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DELIVERY PAYMENT SOURCE DIFFERENCES IN INFANT MORTALITY ATTRIBUTABLE TO BIRTH DEFECTS BY RACE AND ETHNICITY— UNITED STATES, 2011 TO 2013

BY

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Degree to be awarded: M.P.H.

Executive MPH

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Abstract

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In the United States (U.S.), 1 in 33 infants are born with a birth defect. Infant mortality attributable to birth defects (IMBD) is the leading cause of infant mortality in the U.S. with 1 in 5 infant deaths resulting from complications from birth defects. IMBD rates differ across neonatal (birth to <28 days) and postneonatal (≥28 days to <1 year) periods and by race and ethnicity. Socioeconomic status (SES) is also hypothesized to affect IMBD, but how SES affects IMBD has not been well-studied. The 2003 revision of the birth certificate included a new variable on health insurance payment source for delivery, a proxy indicator for one aspect of SES. Starting in 2011, the CDC's National Center for Health Statistics added this variable to their data linking birth certificates and infant death certificates. We used data on births from 2011–2013 to examine whether there is an association between IMBD and payment source for delivery and whether the association is modified by racial or ethnic group (non-Hispanic white, non-Hispanic black, and Hispanic). We examined neonatal and postneonatal IMBD separately using Poisson regression to calculate the adjusted rate ratio (aRR) comparing IMBD rates among births covered by Medicaid with those covered by private insurance, adjusting for maternal age. For births covered by Medicaid, the neonatal IMBD rate was 38% higher and the postneonatal IMBD rate was 63% higher compared with births covered by private insurance. All race and ethnic groups had a similar trend of increased IMBD for births covered by Medicaid compared with private insurance, although the postneonatal mortality aRR for infants of non-Hispanic black mothers was slightly attenuated. Our results suggest that Medicaid is associated with higher IMBD rates, particularly during the postneonatal period, and that the association between payment source and IMBD is minimally modified by race and ethnicity. Insurance status could be a marker for SES or for access to health care. Examining the role of additional SES measures and of access to quality care could clarify the reason insurance status is associated with IMBD and help plan intervention strategies to reduce IMBD rates in the U.S.

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Chapter 1. Introduction

Birth defects affect 1 in every 33 infants in the United States (U.S.) (1, 2). Infant mortality attributable to birth defects (IMBD) is the leading cause of infant mortality in the U.S. with 1 in 5 infant deaths resulting from complications from birth defects (3, 4). Morbidity and mortality from birth defects impact families both emotionally and financially; in addition, birth defects considerably influence health care costs, and the nation's burden of disease and long-term disability (5, 6). Reducing infant mortality in general and IMBD specifically are current goals of Healthy People 2020 (7). The aim of this thesis is to assess the relation between insurance coverage and IMBD. Below we detail factors influencing IMBD rates, including the prevalence of birth defects, race and ethnicity, gestational age at birth, and socioeconomic status (SES).

Prevalence of Birth Defects

State and local surveillance data indicate 3 out of 100 infants in the U.S. are born with a birth defect (1, 2). National estimates of birth defects prevalence prior to 2000 (8) were based on data from the Birth Defects Monitoring Program (BDMP), which used hospital discharge data. Much of the current data on national birth defects prevalence comes from the National Birth Defects Prevention Network (NBDPN), which collects population-based surveillance data from participating states starting in 1997. From 2004–2006, national prevalence estimates were calculated from 14 programs in the NBDPN with active/passive case-finding with case verification (32.2% of all live births in the U.S.) for 21 defects in the following categories: central nervous system (CNS) defects, eye defects, cardiovascular defects, orofacial, gastrointestinal, musculoskeletal defects, and chromosomal anomalies (9). The estimates were standardized to the racial and ethnic distribution of the U.S. live birth population from 2004 to 2006. After adjusting for maternal race and ethnicity, a chromosomal abnormality—Down syndrome (Trisomy 21)—was the most prevalent with 13.56 cases per 10,000 live births, followed by orofacial clefts (cleft lip with or without cleft palate at 10.63 cases per 10,000 live births, and cleft palate without cleft lip at 6.35 cases per 10,000 live births), and then atrioventricular septal defect (AVSD) at 4.71 cases per 10,000 live

births. Of the 21 defects studied, encephalocele (0.84 cases per 10,000 live births) and common truncus (0.74 cases per 10,000 live births) were the least prevalent. In the 2004–2006 data from the NBDPN, significant changes in the estimated national prevalence from those in 1999–2001 were observed for anencephaly (2.51 to 2.06 cases per 10,000 live births), transposition of great arteries (4.73 to 3.00 cases per 10,000 live births), and gastroschisis (3.73 to 4.49 cases per 10,000 live births) (10).

There are differences in the prevalence of birth defects by race and ethnicity as described here using a representative set of studies. Using data from 1978–2005, the Metropolitan Atlanta Congenital Defects Program (MACDP) reported a lower overall prevalence of birth defects among births to black mothers [prevalence ratio (PR): 0.94, 95% confidence interval (CI): 0.93–0.95)] and Hispanic mothers (PR: 0.89, CI: 0.86–0.93) than among births to white mothers (1). Between 1999–2011, a population-based study from Texas reported that non-Hispanic white women are 9% more likely than non-Hispanic black women and 7% more likely than Hispanic women to deliver an infant with a birth defect (2). Similarly, using the Nationwide Inpatient Sample, administrative data from 2008, the risk of all birth defects was reportedly higher in infants of non-Hispanic whites compared with those of non-Hispanic blacks [relative risk (RR): 0.9, CI: 0.8–0.9] and Hispanics (RR: 0.9, CI: 0.8–0.9) (11). In contrast, the overall prevalence of birth defects was found to be lower among births to non-Hispanic white women than that among births to African-American women using surveillance data from California between 1989–1997 (12). Overall prevalence rates by race and ethnicity likely depend on the type of data source as well as which defects are in included in the study.

Comparison of specific birth defects shows that prevalence rates differ by race and ethnicity across defects (Table 1). Differences in the prevalence of birth defects between racial and ethnic groups may indicate racial or ethnic disparities in different underlying genetic susceptibilities, prenatal diagnosis rates, pregnancy termination rates (13), SES, or exposures to risk factors (14, 15). On the other hand, these differences may be a result of using different data sources with different methodology related to case ascertainment, adjustment for confounders, inclusion of still births and elective terminations, and the coding of birth defects (16).

Birth Defects Mortality

Birth defects are a leading cause of infant mortality in the U.S., with 20% of infant deaths occuring from complications related to birth defects. In 2013, the rate of IMBD was 121 cases per 100,000 live births (3). The birth defects with the highest rates of infant mortality include heart defects (29.0 cases per 100,000 live births), defects of the musculoskeletal system, limbs and integument (14.0 cases per 100,000 live births), defects of the genitourinary system (14.0 cases per 100,000 live births), trisomy 18 (11.6 cases per 100,000 live births), defects of the respiratory system (9.4 cases per 100,000 live births), and anencephaly (7.5 cases per 100,000 live births) (3). Several factors have been proposed to affect IMBD rates including severity of defect, preterm birth/gestational age (17), and SES measures (18).

Racial and ethnic disparities in IMBD rates have been reported for more than two decades (19-21), and the general trend is the following: infants of non-Hispanic white mothers have lower IMBD than Hispanic and non-Hispanic black mothers (22, 23). This is particularly true in the postneonatal period (>28 days and <1 year), and at the turn of the 21st century, these disparities were increasing (18). For instance, a study using national vital statistics data found that the racial and ethnic disparity of postneonatal IMBD increased significantly from 1989–1991 to 2000–2002 (18). Specifically, the racial and ethnic disparity in postneonatal IMBD rates increased over time by 17.7% for infants of non-Hispanic black mothers [1997–1998: adjusted rate ratio (aRR): 0.96, CI: 0.88–1.06; 2001-2002: aRR: 1.13, CI: 1.02–1.24] and by 14.4% for infants of Hispanic mothers (1997–1998: aRR: 1.04, CI: 0.95–1.15; 2001–2002: aRR: 1.19, CI: 1.09–1.30) compared to infants of non-Hispanic white mothers. In contrast, the aRR for neonatal (birth to <28 days) IMBD of infants born to non-Hispanic black and Hispanic mothers versus non-Hispanic white mothers was unchanged from 1989-1991 and 2000-2002. There are likely disparate underlying risk factors for neonatal versus postneonatal mortality; thus, the study highlights the importance of assessing IMBD rates by age of death. Elucidating the mechanisms of IMBD, and racial and ethnic disparities in IMBD rates in particular, will help target intervention strategies.

Complexities with Gestational age and IMBD

Infant morbidity and mortality in general is heavily influenced by preterm delivery or immaturity of the infant (24). Infants with birth defects are more often born at earlier gestational ages, i.e., preterm (<37 completed weeks of gestation) and smaller sizes at birth, i.e., small for gestational age (<10th percentile of birth weight for gestational age) than infants without birth defects (17, 25); thus, both immaturity and the birth defect may contribute to increased mortality of the preterm infant (24). Preterm birth is also associated with race or ethnic group; for instance, rates of preterm birth are higher in infants born to non-Hispanic black mothers when compared to non-Hispanic white mothers (26). Gestational age at birth then becomes a mediating variable between race and ethnicity and IMBD. A recent report attempted to disentangle the effects of racial and ethnic disparities in IMBD by stratifying on gestational age categories at birth (27). Using national vital statistics data from 2003–2006, the study reported that infants born to non-Hispanic black and Hispanic mothers delivered at 37–44 weeks had significantly higher neonatal IMBD (non-Hispanic black: aRR: 1.2, CI: 1.1–1.3; Hispanic: aRR: 1.2, CI: 1.1–1.2) than non-Hispanic whites; while infants of non-Hispanic black mothers delivered at 20–36 weeks had significantly lower neonatal IMBD (20–33 weeks gestational age: aRR: 0.6, CI: 0.5–0.6; 34–36 weeks: aRR: 0.8 ,CI: 0.7–0.9) than non-Hispanic whites (27). While this study is informative, adjusting for a mediating variable (via stratification) may produce bias when unmeasured confounders act on both the mediating factor (i.e., gestational age) and the outcome (i.e., IMBD) (24).

Socioeconomic Status and IMBD

IMBD rates are likely influenced by SES, though this has not been extensively studied in the U.S. SES is often measured by family income, parental education and/or occupation status; however, many other variables are also considered proxies of SES including access to care and insurance status. A recent study assessed the relationship between IMBD and SES by linking population-based data from four state birth defects surveillance programs to the 2000 U.S. Census to obtain census-tract-level SES indicators (28). This study found that lower community-level indicators of SES were associated with lower survival

of infants born with congenital heart defects (CHDs). Furthermore, in terms of racial and ethnic disparities, infants of non-Hispanic black and Hispanic mothers still had a higher mortality rates than infants of non-Hispanic white mothers even after adjustment for SES measures (28). Thus, SES may be correlated with risk of birth defects (14), and also risk for mortality from birth defects (28). However, varying SES definitions and measurements make interpreting study findings challenging (28). For instance, categorization of SES variables, measurement error in SES indicators, and incommensurate SES indicators may bias the result either towards or away from the null (29).

SES is often associated with access to perinatal and other health care (30). Access to care, particularly timely care and high quality care, is difficult to assess in studies using national vital statistics data. For example, the prenatal care variables on the birth certificate may be correlated with access to quality care, but those variables do not have high reliability or validity (31, 32). To our knowledge, no published studies have directly assessed the effects of access to health care, including prenatal care, on IMBD; however, it is the most commonly hypothesized reason for racial and ethnic disparities in IMBD (21, 27). As suggested by other types of health status disparities (e.g., mortality other than IMBD), racial and ethnic disparities in access to health care are not adequately explained by insurance, income or other measures of SES (33).

Insurance status is another measure of SES that is strongly related to health outcomes (34, 35). A population-based study in Florida found that uninsured infants had 3 times the risk of IMBD compared with those with private insurance [adjusted hazard ratio (AHR): 3.0, CI: 1.3–6.9] (36). In the neonatal period, infants with Medicaid had decreased mortality (AHR: 0.7, CI: 0.6–0.8) compared to infants with private insurance, but had a 30% increased risk in the postneonatal period (AHR: 1.3, CI: 1.1–1.6) (36). Another report suggested that infants without insurance may have decreased risk of mortality due to specialized care (37). Specifically, this study used the Kids' Inpatient Database and found that uninsured neonates with birth defects were 2.6 times more likely to be transferred to children's hospitals, while those with private insurance were retained at non-children's hospital (37). Children's hospitals are better

equipped to treat high risk neonates and therefore uninsured neonates may receive better care early in life than those with private insurance.

Substantial disparities in insurance status have been observed among racial and ethnic groups (34, 35), which complicates analyses of the associations between IMBD and insurance status. For instance, data from the Kaiser Commission on Medicaid and the Uninsured and the Urban Institute analysis of 2012 Annual Social and Economic Supplement (ASEC) Supplement to the Current Population Survey (CPS) (38) showed that for non-elderly non-Hispanic whites, 71% had employer/other private insurance, 15% were covered under Medicaid, and 13% were uninsured. For non-elderly Hispanics, 39% had employer/other private insurance, 30% were covered under Medicaid, and 32% were uninsured. For non-elderly non-Hispanic blacks, 40% had employer/other private insurance, 32% were covered under Medicaid, and 21% were uninsured.

Additional factors to consider in assessing IMBD rates include disparities in prenatal testing and pregnancy termination (13). Although dated, a study from the late 1990s reported that Hispanic and non-Hispanic black women were much less likely to undergo prenatal testing (39). This is important because if women are aware early in pregnancy that their infant had an anomaly affecting his/her chances of survival, they could choose to have an abortion [i.e., termination of pregnancy for fetal anomalies (TOPFA)]. After an abnormal result from prenatal testing, non-Hispanic black and Hispanic women were less likely to undergo TOPFA (40). There is also an issue of residence status of pregnant mothers. Only 17 states currently allow Medicaid funds to be used for medically necessary abortion beyond those allowed under the Hyde Amendment (41). A study using national vital statistics data combined with Nationwide Inpatient Sample data reported that black women were more likely than non-Hispanic white women to live in a state without Medicaid funding for TOPFA (65.8% compared to 59.6%, $p<0.001$) (42). Furthermore, among infants of non-Hispanic black mothers on Medicaid, birth in a state without such funding was associated with a 94% increased risk of anomaly-related death compared with birth in a state with funding (42). IMBD rates are affected by differential prenatal testing and TOPFA as a function of race or ethnic group or insurance status, because if the pregnancy is not terminated, and the infant dies,

the death will be counted as an IMBD. In summary, determining the underlying factors for the apparent racial and ethnic disparity in IMBD is complex and would benefit from a current assessment using national vital statistics data.

Conclusions

IMBD rates vary by gestational age, age of death, and race and ethnicity. Research on the association between SES and IMBD as well as the racial and ethnic disparities in IMBD rates is challenging because these variables are interrelated and their impact on IMBD are poorly understood. Racial and ethnic disparities are observed for many factors that influence IMBD rates, including birth defect prevalence and SES such as insurance status. However, a study on CHDs in Florida reported that racial and ethnic differences in IMBD were attenuated when adjusting for insurance status (36). Nevertheless, there currently are no published studies on the effect of insurance status on IMBD for all defects by race and ethnicity using national vital statistics data. Moreover, the CHD study used insurance status from hospital discharge data; this variable can be hard to interpret given the transition of insurance status before pregnancy until hospital discharge after delivery, which is often affected by the health of the infant (43). Health insurance payment source for delivery, however, is frequently used as a marker of maternal SES (e.g., 44, 45) and is not affecteed by the health of the infant. The 2003 revision of the Certificate of Live Birth requested for the first time information on the source of payment for the delivery, and national vital statistics data incorporated this variable in their datasets beginning in 2010. We will use national vital statistics data to study the association between payment source for delivery (Medicaid or private insurance) on IMBD and whether there is modification of the effect of payment source by race and ethnicity.

Table 1. **Racial and ethnic disparities in the prevalence of specific birth defects.**

Ref.	Study Type	Location, Years	Infants of Hispanic mothers compared to non-Hispanic white mothers	Infants of non-Hispanic black mothers compared to non-Hispanic white mothers
(12)	population-based	California, 1989-1997	-↓ CNS defects (other than anencephaly and spina bifida) -↑ chromosomal abnormalities -↓ genitourinary defects -↓ musculoskeletal system defects	-↑ CNS defects (other than anencephaly and spina bifida) -↑ chromosomal abnormalities -↓ genitourinary defects -↓ musculoskeletal system defects (other than polydactyly) -↑ respiratory system defects -↓ digestive system/cleft defects -↑ integument defects
(2)	population-based	Texas, 1999-2011*‡	-↑ CNS defects (anencephaly, spina bifida) -↑ chromosomal abnormalities (trisomy 21) -↑ CHDs (mVSD, PDA, except ↓ AVSD) - digestive system/cleft defects (↑ gastroschisis, ↓ Hirshsprung disease) -↓ genitourinary defects (hypospadias, epispadias) -↑ anotia/microtia	-↓ CNS defects (spina bifida) -↓ chromosomal abnormalities (trisomy 21) -↓ CHDs (mVSD, AVS) - digestive system/cleft defects (↓ gastroschisis, ↑ Hirschsprung disease) -↓ musculoskeletal system defects (craniosynotosis, hip dislocation) -↓ anotia/microtia
(46)	population-based	New York State 1983-2007	-↑ CNS defects (spina bifida without anencephalus, encephalocele) -↑ chromosomal abnormalities (trisomy 21) -↓ CHDs (AVSD, tetralogy of Fallot) - digestive system/cleft defects (↑ gastroschisis, ↓ esophageal atresia/tracheoesophageal fistula) -↓ musculoskeletal system defects (upper limb deficiencies)	- CNS defects (↑ encephalocele, ↓ anecephalus) -↓ CHDs (transposition of the great arteries) -↓ chromosomal abnormalities (trisomy 21) - digestive system/cleft defects (↑ omphalocele, ↓ orofacial clefts, ↓ gastroschisis, ↓ diaphragmatic hernia) - musculoskeletal system defects (↑ lower limb deficiencies, ↓ upper limb deficiencies)
(15)	population-based	Metropolitan Atlanta 1994-2005	-↑ chromosomal abnormalities (trisomies 13, 18, and 21) - CHDs (↑ ASD2, ↑ mVSD, ↓ coarctation of the aorta, ↓ AVSD) - digestive system/cleft defects (↑ diaphragmatic hernia, ↓ hypospadias, ↓ pyloric stenosis) -↓ musculoskeletal system defects (clubfoot, hip dislocation)	-↓ CNS defects (spina bifida) -↑ chromosomal abnormalities (trisomies 13, 18) - CHDs (↑ ASD2, ↓ mVSD, ↓ aortic stenosis) - digestive system/cleft defects (↑ Hirschsprung disease, ↓ pyloric stenosis, ↓ orofacial defects) - genitourinary defects (↑ cystic kidney, ↓ hypospadias) - musculoskeletal system defects (↑ polydactyly, ↓ hip dislocation, ↓ craniosynostosis, ↓ clubfoot without spina bifida)
(47)	population-based	multistate 1999-2007	-↑ CNS defects (anencephalus, spina bifida, encephalocele) -↑ chromosomal abnormalities (trisomies 18 and 21)	-↑ CNS defects (encephalocele) -↑ chromosomal abnormalities (trisomies 13 and 18) -↓ CHDs such as tetralogy of Fallot and AVSD

			-↓ CHDs (AVSD, AVS) -↓ digestive system/cleft defects (cleft palate without cleft lip) -↓ genitourinary defects (hypospadias) -↑ anotia/ microtia	-↓ digestive system/cleft defects (gastroschisis, alimentary tract defects, orofacial defects) -↑ musculoskeletal system defects (lower limb deficiency)
(16)*	population-based	multistate 2008-2012*†	-↑ CNS defects (anencephaly, spina bifida) -↑ chromosomal abnormalities (trisomy 21) -↑ CHDs (total anomalous pulmonary venous return) - genitourinary defects (↓ hypospadias, ↑ bladder exstrophy and renal agenesis/hypoplasia)	-↑ chromosomal abnormalities (trisomies 13 and 18) -↑ CHDs (interrupted aortic arch, AVSD, tetralogy of Fallot) - digestive system/cleft defects (↑ biliary atresia, ↑ omphalocele, ↓orofacial clefts, ↓ rectal and large intestinal atresia/stenosis) -↑ genitourinary defects (congenital posterior urethral valves)
(10)	population-based	National 1999-2001	-↑ CNS defects (anencephalus, spina bifida, encephalocele) -↑ chromosomal abnormalities (trisomy 21) -↓ CHDs (tetralogy of Fallot, hypoplastic left heart syndrome) - digestive system/cleft defects (↑ gastroschisis, ↓ cleft palate without cleft lip, ↓ esophageal atresia/tracheoesophageal fistula)	- chromosomal abnormalities (↑ trisomy 18, ↓ trisomy 21) -↑ CHDs (tetralogy of Fallot) -↓ digestive system/cleft defects (orofacial defects, gastroschisis, esophageal atresia/tracheoesophageal fistula) -↑ musculoskeletal system defects (lower limb reduction defects)
(11)	administra-tive (NIS)	National 2008	-↑ CHDs (ASD2) - genitourinary defects (↑ renal dysplasia, ↓ hypospadias) - digestive system/cleft defects (↑ omphalocele, ↑ gastroschisis, ↓ upper gastrointestinal anomaly)	-↓ CNS defects (neural tube) -↓ CHDs (mVSD) -↓ genitourinary defects (lower urinary tract obstruction, hypospadias) -↑ musculoskeletal system defects (hip dislocation, foot anomaly) -↓ digestive system/cleft defects (orofacial defects)

* Prevalence of one group compared to other two groups
↑ increased risk or prevalence of defect group
↓ decreased risk or prevalence of defect group
‡ non-Hispanic whites had an increased prevalence of cleft palate without cleft lip, craniosynostosis, and hypospadias, and a decreased prevalence of atrial septal defects compared to the other 2 groups)
† non-Hispanic whites had a higher average prevalence for aortic valve stenosis and coarctation of the aorta
Abbreviations: aortic valve stenosis, AVS; atrioventricular septal defect, AVSD; central nervous system, CNS; congenital heart defects, CHDs; muscular ventricular septal defect, mVSD; Nationwide Inpatient Sample, NIS; patent ductus arteriosus, PDA; secundum atrial septal defect, ASD2

Chapter 2. Medicaid is associated with higher rates of infant mortality attributable to birth defects when compared with private insurance

Approximately 3% of infants born in the United States (U.S.) have a birth defect (1, 2), and 20% of infant death results from complications of birth defects (3, 4). However, infant mortality attributable to birth defects (IMBD) is not equally distributed across gestational age (27), age of death (i.e., before or after the first 28 days of life) (18), or racial and ethnic groups. Much research over the past two decades has focused on racial and ethnic disparities in IMBD (19-21). In general, preterm infants of non-Hispanic black mothers have lower IMBD than Hispanic and non-Hispanic white mothers, while term infants of non-Hispanic white mothers have lower IMBD than those of non-Hispanic black and Hispanic mothers (27). The underlying factors contributing to these racial and ethnic disparities are unresolved. Reducing infant mortality in general, and from birth defects specifically, are goals of Healthy People 2020 (7); thus, identifying factors contributing to IMBD are in the forefront of public health research priorities.

Socioeconomic status (SES) is also hypothesized to affect IMBD (28), but how SES affects IMBD has not been well-studied. Insurance coverage, one proxy indicator for SES, is strongly related to better health outcomes (34, 35), including birth outcomes (48). Substantial disparities in insurance status have been observed among racial and ethnic groups (38). Recently, a population-based study on infant mortality from congenital heart defects (CHDs) in Florida reported that IMBD differed by insurance status and furthermore, that adjusting for insurance status attenuated racial and ethnic differences in survival (36). The purpose of our study is to increase our understanding of the association between SES and IMBD using national vital statistics data, which now include information on health insurance payment source for delivery, and to assess whether there is modification of this association by race and ethnicity.

Methods

This analysis used 2011–2013 National Center for Health Statistics (NCHS) period linked data from birth certificates and infant death certificates for all infants (<1 year) born to U.S. residents, which represents the most recent data available with information on payment source for delivery. Live births between January 2011 and December 2013 were eligible. A small percentage of infant death records could not be linked to their corresponding birth certificates (approximately 1.0–1.2%), and the linkage completion by state ranged from 95.5% to 100% with approximately 50% of states linking all of their records each year. To accommodate non-linked records, estimates of the number of infant deaths were weighted according to the percentage of records linked by state and age at death [(number of linked infant deaths + number of unlinked infant deaths)/number of linked infant deaths]. Records with unknown gestational age, gestational age <20 or >44 weeks and implausible combinations of gestational age and birthweight (49) were excluded (7.9% of infant deaths and 1.2% of live births). Deaths attributable to major birth defects included those whose underlying cause of death on the death certificate was classified as a birth defect according to the International Classification of Diseases, 10th Revision, codes Q00.0–Q99.9. Exceptions include the following: undescended testicles (Q53.1, 53.2, 53.9) or cardiovascular conditions that were not considered structural heart defects (Q27.0–Q28.9); preterm births with underlying cause of death considered normal conditions of prematurity [Q33.6: lung hypoplasia, Q21.1: persistent foramen ovale (PFO), and Q25.0: patent ductus arteriosus (PDA)]; and all neonatal deaths among term infants due to PFO and PDA.

All key variables used in these analyses are from birth certificates from states that adopted the 2003 revision of the U.S. Standard Certificate of Birth or from the U.S. Standard Certificate of Death (either the 1989 or 2003 revisions). The number of states implementing the 2003 revision of the birth certificate varied by year: 36 states plus the District of Columbia (DC) implemented it by 2011, 38 plus DC by 2012, and 41 plus DC by 2013 (Figure). Similarly, the number of states implementing the 2003 revision of the death certificate varied by year: 35 states implemented it by 2011, 40 by 2012, and 41 by 2013; however, the variables used from the death certificate are comparable across the two revisions. For

the analytic dataset, 88% of all live births and 87% of all infant deaths were eligible and represent the subset of states that adopted the 2003 revision of the birth certificate, which included information on payment source for delivery. A flow chart of the sample exclusions are presented in the Appendix (Figure A).

The following variables were extracted from the birth certificate: resident status at time of delivery, gestational age at delivery (categorized as preterm: <37 weeks; term: ≥37 weeks) based on mother's last menstrual period (LMP); maternal race and ethnicity combined (non-Hispanic white, non-Hispanic black, or Hispanic); maternal age (<20 years, 20-34 years, >34 years); maternal education (highest level completed at the time of delivery, categorized as less than 12^{th} grade; 12^{th} grade and higher); parity (categorized as 1, 2–3, and >3 previous live-born children); plurality (categorized as single or multiple); and the principal source of payment for delivery (Medicaid and private insurance). The following variables were extracted from the death certificate: birth defect classification [ICD-10 codes, (50)] for underlying cause of death, and age of death (neonatal: <28 days after birth; postneonatal: ≥28 days after birth but less than 1 year).

Analytic Methods

All analyses were performed in SAS v. 9.3. We estimated overall IMBD rates and rates stratified on the three largest race and ethnic groups (non-Hispanic white, non-Hispanic black, and Hispanic). Poisson regression was used to estimate the adjusted rate ratio (aRR) comparing neonatal and postneonatal IMBD among births covered by Medicaid with births covered by private insurance overall and stratified by prematurity status and by race and ethnicity. Potential confounding variables were chosen based on the literature and biological plausibility. Confounding was assessed by removing each variable one at a time from the full model (of all confounding variables) to determine whether they changed the effect estimate by greater than 5%. The final model included only maternal age as a confounder; maternal education was dropped because of substantial missing data. All analyses were repeated among singleton births only (Appendix Table A).

Results

Using the linked infant birth/death data from 2011–2013, the analysis included 8,584,190 live births and 46,729 infant deaths in the U.S. For 9,807 (20.9%) infant deaths, there was a birth defect noted as the underlying cause of death, resulting in an overall IMBD rate of 11.4 cases per 10,000 live births. The IMBD rate varied by race and ethnicity (10.7 cases per 10,000 live births for infants of non-Hispanic white, 13.5 cases for infants of non-Hispanic black, and 12.5 cases per 10,000 live births for infants of Hispanic mothers) and by prematurity (49.4 cases per 10,000 live births for preterm births and 6.7 cases per 10,000 live births for term births). Neonatal mortality attributable to birth defects accounted for 65% of all IMBD.

Characteristics of the analytic sample

In general, compared to deliveries covered by private insurance, deliveries covered by Medicaid had higher rates of IMBD within strata of each covariate and across strata of race and ethnic groups (Tables 2 and 3). For infant births covered by Medicaid, those of non-Hispanic black mothers had the highest rate of IMBD, followed by those of Hispanic mothers, and then those of non-Hispanic whites (Table 2). The racial and ethnic pattern was the same for infant births covered by private insurance at delivery, but the rates were lower for all groups (Table 3).

The association between maternal age and IMBD rates was similar across race and ethnic groups. Infants born to mothers over 34 years tended to have higher IMBD rates than the other age groups for births covered by Medicaid. This pattern was weaker for births covered by private insurance. For maternal education, IMBD rates were higher for mothers who did not graduate from high school, although the pattern was weaker for births covered by Medicaid compared with private insurance. For gestational age categories at birth, IMBD rates for preterm births were over 5 times higher than term births for deliveries covered by Medicaid and private insurance. The pattern of higher IMBD rates for women with more children was weaker for births covered by Medicaid compared with births covered by private insurance. In contrast, the association between higher plurality and IMBD rates was stronger for

births covered by Medicaid compared to births covered by private insurance, except among births covered by private insurance to non-Hispanic black mothers. There was no association between infant sex and IMBD rates except among infants of non-Hispanic white mothers, where male sex was associated with higher IMBD for both Medicaid and private insurance.

Association between IMBD and payment source for delivery

For infant births covered by Medicaid, the neonatal IMBD rate was 38% higher and the postneonatal IMBD rate was 63% higher compared with infant births covered by private insurance (Table 4). All race and ethnic groups had a similar pattern of increased IMBD for births covered by Medicaid compared with private insurance, although the postneonatal mortality adjusted rate ratio for non-Hispanic black mothers was slightly attenuated [aRR: 1.21, 95% confidence interval (CI): 1.01–1.46] compared with other groups.

Among preterm infants, the effect of payment source on IMBD differed by age of death (Table 4). In general, the neonatal IMBD rate was approximately 4 times higher than postneonatal mortality among preterm births within strata of payment sources for delivery (private insurance: 38.6 neonatal cases vs 8.1 postneonatal cases per 10,000 live births; Medicaid: 40.1 neonatal cases vs 11.7 postneonatal cases per 10,000 live births). In the neonatal period, payment source for delivery was not associated with IMBD rates for preterm infants. The postneonatal IMBD rate was approximately 50% higher for preterm infants whose delivery was paid for by Medicaid when compared with private insurance, and this difference was observed for each racial or ethnic group. Infants of Hispanic mothers had the highest aRR, showing the most discrepant IMBD rates between Medicaid and private insurance among the race and ethnic groups (aRR: 1.61, CI: 1.16–2.25).

Among term infants, the overall adjusted rate ratios for payment source were similar for neonatal and postneonatal mortality (Table 4; neonatal: aRR: 1.49, CI: 1.38–1.61; postneonatal: aRR: 1.57, CI: 1.44–1.72). When stratified by race and ethnic group, only term infants of non-Hispanic whites had an elevated adjusted rate ratio for postneonatal mortality compared with neonatal mortality (neonatal: aRR:

1.36, CI: 1.22–1.51; postneonatal: aRR: 1.56, CI: 1.37–1.78). There was an association between IMBD rates of term infants of non-Hispanic black mothers with payment source in the neonatal period, but not in the postneonatal period (neonatal: aRR: 1.31, CI: 1.06–1.61; postneonatal: aRR: 1.05, CI: 0.84–1.32). Among infants of Hispanic mothers, the adjusted rate ratio for neonatal mortality was elevated compared to postneonatal mortality (neonatal: aRR: 1.59, CI: 1.34–1.89; postneonatal: aRR: 1.49, CI: 1.22–1.93).

IMBD assessment of singleton births only

In a subanalysis, we removed 6.5% of the records that included twin births or higher plurality. When only singleton births were analyzed, the IMBD rates and adjusted rate ratios were similar to those shown in Table 4 for all births (Appendix Table A). Due to smaller sample sizes, there was decreased power for this analysis and thus the confidence intervals were wider.

Discussion

We were able to estimate IMBD rates by health insurance payment source for delivery for the majority of states for NCHS linked data from 2011–2013. We found that Medicaid was associated with increased IMBD rates compared to private insurance. When stratifying by prematurity status, we found that Medicaid was associated with increased IMBD rates among preterm infants for postneonatal mortality, but not neonatal mortality. Among term infants, we found that Medicaid was associated with increased IMBD rates for both neonatal and postneonatal mortality. One exception to this trend was postneonatal IMBD among term infants of non-Hispanic black mothers where IMBD rates between Medicaid and private insurance were similar. Our results suggest that the association between payment source and IMBD is minimally modified by race and ethnicity

In contrast to extensive literature on racial and ethnic disparities in IMBD, there have been few studies assessing IMBD rates by SES. To our knowledge only one study described the role of health insurance on survival of infants with CHDs, and it reported similar trends in higher IMBD rates of infants born under Medicaid compared to private insurance for the postneonatal period. However, that study

found lower IMBD rates for infants born under Medicaid compared with private insurance for the neonatal period [adjusted hazard ratio (AHR): 0.7, CI 0.6–0.8] (36). We did not find this pattern of lower IMBD rates for Medicaid in the neonatal period, but there were several differences between that study and ours. We used poisson regression and the previous study used Cox proportional hazard models, but these models should provide similar results given that the hazard model assumed constant hazard rates over a fixed time (the neonatal and postneonatal periods). The other study used data from a population-based cohort from Florida matched with death certificates, and they evaluated IMBD for CHDs only. In contrast, we used national vital statistics data and included all defects. Given that CHDs are the most common birth defect category, have the highest mortality rates, and as a group result in the highest costs (51), it seems possible that the relationship between insurance and IMBD could be different. We evaluated IMBD for births covered by Medicaid and private insurance (excluding other government insurance and the uninsured); the previous study evaluated all insurance categories. Finally, we used current data (2011–2013), while the older study used data from 1998–2007. This difference is important given the changes in health insurance over time; one change was the expansion of Medicaid over the last two decades, which has led to increasing Medicaid coverage during pregnancy (52).

Another important distinction between the previous study and the present study is that insurance status was assessed at different time-points: we used payment source at delivery, while the other study used insurance status from hospital discharge records. Mothers lose their Medicaid coverage sixty days after delivery, but infants often become Medicaid recipients. Thus, payment source at delivery may be a proxy for maternal SES (e.g., 44, 45), which does not reflect the health of the infant. In contrast, insurance status assignment at hospital discharge can be hard to interpret given the transition of insurance status before pregnancy until hospital discharge after delivery, which may be affected by the health of the infant (43). Infants whose deliveries were covered by private insurance may receive neonatal care under Medicaid (especially if the infant requires expensive care). Thus, our studies are essentially assessing different but related exposures.

Limitations to our analysis include the inability to examine other payment sources, including the uninsured, due to small numbers. We did not count deaths as IMBD if birth defects were listed only as a contributing cause of death. This likely resulted in an underestimation of the IMBD rate. Gestational age was broadly categorized into preterm and term infants; some misclassification may have occurred given that gestational age may be inaccurate on the birth certificate, and those inaccuracies are more common in women of low SES (53). Finally, although we attempted to adjust for possible confounders of the relationship between payment source and IMBD, there may be residual confounding by unmeasured factors.

Our analysis was the first to explore the role of insurance in the national vital statistics IMBD data. Strengths of our study include the fact that NCHS vital statistics data capture nearly all births and deaths in the U.S., and the data undergo stringent quality control. The source of payment variable has been assessed for data quality. Birth certificate information compared with medical records data among 8 hospitals in 2 states from 2010–2011 had good sensitivity for privately- and Medicaid-insured births. Data from self-pay and other categories were less reliable due to small numbers.

Insurance coverage and SES are correlated, with low income and unemployment often being associated with Medicaid or uninsured status. Our study may reflect the association between maternal SES, i.e., maternal income and employment, and IMBD, given the time that insurance information is being collected for the birth certificate. Examining the role of additional SES measures and of access to quality care could clarify the reason insurance status is associated with IMBD and help plan intervention strategies to reduce IMBD rates in the U.S.

Figure. Analytic sample is composed of states who adopted the 2003 revision of the birth certificate.

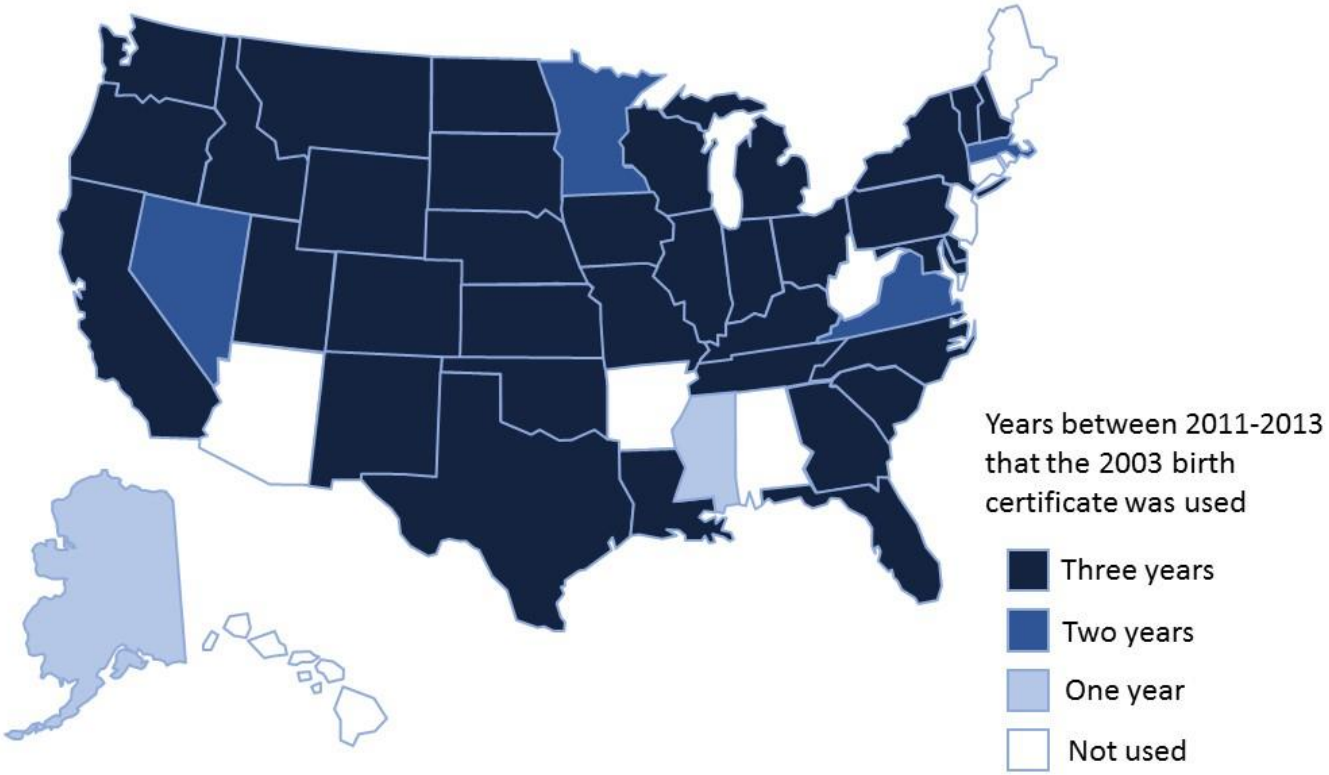

Table 2. Maternal and infant characteristics among infant deaths attributable to birth defects by race/ethnicity for births covered by Medicaid.

Characteristic	Non-Hispanic White			Non-Hispanic Black			Hispanic		
	IMBD (n)	Live births	IMBD/10,000 live births (95% CI)	IMBD (n)	Live births	IMBD/10,000 live births (95% CI)	IMBD (n)	Live births	IMBD/10,000 live births (95% CI)
Total number	2,082	1,617,825	12.9 (12.3-13.4)	1,246	889,764	14.0 (13.2-14.8)	1,921	1,412,422	13.6 (13.0-14.2)
Maternal age									
< 20 years	274	198,336	13.8 (12.2-15.5)	170	132,653	12.8 (10.9-14.7)	280	203,210	13.8 (12.2-15.4)
20-34 years	1,579	1,304,336	12.1 (11.5-12.7)	913	690,312	13.2 (12.4-14.1)	1,294	1,052,112	12.3 (11.6-13.0)
> 34 years	229	115,153	19.9 (17.3-22.5)	163	66,799	24.4 (20.7-28.1)	347	157,100	22.1 (19.8-24.4)
Maternal education									
<12th grade	487	322,197	15.1 (13.8-16.5)	319	208,935	15.3 (13.6-16.9)	902	606,414	14.9 (13.9-15.8)
≥12 grade	1,562	1,286,396	12.1 (11.5-12.7)	901	672,711	13.4 (12.5-14.3)	962	788,304	12.2 (11.4-13.0)
Missing	33	9232	36.1 (23.5-47.9)	26	8,118	32.3 (19.7-44.3)	57	17,704	32.2 (23.8-40.6)
Previous live births									
1	727	627,732	11.6 (10.7-12.4)	398	324,367	12.3 (11.1-13.5)	594	467,885	12.7 (11.7-13.7)
2-3	996	767,135	13.0 (12.2-13.8)	566	397,243	14.3 (13.1-15.4)	891	693,974	12.8 (12.0-13.7)
> 3	348	216,557	16.1 (14.4-17.8)	256	158,514	16.2 (14.2-18.1)	419	245,304	17.1 (15.4-18.7)
Missing	11	6401	17.3 (7.0-27.3)	26	9,640	27.1 (16.6-37.3)	17	5,259	33.0 (17.0-47.7)
Infant birth									
Preterm	1,018	184,034	55.3 (51.9-58.7)	617	146,952	42.0 (38.7-45.3)	867	158,573	54.7 (51.0-58.3)
Term	1,064	1,433,791	7.4 (7.0-7.9)	629	742,812	8.5 (7.8-9.1)	1,054	1,253,849	8.4 (7.9-8.9)
Plurality									
Single	1,968	1,573,372	12.5 (12.0-13.1)	1,176	857,394	13.7 (12.9-14.5)	1,840	1,383,043	13.3 (12.7-13.9)
Multiple	114	44,453	25.7 (20.9-30.4)	70	32,370	21.7 (16.6-26.7)	81	29,379	27.5 (21.6-33.6)
Infant sex									
Male	1,121	830,397	13.5 (12.7-14.3)	657	451,760	14.5 (13.4-15.7)	987	719,515	13.7 (12.9-14.6)
Female	961	787,428	12.2 (11.4-13.0)	589	438,004	13.5 (12.4-14.5)	934	692,907	13.5 (12.6-14.3)

95% confidence interval (CI): upper limit = (10,000 / # IMBD) × [# live births + (1.96 × √# live births)]; lower limit = (10,000 / # IMBD) × [# live births – (1.96 × √# live births)]

Table 3. Maternal and infant characteristics among infant deaths attributable to birth defects by race/ethnicity for births covered by private insurance.

Characteristic	Non-Hispanic White			Non-Hispanic Black			Hispanic		
	IMBD (n)	Live births	IMBD/10,000 live births (95% CI)	IMBD (n)	Live births	IMBD/10,000 live births (95% CI)	IMBD (n)	Live births	IMBD/10,000 live births (95% CI)
Total number	2,901	3,039,303	9.5 (9.2-9.9)	426	349,951	12.2 (11.0-13.3)	565	577,763	9.8 (9.0-10.6)
Maternal age									
< 20 yrs	85	63,980	13.3 (10.5-16.1)	25	21,117	11.9 (7.2-16.5)	40	27,787	14.3 (9.9-18.9)
20-34 yrs	2,142	2,377,547	9.0 (8.6-9.4)	271	254,939	10.6 (9.4-11.9)	363	432,021	8.4 (7.5-9.3)
> 34 yrs	674	597,776	11.3 (10.4-12.1)	130	73,895	17.6 (14.6-20.6)	162	117,955	13.8 (11.6-15.8)
Maternal education									
<12th grade	86	62,394	13.7 (10.9-16.7)	31	22,341	14.0 (9.0-18.8)	103	63,327	16.2 (13.1-19.4)
≥12 grade	2,774	2,962,782	9.4 (9.0-9.7)	385	324,589	11.9 (10.7-13.0)	440	508,674	8.7 (7.8-9.5)
Missing	41	14,127	29.3 (20.1-37.9)	10	3,021	33.5 (12.6-53.6)	22	5,762	38.7 (22.2-54.1)
Previous live births									
1	1,034	1,327,768	7.8 (7.3-8.3)	168	146,831	11.4 (9.7-13.2)	202	227,595	8.9 (7.7-10.1)
2-3	1,496	1,486,335	10.1 (9.6-10.6)	196	163,745	11.9 (10.3-13.6)	283	292,733	9.7 (8.5-10.8)
> 3	347	214,929	16.2 (14.4-17.8)	59	35,448	16.8 (12.4-20.9)	78	55,226	14.2 (11.0-17.3)
Missing	23	10,271	22.6 (13.2-31.5)	3	3,927	7.7 (0-16.3)	2	2,209	9.2 (0-21.6)
Infant birth									
Preterm	1,433	287,437	49.8 (47.3-52.4)	198	51,625	38.3 (33.0-43.7)	269	61,540	43.8 (38.5-48.9)
Term	1,468	2,751,866	5.3 (5.1-5.6)	228	298,326	7.7 (6.7-8.6)	296	516,223	5.7 (5.1-6.4)
Plurality									
Single	2,636	2,902,025	9.1 (8.7-9.4)	389	334,934	11.6 (10.5-12.8)	541	558,830	9.7 (8.9-10.5)
Multiple	265	137,278	19.3 (17.0-21.6)	37	15,017	24.8 (16.7-32.6)	25	18,933	12.9 (8.0-18.4)
Infant sex									
Male	1,557	1,558,872	10.0 (9.5-10.5)	209	177,890	11.8 (10.2-13.3)	276	294,611	9.4 (8.3-10.5)
Female	1,343	1,480,431	9.1 (8.6-9.6)	217	172,061	12.6 (10.9-14.3)	289	283,152	10.2 (9.0-11.4)

95% confidence interval (CI): upper limit = (10,000 / # IMBD) × [# live births + (1.96 × √# live births)]; lower limit = (10,000 / # IMBD) × [# live births – (1.96 × √# live births)]

Table 4. Rates[‡] of infant mortality attributable to birth defects for preterm and term infants by race/ethnicity and adjusted rate ratios[#] comparing infants for whom Medicaid was the payment source for delivery with those for whom private insurance was the payment source for delivery—United States, 2011–2013

Category	Total			Preterm			Term		
	Private Insurance	Medicaid	Adjusted Rate Ratio	Private Insurance	Medicaid	Adjusted Rate Ratio	Private Insurance	Medicaid	Adjusted Rate Ratio
Total *									
Neonatal	6.9	9.1	1.38 (1.31-1.45)	38.6	40.1	1.05 (0.98-1.12)	3.3	4.7	1.49 (1.38-1.61)
Postneonatal	2.8	4.3	1.63 (1.51-1.76)	8.1	11.7	1.49 (1.31-1.71)	2.2	3.3	1.57 (1.44-1.72)
non-Hispanic white									
Neonatal	7.0	8.9	1.29 (1.21-1.39)	41.9	43.7	1.03 (0.93-1.12)	3.3	4.5	1.36 (1.22-1.51)
Postneonatal	2.5	3.9	1.61 (1.45-1.80)	7.9	11.7	1.48 (1.22-1.80)	2.0	3.0	1.56 (1.37-1.78)
non-Hispanic black									
Neonatal	7.7	8.9	1.29 (1.12-1.49)	28.3	29.8	1.13 (0.93-1.37)	4.1	4.8	1.31 (1.06-1.61)
Postneonatal	4.5	5.1	1.21 (1.01-1.46)	10	12.2	1.41 (1.02-1.94)	3.6	3.7	1.05 (0.84-1.32)
Hispanic									
Neonatal	6.9	9.3	1.42 (1.27-1.59)	36.6	43.3	1.21 (1.04-1.41)	3.4	5.0	1.59 (1.34-1.89)
Postneonatal	2.9	4.3	1.55 (1.30-1.84)	7.2	11.4	1.61 (1.16-2.25)	2.4	3.4	1.49 (1.22-1.83)

[‡] Rate per 10,000 live births of U.S. residents from 2011-2013 including states that used the 2003 version of the birth certificate. This varied by year: for 2011, 36 states were included; in 2012, 38 states were included; in 2013, 41 states were included in the analysis.

[#] Adjusted for maternal age

***** The 'Total' category includes Hispanics, non-Hispanic blacks, non-Hispanic whites, non-Hispanic other races and unknown/ unstated race/ethnicity

Neonatal, mortality for infants with birth defects <28 days of age; Postneonatal, mortality for infants with birth defects ≥28 days of age but <1 year

Chapter 3. Conclusions

Issues with infant mortality attributable to birth defects (IMBD) rates as a result of data source

The assessment of IMBD rates is complicated by the fact that there are disparities in participation in prenatal testing and pregnancy termination, particularly among racial and ethnic groups (13). This is important because if women knew early in pregnancy that their infant had a birth defect affecting his/her chances of survival, they may have elective abortions [i.e., termination of pregnancy for fetal anomalies (TOPFA)]. Moreover, only 17 states currently allow Medicaid funds to be used for medically necessary abortion beyond those allowed under the Hyde Amendment (41). Thus, without information about TOPFA, it is difficult to determine whether our results are a reflection of differential rates of abortion, with Medicaid participants having decreased rates of abortion and thus higher IMBD. In other words, it is possible that the pregnancies with the most severe defects may have been terminated for mothers who could afford the procedure. Future studies that include information on TOPFA would be extremely helpful in determining whether our results are affected by differential termination rates.

Further, there are racial and ethnic disparities in prenatal testing and TOPFA rates, which affect our findings. For instance, after an abnormal result from prenatal testing, non-Hispanic black and Hispanic women were less likely to undergo TOPFA than non-Hispanic white women (40). A study using both national vital statistics data and Nationwide Inpatient Sample data suggests that black women were more likely than white women to live in a state that does not allow Medicaid funding for TOPFA (65.8% compared to 59.6%, $p<0.001$) (42). Furthermore, among infants of black mothers on Medicaid, birth in a state without such funding was associated with a 94% increased risk of IMBD (48% increased risk of IMBD for white mothers) compared with birth in a state with funding (42). Our study reports minimal modification of the association between payment source for delivery and IMBD by race or ethnic group; thus, even though infants of non-Hispanic black mothers experienced higher IMBD rates overall compared with infants of Hispanic and non-Hispanic white mothers, they had similar patterns of IMBD rates across insurance payment source (i.e., births covered by Medicaid had higher IMBD rates than those

covered by private insurance). For confidentiality, beginning in 2005, National Center for Health Statistics public use data do not contain information on state of residence, which prevents studies using national linked data to compare differences in the association between IMBD and insurance status by race and ethnicity across states.

Differences in the association between IMBD and payment source by age of death

Previous studies of IMBD have reported minimal racial and ethnic disparities in IMBD rates in the neonatal period, but more pronounced differences in IMBD rates in the postneonatal period. It is possible that when neonates are very sick, they receive a high standard of care that is not affected by insurance coverage. For instance, quality care in the pediatric intensive care unit (ICU) was not shown to be affected by race and ethnicity or insurance carrier in a prospective study of pediatric ICUs in three children's hospitals (54). Similarly, we did not observe differences in IMBD by payment source (Medicaid/private insurance) among preterm births during the neonatal period when they were most likely to be treated in the neonatal ICU, but did observe differences for preterm infants during the postneonatal period and differences for term births, which are less likely to be admitted to the neonatal ICU. Care in the postneonatal period may be more varied depending on insurance status. For instance, a review of published studies reported that children on public insurance were shown to have less access to specialty care (55) and received later treatments (such as surgical repair) (56) compared to privately insured children.

Future directions

The current study was conducted using data prior to the Affordable Care Act and wide-spread Medicaid expansion. Studies are being published on the effects of these changes in health insurance programs, and specifically on birth outcomes. For instance, Medicaid's statewide enhanced prenatal and postnatal care program, the Maternal Infant Health Program (MIHP) in Michigan, reported infants with MIHP had reduced mortality risk in the first year of life compared with matched non-participants (57).

Moreover, children with birth defects are eligible for early intervention services in many states. In Georgia, there are several programs that provide services to families of infants with birth defects including Babies Can't Wait, Children's Medical Services, and Children 1st. Thus, children with birth defects are eligible for specialized services, which should reduce morbidity and mortality, regardless of insurance status. However, if payment source for delivery is a marker of maternal SES, there could be many reasons why these services would be under-utilized in Medicaid recipients, such as financial constraints on traveling to specialized care and providing supportive care, among others. Conducting this analysis in the next several years as the Affordable Care Act continues, and more data are available on payment source for delivery, insurance status in the postneonatal period, and additional markers of SES, could help identify areas of intervention to reduce IMBD rates.

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Appendix

Figure A. Flow chart of included/excluded samples for the 2011-2013 linked birth/infant death data. Excluded conditions are the following: cardiovascular conditions that were not considered structural heart defects; preterm births with underlying cause of death considered normal conditions of prematurity [lung hypoplasia, persistent foramen ovale (PFO), and patent ductus arteriosus (PDA)]; and all neonatal deaths among term infants due to PFO and PDA. Abbreviations: BD, birth defects; GA, gestational age; UCOD, underlying cause of death.

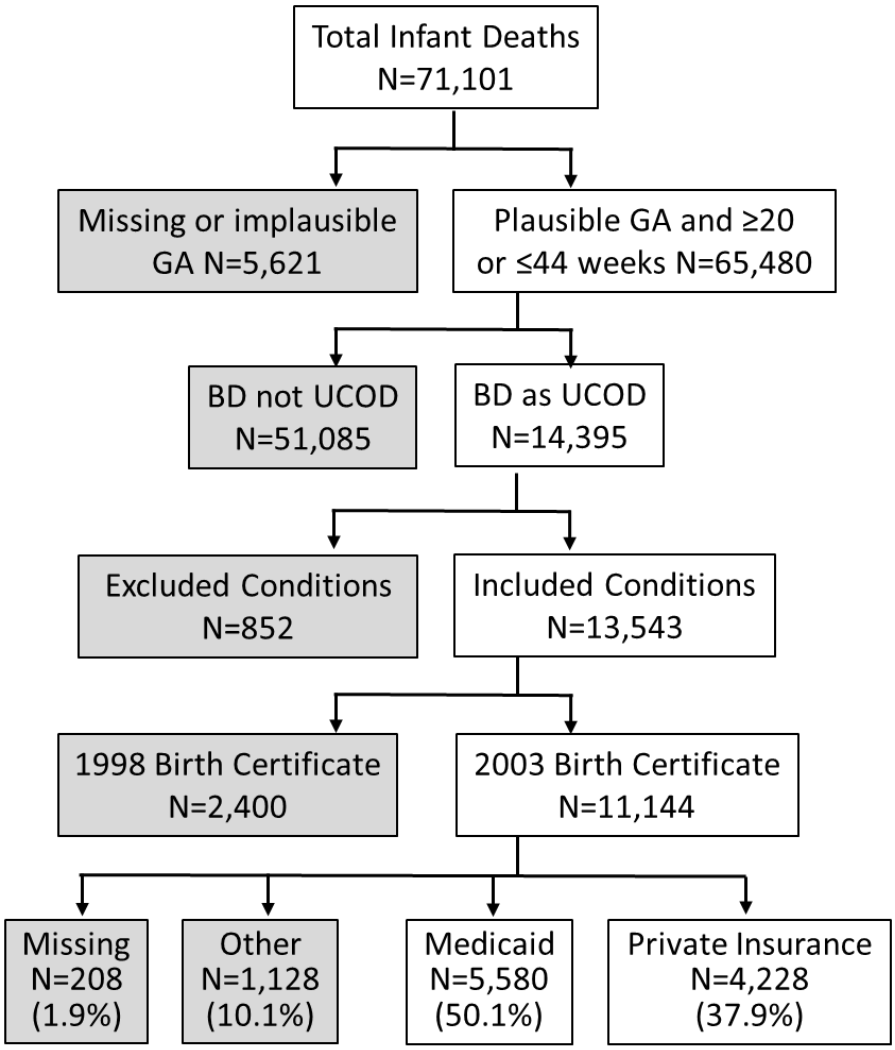

Table A. Rates of IMBD by Birth Defect of Preterm Infants and Adjusted Rate Ratios[§] comparing infants of Mothers who used Medicaid with those of Mothers who used Private Insurance to pay for delivery. Sensitivity analysis for singleton births only.

Category	Preterm			Term		
	Private Insurance	Medicaid	Adjusted Rate Ratio	Private Insurance	Medicaid	Adjusted Rate Ratio
Total *						
Neonatal	44.5	42.0	0.96 (0.90-1.03)	3.2	4.7	1.51 (1.40-1.63)
Postneonatal	9.0	12.3	1.41 (1.22-1.63)	2.2	3.2	1.56 (1.42-1.71)
non-Hispanic white						
Neonatal	49.4	45.9	0.93 (0.84-1.02)	3.3	4.5	1.38 (1.24-1.54)
Postneonatal	9.0	12.5	1.40 (1.14-1.72)	2.0	2.9	1.54 (1.35-1.76)
non-Hispanic black						
Neonatal	29.9	31.4	1.15 (0.94-1.42)	4.0	4.8	1.33 (1.07-1.65)
Postneonatal	9.5	12.7	1.54 (1.08-2.20)	3.5	3.6	1.05 (0.83-1.33)
Hispanic						
Neonatal	41.6	45.1	1.11 (0.95-1.30)	3.3	5.0	1.61 (1.36-1.92)
Postneonatal	8.2	11.8	1.47 (1.04-2.07)	2.4	3.3	1.46 (1.19-1.79)

[‡] Rate per 10,000 live births of U.S. residents from 2011-2013 including states that used the 2003 version of the birth certificate. This varied by year: for 2011, 36 states were included; in 2012, 38 states were included; in 2013, 41 states were included in the analysis.

[#] Adjusted for maternal age

***** The 'Total' category includes Hispanics, non-Hispanic blacks, non-Hispanic whites, non-Hispanic other races and unknown/ unstated race/ethnicity

Neonatal, mortality for infants with birth defects <28 days of age; Postneonatal, mortality for infants with birth defects ≥28 days of age but <1 year