality of care, both for the pregnant mother and infant, is critical to advancing the research related to differential access to health care.

Congenital Malformations

Black infants are 1.3 times more likely to die from congenital malformations than white infants. As shown previously, the black-white disparity is more pronounced in the postneonatal period. Hispanic infants overall are only 1.1 times more likely to die from congenital malformations, but Mexican infants are 1.2 times more likely to die than non-Hispanic white infants. American Indians or Alaska Natives have 1.3 times greater rates of mortality from congenital malformations compared with non-Hispanic white infants.

Lee and colleagues[22] studied trends in congenital malformations from 1970 to 1997, comparing black and white infants for all malformations combined and for 7 subcategories. Infant mortality attributable to congenital malformations during this time decreased from 3.0/1000 to 1.6/1000 lb, a 48.4% decrease. During that same period, total infant mortality decreased 64.8%; thus, congenital malformations became a more prominent cause of death in 1997. The nervous, cardiovascular, and respiratory systems account for more than 60% of all congenital malformations.

The trends in mortality attributable to congenital malformations differ by race. In 1970-1971, black infants had lower mortality (2.6/1000 lb) than white infants (3.1/1000 lb), but this reversed by 1980-1981 because of larger decreases among white infants in that period.[22] After 1981, the decreases in mortality were similar for both groups. In 2006, the infant mortality rate because of congenital malformations for blacks infants was 1.75/1000 lb and for non-Hispanic white infants was 1.27/1000 lb.[3]

The trends in mortality have varied by the type of congenital malformation. In 1970-1971, white infants had double the rate of central nervous system malformations compared with black infants (0.8/1000 and 0.4/1000 lb, respectively). Because decreases were subsequently greater for white infants, the difference in mortality was no longer present by 1996-1997. Rates of cardiovascular malformations were similar between black and white infants in the earlier period, then decreased more rapidly for white infants, with subsequent declines similar for both groups; mortality rates are now similar. Trends were similar for malformations of the digestive system for both groups, with rapid early decreases followed by gradual reduction in decreases since the early 1980s. In the early 1990s, malformations because of chromosomal abnormalities increased for both groups, possibly because of increased testing and diagnosis, and then declined similarly.

A more recent assessment of racial differences in neonatal deaths because of congenital malformations, 2003-06, found that rates were similar among black and white infants (0.21/1000 and 0.20/1000 lb, respectively, $P = 0.28$).[23] However, there were differences by gestational age. The rate was 30% lower for preterm black infants when compared with white infants, but 20% greater for term black infants. The reasons for these differences are not known. One possible explanation is that live-born infants who die shortly after birth may be misclassified as fetal deaths and this may be done more commonly for black infants[23]; the fetal mortality rate for blacks is about twice as high as among white infants.[24]

The decrease in infant deaths because of congenital malformations may be attributed to various factors, including improved preventive measures, increasing prenatal detection of serious defects, selective termination of pregnancy, and improved survival of affected infants.[22] Decreases in infant mortality because of severe malformations incompatible with life, such as anencephaly, are probably the result of preventive measures and/or antenatal diagnosis and termination. Similarly, decreases in the number of infants in developed countries born with neural tube defects may be related more to increased antenatal diagnosis and selective termination than to folic acid supplementation. Although periconceptional supplementation with folic acid has been shown to reduce the risk of neural tube defects,[25,26] decreases in neural tube defects were already occurring before recommendations to supplement with folic acid.[22]

Improvements in the medical and surgical treatment of infants with other congenital malformations, especially those of the cardiovascular system, have also contributed to the greater survival of these infants and subsequent decrease in infant mortality.[22] In addition, antepartum diagnosis through routine pregnancy ultrasound and selective termination of affected infants have contributed to lower prevalence of infants who are born with these anomalies, and thus, to lower infant mortality.[22,27]

It is not known why there has been a reversal in the infant mortality rates for black and white infants because of congenital anomalies. The possible explanations include racial differences in preventive measures (eg, folic acid supplementation), access to and acceptance of antenatal screening and selective termination of pregnancy, and medical and surgical treatment for infants with congenital malformations. In a study of women older than 40 residing in Georgia, only 15% of women used antenatal chromosomal testing, but there was wide variation by race and geographic location.[28] Only 0.5% of rural black women had testing, compared with 60% of urban white women.

Gaps in Knowledge

Significant decreases in infant mortality because of congenital malformations during the past 4 decades are encouraging, but these decreases are leveling off and racial-ethnic disparities persist, albeit smaller than those for the other leading
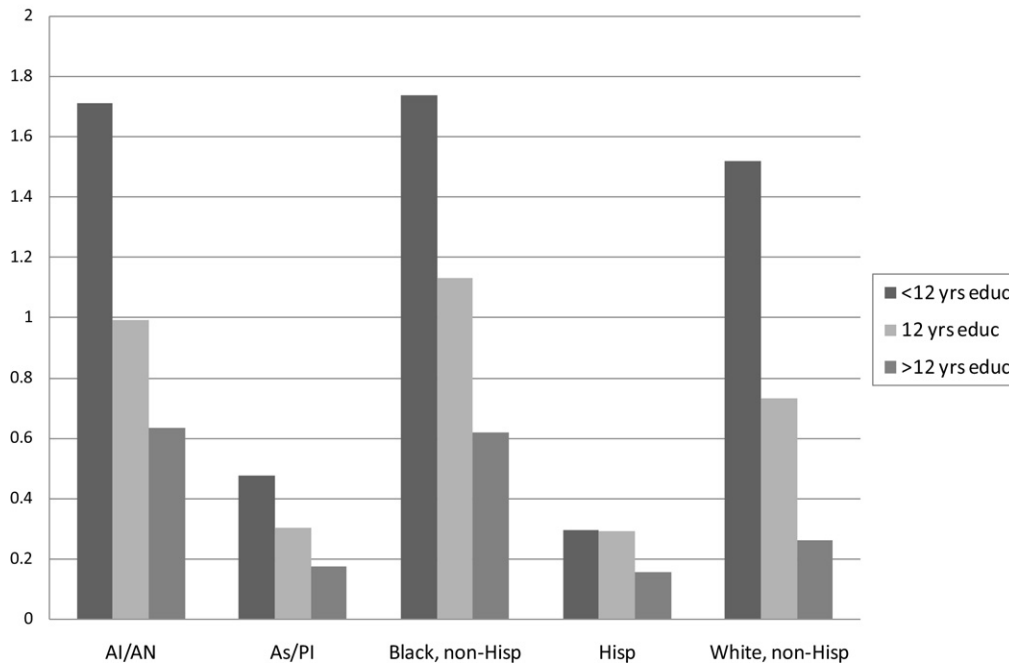
Figure 1 SIDS rates (per 1000 lb) by mother's education, race, and Hispanic origin: United States, 2003-2005.

causes of infant mortality described in this paper. To continue the gains made previously, ongoing research is needed to examine reasons for the disparities in congenital malformations, which are now more prevalent among black, American Indian or Alaska Native, and Hispanic infants compared with white infants. This should include assessment of preventive measures, access to and acceptance of antenatal diagnosis and termination, and access to and acceptance of treatment for infants born with congenital malformations. Qualitative methods may be needed especially to determine differential acceptance of antenatal testing and pregnancy termination.

Although surveillance systems are in place in most states, with the assistance of the Centers for Disease Control and Prevention, to monitor trends in congenital malformations, there may be under-reporting of deaths because of these disorders, and this may also vary by race-ethnicity.[29] Examination of system-specific causes of death as well as gestational-age specific rates by race-ethnicity may help to further understand the causes for racial disparities.

SIDS

Despite the overall decline in SIDS globally, there continue to be racial and ethnic disparities, not only in the United States but in other developed countries, such as Canada, Australia, and New Zealand.[30-35] In the United States, black and American Indian or Alaska Native infants at all socioeconomic levels have SIDS rates that are 2-3 times the national average.[36,37] In 2006, infants born to black and American Indian or Alaskan Native mothers succumbed to SIDS at a rate of 1.04 and 1.19/1000 lb, respectively, approximately double the rate for infants born to white mothers (0.56/1000 lb), more than 3 times the rate for infants born to Hispanic moth-

ers (0.27/1000 lb), and more than 4 times the rate for infants born to Asian or Pacific Islander mothers (0.23/1000 lb).[3] Furthermore, the extent of these disparities has increased during the past 2 decades.[38,39] The largest decrease in SIDS has occurred for infants whose mothers are more educated.[40] Although SIDS rates are generally greater in families of lower socioeconomic status or educational attainment, infants born to highly educated black mothers have similar or greater SIDS rates than infants born to Hispanic and Asian or Pacific Islander women who did not complete high school (Fig. 1).

The current working model for SIDS is that it occurs when an infant with an intrinsic vulnerability, such as a dysfunctional or immature arousal mechanism, is confronted with an exogenous stressor, such as prone sleep position, at a critical stage of development. According to this model, both biological and behavioral factors are important. In addition, biological differences that may result in an intrinsic vulnerability may partially explain the racial disparity in SIDS. For instance, black subjects metabolize nicotine differently from white subjects, so that they have greater levels of serum cotinine (the proximate metabolite of nicotine), despite smoking fewer cigarettes.[41] In utero exposure to tobacco impairs cardiovascular reflexes[42] and adversely affects arousal[43-50] in infants. Black mothers are less likely to smoke, both prenatally and after the infant's birth, than white mothers,[51] but slower metabolism may account for an increased risk because of smoke exposure.

In addition, genetic polymorphisms that may play an etiologic role in SIDS may be different in blacks. For example, a polymorphism (12-repeat intron-2) of the promoter region of the serotonin transporter, which also enhances serotonin transporter efficiency, is increased in black SIDS cases,[52] and not in a Norwegian population.[53] Abnormalities in the medullary serotonergic system have

been associated with SIDS[54-57]; this area of the brainstem plays an important role in arousal and autonomic functions and, if dysfunctional, may prevent normal protective responses to exogenous stressors when the infant is asleep. Therefore, polymorphisms, such as described above may be important in explaining the increased rate in certain populations.

Behavioral risk factors are also important with regards to SIDS. For instance, black subjects are twice as likely to place infants prone for sleep.[58,59] Although the supine sleep position is the norm for American Indian or Alaska Native, Aboriginal Australian, and New Zealand Maori infants, there are high rates of smoke exposure[60] and bed sharing in these groups, both of which place these infants at greater risk of SIDS.[33,61] Hispanic subjects in general are less likely to place infants prone than black subjects.[62,63] In the largest U.S. case-control study, one-third of SIDS deaths could be attributed to prone positioning.[59] The primary reasons for placing infants prone among blacks are concerns about choking while supine and the perception that infants are more comfortable and sleep longer when prone.[64] In addition, black mothers are more likely to receive a prone recommendation at the delivery hospital than non-black mothers.[59]

Aside from sleep position, smoke exposure is the largest contributing risk factor for SIDS.[65] Maternal smoking during pregnancy has been well demonstrated to be a major risk factor.[65-68] Smoke in the infant's environment after birth is a separate risk factor in some studies,[67,69] although separating this variable from prenatal smoke exposure is problematic. It is estimated that one-third of SIDS deaths could be prevented if all maternal smoking during pregnancy was eliminated.[70,71] In the 2005 Pregnancy Risk Assessment Monitoring System survey, the prevalence of smoking during pregnancy was greatest in Alaska Natives (36.3%) and American Indians (20.6%), lower in non-Hispanic whites (18.5%) and non-Hispanic blacks (10.1%), and lowest among Asian or Pacific Islanders (5.4%) and Hispanics (4.0%).[51]

Breastfeeding has been found to decrease SIDS risk.[72] Hispanics are more likely to initiate breastfeeding (80%) compared with blacks (65%), and among low-income families, Hispanics have the greatest percentage of infants ever breastfed (74%, vs 37% in non-Hispanic blacks and 55% in non-Hispanic whites).[73]

To explore the question of biology vs behavior, consideration of differences within different generations of the same ethnic group are helpful. Specifically, in a study that examined the rates of SIDS for infants born to immigrant mothers vs infants born to mothers who were first generation or longer in the Unites States, the authors suggest a role for acculturation in the etiology of SIDS. In this study, Mexican–American infants with U.S.-born mothers had a 50% greater SIDS rate than infants with Mexico-born mothers, even after controlling for birth weight, maternal age, education, marital status, prenatal care, and socioeconomic status.[74] This finding implies that cultural or behavioral factors may be more important than biological factors in SIDS risk.

**Table 3** Leading Causes of Accidental Infant Death in 2005

Cause of Death	Number of Deaths	Infant Mortality Rate*
Accidental suffocation and strangulation in bed	515	0.12
Unspecified threat to breathing	115	0.03
Other specified threats to breathing	67	0.02
Person injured in unspecified motor vehicle accident, traffic	39	0.01
Drowning and submersion while in bathtub	36	0.009
Exposure to uncontrolled fire in building or structure	33	0.008
Car occupant injured in collision with car, pick-up truck, or van; passenger injured in traffic accident	28	0.007
Inhalation and ingestion of other objects causing obstruction of respiratory tract	26	0.006
Exposure to unspecified factor	22	0.005
Car occupant injured in noncollision transport accident; passenger injured in traffic accident	17	0.004
Total	898	0.22

*There were 4,138,573 live births in 2005. The infant mortality rate is the number of deaths per 1000 live births.

Accidental Deaths

Multiple causes of death, including motor vehicle accidents, falls, choking, fire, drowning, poisonings, and foreign body aspiration, comprise the general category of unintentional accidental deaths during infancy (ICD-10 V01-X59). The 10 leading causes of accidental infant death in the United States in 2005[75] are included in Table 3.

In general, black and American Indian or Alaskan Native infants succumb to unintentional accidental deaths at 2-3 times the rate of white infants and 4-6 times the rate of Asian or Pacific Islander and Hispanic infants.[3] Accidental suffocation and strangulation in bed (ASSB) is the most common cause of infant accidental death, accounting for approximately one-half of these deaths. A death is coded "accidental suffocation and strangulation in bed" (ICD-10 W75) when the terms asphyxia, asphyxiated, asphyxiation, strangled, strangulated, strangulation, suffocated, or suffocation are re-
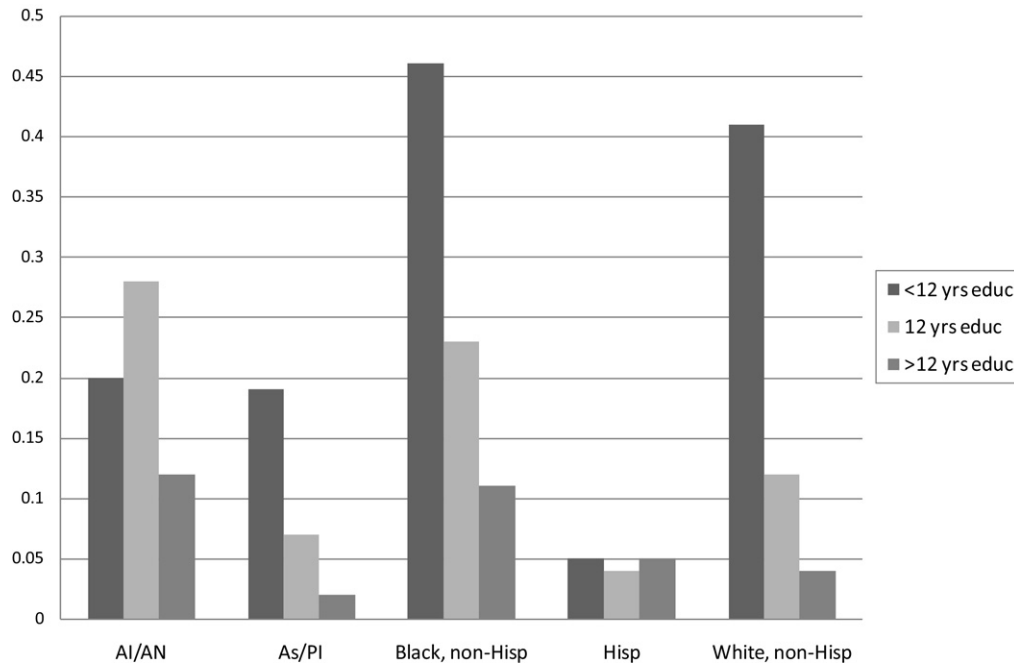
Figure 2 Accidental suffocation and strangulation in bed rates (per 1000 lb) by mother's education, race, and Hispanic origin: United States, 2003-2005.

ported, along with the terms bed or crib. Black and American Indian or Alaskan Native infants have high rates of ASSB (0.24 and 0.23/1000 lb, respectively, compared with 0.13/1000 lb for whites, 0.06/1000 lb for Asian or Pacific Islanders, and 0.06/1000 lb for Hispanics).[76] In recent years, ASSB rates have more than quadrupled in the United States, and black male infants younger than 4 months of age are disproportionately affected.[77] As with SIDS, the rates of ASSB for all racial/ethnic groups are higher with lower socioeconomic status (Fig. 2).

The major risk factors for ASSB are behavioral. Overlaying of the infant (which occurs during bed sharing) and use of soft bedding (such as pillows, blankets, and bumper pads) in the infant sleep environment are important contributing factors to ASSB deaths.[77] Black infants are twice as likely to bed share with their parents as white infants.[78,79] Studies demonstrate that approximately 40% of blacks routinely bed share, compared with fewer than 20% of whites.[79-81]

It is important to note, however, that Hispanic infants also have high rates of bed sharing, comparable with those seen in black infants. However, Hispanic infants have extremely low rates of ASSB. The discrepancy potentially arises from differences in bed sharing practices between these 2 groups. Particular concern has been raised for bed sharing in the presence of one or more risk factors: placing infants in the prone position while bed sharing,[82] bed sharing on couches,[83,84] bed sharing with a parent who smokes or who has used alcohol or drugs,[85] bed sharing with other children and nonparental adults,[82] and bed sharing in the presence of bedding accessories (pillows, blankets).[82] Recent work suggests that black families are more likely than white, Hispanic, and Asian families to bed share in the presence of these other risk factors. Not only do the vast majority of bed sharing deaths

occur in black infants,[86-89] black infants who die are more than 1.5 times as likely to have been placed on a sleep surface other than a crib (eg, adult bed, sofa, or waterbed) than white infants.[89] Furthermore, black infants who bed share are significantly more likely than Hispanic infants to be exposed to parental smoking.[38] Hispanics, by contrast, are 9% less likely than whites to bed share on a couch.[62]
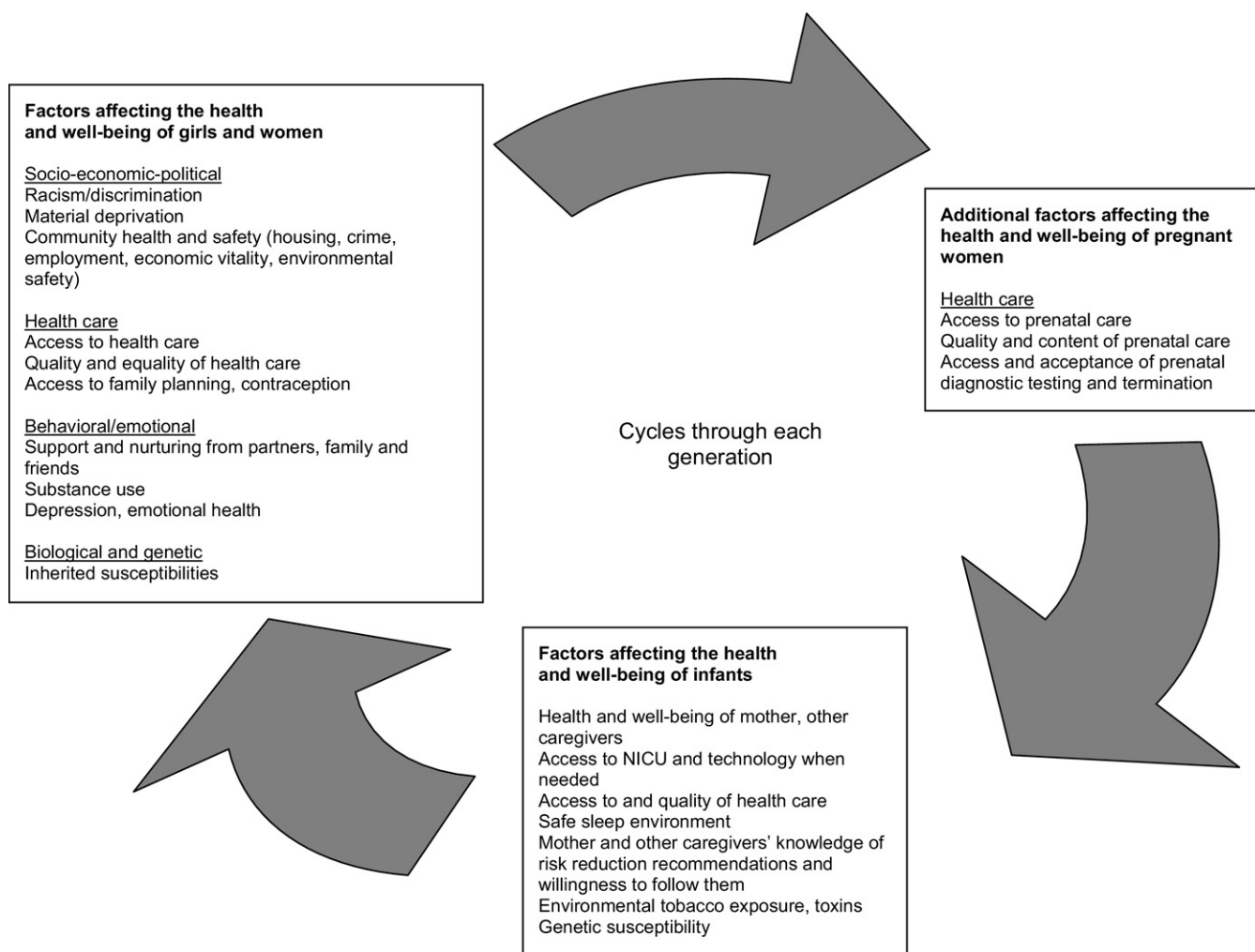
There are multiple reasons that parents choose to bed share, including cultural reasons, convenience for feeding (breast or formula), financial/space considerations, and concern about infant safety.[90] Many parents believe that bed sharing is the best way to monitor the infant and keep the infant safe. In particular, mothers of low socioeconomic status may choose to bed share because of their concerns about environmental dangers, such as vermin, stray gunfire, and random kidnappings.[90]

In addition, use of soft bedding, both under a sleeping infant and in the sleep environment, is common. Many parents perceive that the infant will sleep more comfortably if on a soft surface or that the soft bedding will prevent injury (eg, from bumping into crib railings or falling off the bed or sofa).[91] Despite the American Academy of Pediatrics' recommendations to remove soft bedding and to use firm surfaces for the infant sleep area,[92] rates of soft bedding use have not decreased.[93] Soft bedding use is more common in bed sharing infants[94,95] and in blacks.[94,96]

Gaps in Knowledge

There continue to be important gaps in knowledge with regards to the pathogenesis of SIDS. In addition, because of the hypothesized close relationship of biological and behavioral factors, it is important to understand the mechanisms by

Factors affecting the health and well-being of girls and women

Socio-economic-political
Racism/discrimination
Material deprivation
Community health and safety (housing, crime, employment, economic vitality, environmental safety)

Health care
Access to health care
Quality and equality of health care
Access to family planning, contraception

Behavioral/emotional
Support and nurturing from partners, family and friends
Substance use
Depression, emotional health

Biological and genetic
Inherited susceptibilities

Additional factors affecting the health and well-being of pregnant women

Health care
Access to prenatal care
Quality and content of prenatal care
Access and acceptance of prenatal diagnostic testing and termination

Cycles through each generation

Factors affecting the health and well-being of infants

Health and well-being of mother, other caregivers
Access to NICU and technology when needed
Access to and quality of health care
Safe sleep environment
Mother and other caregivers' knowledge of risk reduction recommendations and willingness to follow them
Environmental tobacco exposure, toxins
Genetic susceptibility

Figure 3 An integrative model to explain racial disparities in infant mortality.

which certain behaviors place infants at greater risk for SIDS. Qualitative data demonstrate that parental beliefs about SIDS may play an important role in their decisions relating to sleep behaviors.[97] Many parents do not find the link between SIDS and risk reduction recommendations plausible; they do not understand how certain behaviors can be defined as risk factors for an entity of unknown cause. In addition, many parents do not believe that their behavior (other than vigilance) can affect SIDS risk, as SIDS occurs randomly and is "God's will." Understanding and explanation of the mechanisms by which sleep behaviors affect SIDS risk will likely promote adherence with risk reduction recommendations, as parents may be more likely to follow recommendations that "make sense" to them.

SIDS and ASSB have similar behavioral risk factors, but little is yet known about parental decisions that increase the infant's risk for these 2 causes of death. Further qualitative research is critical to ascertain the underlying reasons, motivations, and influences for parental decisions, and to develop health recommendations that are culturally sensitive and increase parental self-efficacy.

Conclusions

Infant mortality has long been regarded as a key indicator of the health and well-being of a community or country. Implicit in this belief is the central role that social forces and public policy play in determining infant mortality rates. However, biological and medical factors also have a powerful role. Interestingly, racial disparities in infant mortality, regardless of the specific cause or time frame of death, share many features with regard to etiology and potential amelioration. There are critical and complex interactions between biological and behavioral risk factors. For instance, the triple risk theory is frequently cited to explain the cause of SIDS.[98] This states that SIDS is more likely to occur when 3 factors occur at the same time: a vulnerable infant, a critical stage in development and environment stress. The vulnerable infant refers to an abnormality in parts of the brain that control respiration or heart rate, that may lead to an abnormal arousal response; this could be caused by genetic variations or altered development in utero because of maternal substance use or environmental exposures. A critical stage in development refers to the age at which the infant's brain

is developing rapidly and changes in homeostatic controls take place, starting at the age of 1-2 months. Environmental stressors are behaviors (eg, maternal smoking, formula feeding) or unsafe sleeping environments (eg, prone sleeping, soft mattresses, bed sharing) that place the infant in a compromised position. A healthy infant would be expected to respond with an appropriate arousal response if confronted by an environmental challenge, such as sleeping in a face down position. Vulnerable infants are thought to respond inadequately to such an insult, and thus do not arouse, which eventually leads to death. According to this model, both biological and behavioral factors are important in the etiology of SIDS.

Although the specific causes of infant mortality differ, many of the same factors are at play in the causal pathways of these deaths and likely contribute to the racial disparities seen; namely, there are complex interactions of biological, behavioral, health care access, social and political factors that make some infants more vulnerable. When these factors consistently affect infants of specific races and ethnicities, racial disparities result. Health outcomes of infants are tied closely to the health of their mothers, and the health status of mothers is affected by factors that occur long before pregnancy (Fig. 3).[19] These complex risks include chronic emotional or medical conditions, adverse maternal behaviors (such as drug, tobacco or alcohol use), previous obstetrical complications, poor nutrition, dangerous communities, limited access to high-quality health care and reproductive health services, and poverty and extreme social needs. Thus, to influence the persistent racial disparity in infant mortality, a highly integrated approach is needed, with interventions adapted along a continuum from childhood through the periods of young adulthood, pregnancy, postpartum and beyond.[18] Furthermore, it will not be sufficient to provide interventions to mothers in isolation; the integrated approach needs to also involve mothers' partners, family members, and communities. The content and methodologies of these interventions need to be adapted to the underlying behaviors, social influences, and technology and access issues they are meant to address. There is no doubt that the success of any intervention will require a deeper understanding of the lived experience of each target group and their motivations to make changes.

Acknowledgments

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