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BRIEF COMMUNICATION

Fetal Growth Among Infants With Congenital Heart Defects by Maternal Race/Ethnicity

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PURPOSE: Congenital heart defects (CHDs) are the most prevalent birth defects. Infants with CHDs more often are small-for-gestational age (SGA) than infants without CHD; however, little is known about racial/ethnic variations in prevalence of SGA or large-for-gestational age (LGA) for infants born with CHDs. This study determined the risk of SGA and LGA for non-Hispanic (NH)-black and Hispanic infants with CHDs.

METHODS: Data from the Florida Birth Defects Registry were used in a retrospective cohort study of 10,027 live-born infants to resident NH-White, NH-Black, and Hispanic women ages 15–49 years from January 1, 1998, to December 31, 2003, and diagnosed with 11 CHDs. Defect-specific odds ratios and 95% confidence intervals were computed for risk of SGA and LGA by race/ethnicity and adjusted for covariates using multinomial logistic regression.

RESULTS: After adjusting for covariates, we found there were no statistically significant racial/ethnic differences in risk of SGA. However, NH-Blacks with ventricular septal defect had increased risk of LGA and NH-Blacks with tetralogy of Fallot had decreased risk of LGA compared to NH-Whites.

CONCLUSIONS: Very few racial/ethnic differences in fetal growth are present among infants with CHD. Further elucidation of the factors involved in fetal growth and the impact of CHD itself on fetal development is needed.

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KEY WORDS: Birth Defects, Black, Congenital Heart Defects, Fetal Growth, Hispanic, Infants, Intrauterine Growth Restriction, Large-for-Gestational Age, Small-for-Gestational Age.

INTRODUCTION

Congenital heart defects (CHD), malformation of the heart and major blood vessels between the third and eighth week gestation, are the most prevalent birth defects, with an estimated annual prevalence of 6–12 affected infants per 1000 live births (1, 2) and remain the leading cause of infant mortality from birth defects (3). But despite advances in the diagnosis and treatment of CHDs and our understanding embryonic and fetal development, the etiology of CHD remains unclear, with only 5–10% of CHDs attributed to chromosomal abnormalities and single-gene defects (4).

Infants with CHDs are at increased risk of infant morbidity and mortality, often requiring invasive surgical and medical interventions to repair or manage the malformation. Compared with infants without birth defects, infants with CHD have a 1.8 to 3.6 times increased risk of being small-for-gestational age (SGA; <90th percentile for birthweight-gestational age curve) than infants without CHDs (5, 6). Infants with CHD who also are SGA may have increased risk of morbidity during childhood because size at birth is one of the factors that determines medical and surgical outcomes (7, 8).

Although differences in fetal growth between infants with CHDs and infants without CHDs has been established, it is unclear whether there are racial/ethnic differences in fetal growth among infants with CHD. It is well established that black infants without birth defects are more likely to be born SGA (9), whereas Hispanic infants without birth defects have SGA rates similar to non-Hispanic (NH) white infants (10). Furthermore, black infants have an increased risk of other adverse infant outcomes, such as preterm birth and low birth weight, compared with NH-white infants (11, 12). Previous research also has shown that black infants with CHDs have increased risk of preterm birth compared with NH-white infants (13). Hence, the pattern of racial/ethnic differences in fetal growth may persist among infants with CHDs. Thus, the purpose of this study was to determine whether there were racial/ethnic differences in the distribution of fetal growth and the risk of SGA and large-for-gestational age (LGA) among infants with CHD.

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Selected Abbreviations and Acronyms

CHD = congenital heart defects
SGA = small-for-gestational age
NH = non-Hispanic
LGA = large-for-gestational age
FBDR = Florida Birth Defects Registry
OR = odds ratio
CI = confidence interval
AGA = appropriate-for-gestational age
IRB = Institutional Review Board

METHODS

Case Ascertainment

We used data from the Florida Birth Defects Registry (FBDR), to conduct a retrospective cohort study of live births. The FBDR has monitored birth defects in Florida since 1998 by merging data from birth vital records, hospital discharge databases for both inpatients and ambulatory patients, and from programs administered by the Florida Department of Health's Children's Medical Services. Infants are included in the FBDR if they are live-born to a Florida resident and have an included birth defect as coded by the *International Classification of Diseases, Ninth edition, Clinical Modification* diagnosis coding system.

Classification of Congenital Heart Defects

All live-born, singleton infants diagnosed with a CHD in the first year of life, born between January 1, 1998, and December 31, 2003, to Florida resident women ages 15 to 49 years were eligible for inclusion. CHDs were classified with the use of select *International Classification of Diseases, Ninth edition, Clinical Modification* diagnosis codes in the 745.00–747.99 range and affected infants were placed into four categories. Conotruncal CHDs included common truncus arteriosus (745.0), transposition of the great arteries (745.10–745.12 or 745.19), and tetralogy of Fallot (745.2). Right obstructive CHDs included tricuspid valve atresia and stenosis (746.1), pulmonary valve atresia and stenosis (746.01 or 746.02), and Ebstein's anomaly (746.2). Left obstructive CHDs included hypoplastic left heart syndrome (746.7), aortic valve atresia and stenosis (746.3), and coarctation of the aorta (747.10). Septal CHDs included ventricular septal defect (745.4), and atrioventricular septal defect (745.60, 745.61, or 745.69).

Study Variables

Data on gestational age, infant birth weight, maternal race/ethnicity, and potential confounders such as maternal age, maternal education, parity, maternal prenatal tobacco use, and infant sex were taken from the Florida Office of Vital Statistics birth certificate. Maternal race/ethnicity was based on maternal self-report and was first grouped by ethnicity (Hispanic or NH) and the NH group was subdivided into white, black, and other. Maternal age was categorized as 15–19, 20–29, 30–39, and 40–49 years and maternal education was group, based on years of education, as < 12, 12, and 13+ years. Fetal growth was determined using race-specific growth curves (14). Categories of fetal growth were defined as SGA (birth weights less than 10th percentile), appropriate-for-gestational age (AGA; birth weights between the 10th and 90th percentiles), and LGA (birth weights greater than 90th percentile).

We excluded 966 (9.6%) infants because they were not from a singleton birth (3.9%), had a maternal race/ethnicity designated as "Other" (2.2%), or had a maternal age less than 15 or greater than 49 years (0.3%). Infants missing data on fetal growth indices (2.5%) gestational age (1.8%), maternal education (0.4%), parity (0.03%), birth weight (0.03%), or prenatal maternal smoking (0.02%) also were excluded (numbers do not add up to 966 because some infants had more than one exclusion).

Data Analysis

Univariate analyses were used to calculate descriptive statistics, odds ratios (OR), and 95% confidence intervals (CI) to evaluate the distribution of study variables and crude associations. In multivariate multinomial logistic regression analyses, ORs and 95% CIs were used to determine the independent effects of ethnicity on risk of SGA and LGA (compared with AGA) while adjusting for maternal age, maternal education, maternal prenatal smoking, parity, and infant sex. We calculated separate multivariate multinomial regression models for each type of CHD. All statistical tests performed were two-sided and declared at the 5% significance level. The Office of Research Integrity and Compliance, Institutional Review Board (IRB) at the University of South Florida approved the study. The Florida Department of Health IRB approved the use of data from Florida birth records and FBDR data.

RESULTS

During the study period, 1,216,142 infants were live births to Florida residents, of these 10,027 had at least one of the selected CHD. After exclusions, our final study population included 9061 infants with CHD; 4851 (53.5%) were born to NH-white, 2004 (22.1%) to NH-black, and 2206 (24.4%) to Hispanic women. As seen in Table 1, we found no statistically significant racial/ethnic differences in the distribution of intrauterine growth for any type of CHD. Table 2 displays adjusted ORs and 95% CI for risk of SGA and LGA for infants with CHD by race/ethnicity. Although there were several ORs greater than or less than the null value (1.0), very few were statistically significant, indicating