Racial/Ethnic Disparities in Risk of Early Childhood Mortality Among Children With Congenital Heart Defects

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*Pediatrics* published online Apr 18, 2011;
DOI: 10.1542/peds.2010-2702

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Racial/Ethnic Disparities in Risk of Early Childhood Mortality Among Children With Congenital Heart Defects

WHAT’S KNOWN ON THIS SUBJECT: Congenital heart defects (CHDs) are the leading cause of death among all infants with birth defects. Mortality and survival vary greatly and are influenced by several factors including length of follow-up, phenotype, number of co-occurring, and severity of defects.

WHAT THIS STUDY ADDS: Numerous reports have described growing disparities in infant and childhood mortality between non-Hispanic white and minority children. However, little is known about survival among minority children with CHDs. These data demonstrate racial/ethnic disparities in early childhood survival among children with CHDs.

abstract

BACKGROUND: Infants with congenital heart defects (CHDs) have increased risk of childhood morbidity and mortality. However, little is known about racial/ethnic differences in early childhood mortality.

PATIENTS AND METHODS: We conducted a retrospective cohort study with data from the Texas Birth Defect Registry on 19,530 singleton, live-born infants with a CHD and born January 1, 1996, to December 31, 2003, to non-Hispanic (NH) white, NH black, and Hispanic women. Texas Birth Defect Registry data were linked to Texas death records and the National Death Index to ascertain deaths between January 1, 1996, and December 31, 2005. Kaplan-Meier survival estimates were computed, and hazard ratios (HRs) and 95% confidence intervals (CIs) were calculated from multivariable Cox-proportional hazard regression models to determine the effect of maternal race/ethnicity on mortality for selected CHD phenotypes.

RESULTS: After adjusting for covariates, compared with NH white children, NH black children had increased early childhood mortality risk for transposition of the great arteries (HR: 2.04 [95% CI: 1.40–2.97]), tetralogy of Fallot (HR: 1.85 [95% CI: 1.09–3.12]), pulmonary valve atresia without ventricular septal defect (VSD) (HR: 2.60 [95% CI: 1.32–5.12]), VSD (HR: 1.56 [95% CI: 1.19–2.03]), and atrial septal defect (HR: 1.34 [95% CI: 1.08–1.66]). Hispanic children had higher mortality risk for pulmonary valve atresia without VSD (HR: 1.76 [95% CI: 1.06–2.91]) and hypoplastic left heart syndrome (HR: 1.51 [95% CI: 1.13–2.02]).

CONCLUSIONS: We provide evidence that supports racial/ethnic disparities in early childhood mortality among infants with CHDs. Identifying infants with the greatest risk of early childhood mortality will facilitate development of interventions and policies to mitigate these risks. Pediatrics 2011;127:e1128–e1138

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KEY WORDS congenital heart defects, race/ethnicity, mortality, survival, childhood

ABBREVIATIONS
CHD—congenital heart defect
NH—non-Hispanic
TBDR—Texas Birth Defects Registry
TGA—transposition of the great arteries
VSD—ventricular septal defect
HLHS—hypoplastic left heart syndrome
ASD—atral septal defect
RUCA—rural urban commuting area
HR—hazard ratio
CI—confidence interval

www.pediatrics.org/cgi/doi/10.1542/peds.2010-2702
doi:10.1542/peds.2010-2702
Accepted for publication Mar 2, 2011
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FINANCIAL DISCLOSURE: The authors have indicated they have no financial relationships relevant to this article to disclose.

COMPANION PAPERS: Companions to this article can be found on pages e1120 and e1133 and online at www.pediatrics.org/cgi/doi/10.1542/peds.2010-2608 and www.pediatrics.org/cgi/doi/10.1542/peds.2011-0182.
Birth defects are the leading cause of infant mortality in the United States. Congenital heart defects (CHDs), abnormalities in the formation of the heart and major vessels, are the most common of all birth defects (annual prevalence: 6–12 affected infants per 1000 live births1–3) and are the leading cause of death among all infants with birth defects. Although most infants with CHDs who are diagnosed early benefit from successful surgical repair, mortality rates can be quite high; survival rates vary greatly and are influenced by several factors including length of follow-up, phenotype, number of co-occurring defects, severity of defect, and also maternal race/ethnicity.4–13 Numerous study reports14–18 and a recent technical report19 have described growing disparities in health and health care between non-Hispanic (NH) white and minority children, especially in infant and childhood mortality. Minority children are more likely to receive lower-quality general pediatric care and have increased risk of death in infancy.19 Racial/ethnic disparities in health and health care also exist among children with CHDs.20 Minorities now comprise ~43% of all children in the United States, which is a 58% increase since 1990,19 and conservative projections estimate that minorities will comprise half of US children by 2040. Therefore, even modest decreases in childhood survival rates or increases in mortality rates among minority children will cause greater burden on the health care system and adversely affect the lives of families. However, little is known about survival experiences among minority children with CHDs. We identified 4 studies in the peer-reviewed literature that were cross-sectional studies based on single-year administrative data21,22 or national death-certificate data23,24; racial/ethnic disparities in mortality

rates for children with CHDs were reported from all 4 of these studies. However, none was a population-based cohort study or used birth defects registry data. We conducted a population-based cohort study to determine survival rates and risk of early childhood mortality according to maternal race/ethnicity among children with CHDs.

**PATIENTS AND METHODS**

**Study Design**

We conducted a retrospective cohort study with data from the Texas Birth Defects Registry (TBDR), which is maintained by the Birth Defects Epidemiology and Surveillance Branch at the Texas Department of State Health Services. The TBDR is a population-based, active surveillance system that collects information on all structural and chromosomal birth defects diagnosed within 1 year after delivery. The TBDR began birth defects surveillance from 1996 to 1998 in increasingly larger areas of the state (35% of all resident live births in 1996, 56% in 1997, and 85% in 1998) and expanded to statewide coverage in 1999. The TBDR staff routinely visit delivery units, pediatric hospitals, and birthing centers where affected children are delivered or treated to identify and collect information for the registry.

To obtain additional demographic information, TBDR staff link registry cases to Texas birth and fetal death certificates filed with the Vital Statistics Unit of the Texas Department of State Health Services on the basis of the infant’s and mother’s names and dates of birth.24 For 1996–2003 deliveries, the registry linked ≥99.0% of live-born case-infants to their Texas birth certificates. TBDR case-infants are also linked to their Texas death certificates through the death-to-birth certificate matching routinely performed by the Texas Vital Statistics Unit.

**Study Population**

We selected all live-born singleton infants diagnosed with a CHD within the first year of life and born January 1, 1996, to December 31, 2003, to NH white, NH black, or Hispanic women. CHDs were classified by using the British Pediatric Association extension of the International Classification of Diseases, Ninth Edition, Clinical Modification (ICD-9-CM) coding system, as modified by the Division of Birth Defects and Developmental Disabilities of the Centers for Disease Control and Prevention and by the TBDR. CHDs were categorized as 1 of 4 types of defects: conotruncal; right obstructive; left obstructive; or septal. Conotruncal CHDs included common truncus (745.000–745.010), transposition of the great arteries (TGA) (745.100–745.190), and tetralogy of Fallot (745.200–745.290, 745.300–745.400, or 745.000 with 745.400–745.490). Right obstructive CHDs included pulmonary valve atresia without ventricular septal defect (VSD) (746.000 without 745.400–745.490), pulmonary valve stenosis (746.010), anomalies of the tricuspid valve (746.100), and Ebstein anomaly (746.200). Left obstructive CHDs included hypoplastic left heart syndrome (HLHS) (746.700), aortic valve stenosis (746.300), and coarctation of the aorta (747.100–747.190). Septal CHDs included VSD (745.400–745.490), atrial septal defect (ASD) (745.510–745.590), and atrioventricular septal defect (745.600–745.690). Infants who had both pulmonary valve atresia and VSD were classified as having tetralogy of Fallot.

**Study Variables**

Information on medical, pregnancy, and sociodemographic factors was obtained from birth certificates and medical records. We included gender, gestational age (categorized as 20–36 or ≥37 completed weeks), birth weight...
Ascertainment of Death

We linked cases of CHDs to Texas birth and death records to ascertain in-state deaths; we used the National Death Index at the National Center for Health Statistics to ascertain out-of-state deaths. Children were considered deceased if noted as deceased in Texas death records, the National Death Index, or the TBDR.

Statistical Analysis

We calculated descriptive statistics for the main study variables and covariates. Survival time was calculated as the time from date of birth to date of death; for infants who were not identified as deceased, survival time was calculated as the time from date of birth to the end of the study period (December 31, 2005). We computed Kaplan-Meier survival curves to describe the pattern of survival within early childhood for each type of CHD. Using proportional hazards regression (PHREG procedure in SAS 9.2 [SAS Institute, Inc, Cary, NC]), we calculated crude and adjusted hazard ratios (HRs) and 95% confidence intervals (CIs) to determine the effect of maternal race/ethnicity on early childhood survival; NH white children were used as the referent group.

The following variables were significantly associated with survival and were included as potential confounders in our models: maternal age; education; residence at delivery variables; infant gender; number of co-occurring defects; and gestational age/birth weight combinations. The statistical significance level was P < .05 for main effects.

The study was approved by the institutional review board at the University of South Florida, the National Death Index, and the Texas Department of State Health Services, including the use of data from Texas vital records and the TBDR.

RESULTS

During the study period, 2.35 million singleton live-born infants were born in the areas covered by the TBDR; 20 405 had at least 1 of the selected CHDs. We excluded infants not born to an NH white, NH black, or Hispanic mother (n = 529), diagnosed with trisomy 13 or 18 (n = 343), or children deceased but missing date of death (n = 15), which resulted in an unduplicated total of 875 excluded children (4.3%) (numbers do not add up because 12 children had >1 reason for exclusion). Our final study population included 19 530 children, 1572 (8.0%) of whom died before 1 year of age and 1826 (9.3%) of whom died during follow-up. Of these children, 67 (3.7%) did not have a death-certificate number, and 1 child had a death-certificate number that did not link to a death certificate. Thus, 1758 children had cause-of-death information; 840 (47.8%) had a CHD as the underlying cause of death, and 1046 (59.5%) had a CHD as any cause of death (includes the 840).

Approximately 26.7% of the study children were born preterm, and 21.2% were born at very low or low birth weight (Table 1). Of those who died during the study period, 39.9% were born preterm, 15.6% were born at very low birth weight, and 23.6% were born at low birth weight. Overall, after adjusting for covariates, NH black children with CHDs had a 32% increased risk of early childhood mortality compared with affected NH white children (HR: 1.32 [95% CI: 1.14–1.54]). In contrast, Hispanic children with CHDs had no increased risk overall of early childhood mortality compared with NH white children (HR: 0.96 [95% CI: 0.85–1.08]) (Table 2).

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>NH White (n = 15,492)</th>
<th>NH Black (n = 3,566)</th>
<th>Hispanic (n = 3,566)</th>
<th>Total (n = 22,624)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mothers</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal age</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;20 y</td>
<td>712 (10.0)</td>
<td>83 (13.1)</td>
<td>334 (17.8)</td>
<td>1,800 (17.1)</td>
</tr>
<tr>
<td>20–29 y</td>
<td>5,572 (50.0)</td>
<td>321 (50.6)</td>
<td>986 (52.5)</td>
<td>6,825 (53.3)</td>
</tr>
<tr>
<td>30–39 y</td>
<td>2,500 (36.3)</td>
<td>210 (33.1)</td>
<td>494 (26.3)</td>
<td>2,875 (26.5)</td>
</tr>
<tr>
<td>≥40 y</td>
<td>265 (3.7)</td>
<td>20 (3.2)</td>
<td>65 (3.5)</td>
<td>312 (3.0)</td>
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<td>Maternal education</td>
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<tr>
<td>&lt;High school</td>
<td>1,064 (14.9)</td>
<td>108 (17.0)</td>
<td>375 (20.0)</td>
<td>1,458 (16.6)</td>
</tr>
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<td>High school</td>
<td>2,231 (31.3)</td>
<td>200 (31.5)</td>
<td>757 (40.3)</td>
<td>2,858 (33.6)</td>
</tr>
<tr>
<td>&gt;High school</td>
<td>3,711 (55.8)</td>
<td>309 (48.7)</td>
<td>686 (35.8)</td>
<td>4,692 (43.8)</td>
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<td>Residence in a border county</td>
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<td></td>
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<tr>
<td>Yes</td>
<td>206 (2.9)</td>
<td>26 (4.1)</td>
<td>15 (0.8)</td>
<td>347 (3.0)</td>
</tr>
<tr>
<td>No</td>
<td>6,833 (97.1)</td>
<td>608 (95.9)</td>
<td>1,864 (99.2)</td>
<td>9,505 (97.0)</td>
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<td>RUCA</td>
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<td></td>
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<td>Urban core areas</td>
<td>4,823 (67.6)</td>
<td>413 (65.1)</td>
<td>1,648 (87.7)</td>
<td>7,984 (67.8)</td>
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<tr>
<td>Suburban areas</td>
<td>1,031 (14.4)</td>
<td>94 (14.8)</td>
<td>61 (3.2)</td>
<td>1,298 (11.1)</td>
</tr>
<tr>
<td>Micropolitan areas</td>
<td>707 (10.9)</td>
<td>71 (11.2)</td>
<td>90 (4.8)</td>
<td>1,518 (13.2)</td>
</tr>
<tr>
<td>Small town area/ rural areas</td>
<td>558 (8.7)</td>
<td>56 (8.8)</td>
<td>74 (3.9)</td>
<td>788 (8.0)</td>
</tr>
<tr>
<td>Infants</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Male</td>
<td>3,653 (51.2)</td>
<td>343 (51.4)</td>
<td>943 (50.2)</td>
<td>5,939 (51.2)</td>
</tr>
<tr>
<td>Female</td>
<td>3,481 (48.8)</td>
<td>290 (44.6)</td>
<td>933 (48.7)</td>
<td>6,404 (48.8)</td>
</tr>
<tr>
<td>Birth weight</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1,500 g</td>
<td>457 (6.4)</td>
<td>91 (14.4)</td>
<td>289 (15.4)</td>
<td>797 (6.1)</td>
</tr>
<tr>
<td>1,500–2,499 g</td>
<td>1,005 (14.1)</td>
<td>146 (23.0)</td>
<td>327 (17.4)</td>
<td>1,478 (12.6)</td>
</tr>
<tr>
<td>≥2,500 g</td>
<td>5,675 (79.5)</td>
<td>396 (62.5)</td>
<td>1,260 (67.1)</td>
<td>9,331 (79.5)</td>
</tr>
<tr>
<td>Gestational age</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>&lt;37 wk</td>
<td>1,850 (25.9)</td>
<td>240 (37.9)</td>
<td>686 (35.5)</td>
<td>2,776 (24.0)</td>
</tr>
<tr>
<td>≥37 wk</td>
<td>5,287 (74.1)</td>
<td>393 (62.0)</td>
<td>1,192 (63.4)</td>
<td>7,872 (76.0)</td>
</tr>
<tr>
<td>Birth weight/gestational age</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;37 wk, &lt;1,500 g</td>
<td>440 (6.2)</td>
<td>86 (13.6)</td>
<td>284 (15.1)</td>
<td>710 (6.1)</td>
</tr>
<tr>
<td>&lt;37 wk, 1,500–2,499 g</td>
<td>655 (9.2)</td>
<td>86 (13.6)</td>
<td>207 (11.0)</td>
<td>948 (8.3)</td>
</tr>
<tr>
<td>&lt;37 wk, ≥2,500 g</td>
<td>754 (10.6)</td>
<td>67 (10.5)</td>
<td>195 (10.3)</td>
<td>996 (8.8)</td>
</tr>
<tr>
<td>≥37 wk, &lt;1,500 g</td>
<td>17 (0.2)</td>
<td>5 (0.8)</td>
<td>5 (0.3)</td>
<td>27 (0.3)</td>
</tr>
<tr>
<td>≥37 wk, 1,500–2,499 g</td>
<td>348 (4.9)</td>
<td>59 (9.3)</td>
<td>120 (6.4)</td>
<td>527 (4.6)</td>
</tr>
<tr>
<td>≥37 wk, ≥2,500 g</td>
<td>4,920 (68.9)</td>
<td>329 (51.9)</td>
<td>1,068 (56.7)</td>
<td>6,317 (54.8)</td>
</tr>
<tr>
<td>Type of defect</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Isolated heart defect</td>
<td>2,872 (40.2)</td>
<td>97 (15.3)</td>
<td>827 (44.0)</td>
<td>3,796 (33.1)</td>
</tr>
<tr>
<td>Multiple heart</td>
<td>2,718 (38.1)</td>
<td>240 (37.9)</td>
<td>650 (37.0)</td>
<td>5,618 (48.8)</td>
</tr>
<tr>
<td>Extracardiac defects</td>
<td>1,549 (21.7)</td>
<td>297 (46.8)</td>
<td>356 (18.9)</td>
<td>2,202 (18.1)</td>
</tr>
</tbody>
</table>

Percentages do not add up to 100% and counts do not add up to the total because of missing values.

hood. Most notably, NH black children with TGA (Fig 1) had a much lower survival rate during early childhood than NH white or Hispanic children (52.4% vs 76.9% and 71.5%, respectively). NH black and Hispanic children had poorer survival rates than NH white children for tetralogy of Fallot (73.3% and 76.0%, respectively, vs 84.2%) (Fig 2).

After adjusting for covariates, NH black and Hispanic children with common truncus had an ~88% increased risk of early childhood mortality compared with similarly affected NH white children, but the increase was not statistically significant (Table 2). NH black children with TGA had a statistically significant twofold increased risk of death in early childhood compared with similarly affected NH white children (95% CI: 1.40–2.97). NH black children with tetralogy of Fallot also had almost a twofold increased risk of death in early childhood compared with similarly affected NH white children (HR: 1.85 [95% CI: 1.09–3.21]). Hispanic children with tetralogy of Fallot had a 39% increased risk of mortality compared with NH white children, but
the increase was not statistically significant.

**Right Obstructive CHDs**

NH black children with tricuspid valve defects had a notably poorer survival rate during early childhood compared with similarly affected NH white and Hispanic children (58.2% vs 70.9% and 70.1%, respectively) (Table 3). Figure 3 shows that NH black and Hispanic children with pulmonary valve atresia without VSD had lower survival rates (48.3% and 59.0%, respectively) than NH white children (72.4%). Both NH black and Hispanic children with Ebstein anomaly fared much worse during the first 5 years than similarly affected NH white children (61.5% and 64.6% vs 77.5%, respectively) (Table 3).

After adjusting for covariates, NH black children with tricuspid valve defects had a 42% increased risk of early childhood mortality compared with NH white children with tricuspid valve de-
fects, but the increase was not statistically significant (Table 2). NH black and Hispanic children with pulmonary valve atresia without VSD had a statistically significant 160% and 76%, increased risk of death in early childhood, respectively, after adjusting for covariates, compared with NH white children with the same CHD (95% CI: 1.32–5.12 and 1.06–2.91, respectively). Although NH black and Hispanic children with Ebstein anomaly had increased risk of mortality, the increases were not statistically significant in the adjusted analyses.

**Left Obstructive CHDs**

Racial/ethnic disparities in survival rates were seen among children with left obstructive CHDs (Table 3). Although survival rates were low for all children with HLHS, Hispanic children had the lowest survival rate (30.9%), followed by NH black (37.2%) and NH white (43.5%) children (Fig 4). After adjusting for covariates, Hispanic children with HLHS had a 51% greater risk of death compared with NH white children (95% CI: 1.13–2.02) (Table 2). NH black children with aortic valve stenosis and coarctation of the aorta had poorer survival rates than NH white and Hispanic children (Table 3). In contrast to the unadjusted analyses (Kaplan-Meier), NH black children with aortic valve stenosis or coarctation of the aorta had no increased risk of early childhood mortality compared with NH white children after adjusting for covariates (Table 2).

**Septal CHDs**

Overall, the survival rate for infants with septal defects was quite high regardless of maternal race/ethnicity; however, NH black children had a consistent pattern of having the lowest survival rate of the 3 racial/ethnic groups (Table 3). NH black and Hispanic children with atrioventricular septal defect had poorer survival rates (68.8% and 69.6%, respectively) than NH white children (75.2%) with the same defect.

As shown in Table 2, after adjusting for covariates, NH black children with VSD had a 56% increased risk and those with ASD had a 34% increased risk of early childhood mortality compared with NH white children with the same CHD (95% CI: 1.19–2.03 and 1.08–1.66, respectively).

**DISCUSSION**

We investigated maternal race/ethnicity and its effects on early childhood survival among children with CHDs; NH black race/ethnicity was more strongly associated with increased risk of early childhood mortality than Hispanic race/ethnicity. NH black children with TGA, tetralogy of Fallot, pulmonary valve atresia without VSD, VSD, and ASD had a statistically significant increased risk of mortality compared with NH white children with the same defects. Hispanic children with pulmo-
nary valve atresia without VSD or HLHS also had a statistically significant increased risk of mortality compared with NH white children. With inspection of the survival curves for TGA, pulmonary valve atresia without VSD, and HLHS (Figs 1–3) it is apparent that the disparity in ethnic mortality develops during months 2 to 3 of life. Because these cases require cardiac surgery during the first month, this temporal trend suggests that deficiencies in postdischarge care play a role in the increased mortality rates among minorities. Such findings indicate a need to intensify home health care and enhance communication between the cardiac center and the families.

Our findings provide additional evidence of racial/ethnic disparities in early childhood survival among children with CHD and are consistent with those of earlier reports. Using 1996 data from the Healthcare Cost and Utilization Project (HCUP) Kids’ Inpatient Database (KID) for 4 states, Gonzalez et al found that Hispanic children had a higher risk of dying after surgery for CHD than NH white children. Although there was no statistically significant difference in mortality rates overall between NH black and white children, there was variation in mortality rates for NH black children between states. Benavidez et al used 2000 HCUP-KID data for 19 states and found that black and Hispanic children with CHD had higher odds of in-hospital mortality after CHD surgery than white children after adjusting for covariates. Another study used national death-certificate data from 1979–1997 to determine trends in CHD-associated mortality in the United States. Black children had a 19% higher mortality rate from CHDs compared with NH white children and a lower decline in mortality rates over the 18-year period than NH white children. The average ages at death of CHD were also 3 to 6 times lower for black children compared with NH white children and approximately half the average age at death compared with NH white children for children with TGA, tetralogy of Fallot, VSD, and single ventricle. The infant mortality rate for VSD was higher during the 18-year study period for NH black children.

The racial/ethnic disparities we observed in early childhood survival and mortality may be caused by (1) underlying racial/ethnic biological differences, (2) differences in access to health care, and (3) differences in cultural preferences. Biological differences that influence survival include the severity of the defect, number of co-occurring defects, and prevalence at live birth. In our study, we found higher mortality rates for NH black children with several types of CHDs. Defects greatly vary in severity, which may partially explain our findings. Although we were unable to evaluate the effect of severity on early childhood mortality and whether severity differed according to race/ethnicity, there is no published evidence.
that suggests that defect severity differs according to maternal race/ethnicity. Our findings may also be related to racial/ethnic differences in the distribution of extracardiac defects and syndromes; however, we did adjust for the number of co-occurring defects in our analyses and excluded infants with trisomies 13 and 18.

The racial/ethnic differences we observed in early childhood survival could also be a result of lack of timely access to good-quality health care. Factors associated with access to good-quality health care include age at operation, place of residence, hospital volume, surgical case volume, and socioeconomic factors. Racial/ethnic differences in access to pediatric care and treatment received when care is obtained have been well documented. For example, minority children on waiting lists for heart transplants have higher wait-list mortality rates than NH white children. Mahle et al showed that NH black children had a lower 5-year transplant graft survival rate than white children; the median survival time was ~6 years lower than that for white children. NH black children also had a higher median age at transplant (~5 years older) and were also more likely to have an HLA antigen mismatch. Although it is plausible that lack of access to good-quality health care may cause black children to be less likely to have their CHD diagnosed early in infancy than white children, studies have revealed no statistically significant differences between black and white children in the age at diagnosis of CHDs or age at surgical repair for infants with CHDs. Moreover, the age at operation has not been shown to affect health outcomes. Thus, a racial/ethnic differential in age at diagnosis or surgical repair of CHD is an unlikely explanation for our findings.

NH black adults and children also have lower rates of surgical cardiovascular procedures than NH white adults and children, possibly because of socioeconomic status, insurance coverage, access to services, transportation, knowledge and understanding of the procedures, and patient trust and comfort issues. Racial/ethnic differences in the quality of care received may also occur because of differences within a medical facility according to race/ethnicity and between medical facilities (those that serve primarily minority versus primarily white populations). In general, white and black patients receive care at different medical institutions; black patients are treated more often at hospitals that have higher mortality rates, which possibly suggests lower quality of care. Racial/ethnic disparities in access to good-quality care may also be the result of differences in health insurance; type of health insurance has a significant effect on access to care among children.

FIGURE 3
health care needs, black children are more likely to not have a regular clinician, are less likely to have a usual source of care at a physician’s private office or health maintenance organization office, and have reported higher levels of dissatisfaction with care.

Other important influences on early childhood survival are cultural factors and preferences. Differences in prenatal diagnosis of defects that result in a bias favoring the termination of more severely affected fetuses may partially explain our findings. NH black and Hispanic children are less likely to have their defect detected prenatally and less likely to terminate their pregnancies if a defect is detected.

Strengths of our study include its population-based cohort design, a large number of live births, and data from a defined multiethnic population ascertained by an active surveillance system with high sensitivity. However, our study has some potential limitations. We may have had incomplete ascertainment of (1) deaths and (2) cases of birth defects for years when the registry covered limited regions of the state; however, it is unclear how these missing data may have affected our findings. Another limitation was our inability to adjust for the potential effects of racial/ethnic differences in the type of treatment provided to the child, preferences in medical treatments received, health insurance/type of payment for services, and surgical complications.

CONCLUSIONS
Our study contributes to a growing body of literature that indicates that NH black and Hispanic children with specific phenotypes of CHDs have poorer survival rates in early childhood than NH white children. Future investigations should determine if there are racial/ethnic differences in access to care among children with CHDs, the severity of CHDs or defect subtypes, the types of treatments selected, and underlying causes of death. Elucidation of these factors will facilitate development of public health and tertiary prevention strategies to address this important health outcome.

ACKNOWLEDGMENTS
This study was partially supported by the Centers for Disease Control and Prevention–funded Texas Center for Birth Defects Research and Prevention (grant U50/DD613232) through a cooperative agreement with the Texas Department of State Health Services and the Office of Title V and Family Health, using Title V MCH block grant funds. We thank the staff of the Texas Birth Defects Epidemiology and Surveillance Branch.
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Racial/Ethnic Disparities in Risk of Early Childhood Mortality Among Children With Congenital Heart Defects

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*Pediatrics* published online Apr 18, 2011;
DOI: 10.1542/peds.2010-2702

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