Variation in the Prevalence of Congenital Heart Defects by Maternal Race/Ethnicity and Infant Sex

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Objective To determine the prevalence of major congenital heart defects (CHD) by ethnicity and sex.

Study design Data from the Florida Birth Defects Registry was used to conduct a retrospective cohort study with 8029 singleton infants with 11 CHDs born 1998–2003 to resident non-Hispanic (NH) white, NH-black, and Hispanic women aged 15 to 49. Defect-specific prevalence rates, ratios, and 95% confidence intervals were calculated. Poisson regression was used to calculate adjusted ethnic-specific rate ratios (RR) for each CHD. Statistical significance was $P < .0001$.

Results Compared with NH-whites, NH-black males had significantly increased rates of pulmonary valve atresia/stenosis (RR = 1.66) but lower prevalence of aortic valve atresia/stenosis (RR = 0.33) and ventricular septal defect (VSD; RR = 0.78). Hispanic males had lower rates of aortic valve atresia/stenosis (RR = 0.28), coarctation of the aorta (RR = 0.61) and VSD (RR = 0.79). NH-black females had statistically significantly lower rates of VSD (RR = 0.75), and Hispanic females had lower rates of tetralogy of Fallot (RR = 0.54), VSD (RR = 0.84) and atrioventricular septal defects (RR = 0.53) compared with NH-whites.

Conclusions We found differences in ethnic susceptibilities to CHD by sex, but the cause remains unclear. *(J Pediatr 2010;156:259-64)*.

Birth defects are the leading cause of U.S. infant morbidity and death, and congenital heart defects (CHD) remain the leading cause of infant death from birth defects.[1] With an estimated annual prevalence ranging from 4 to 50 affected infants per 1000 live births,[2] CHDs are the most prevalent defects. Despite numerous advances in CHD diagnosis and treatment, as well as an improved understanding of embryonic and fetal development, knowledge about the cause of CHD, with the exception of only 5% to 10% of CHD cases attributed to chromosomal abnormalities and single gene defects, remains limited.[3-5] The underlying mechanism is hypothesized to be multifactorial, involving a complex interplay between genetic and environmental factors. This is supported by evidence from recent studies that noninherited risk of certain CHDs may be modifiable by nutritional intervention, specifically supplementation with folic acid or multivitamins containing folic acid.[6, 7]

Studies show racial/ethnic differences in prevalence of several types of birth defects,[8-11] including CHDs.[8,9,12-14] Racial/ethnic differences in prevalence may reflect differences in genetic predisposition, susceptibility caused by polymorphisms, differences in environmental exposures (eg, environmental contaminants), or access to care (diagnosis of CHDs). Moreover, racial/ethnic differences exist in response to physiologically based CHD interventions; non-Hispanic (NH)-blacks and Hispanics had less reduction in risk of folic acid–sensitive CHDs compared with NH-whites.[15,16] Although exact causes of racial/ethnic differences is unknown, it is also unclear how infant sex may influence racial/ethnic differences in rates of CHD. While sex differences in rates of CHD are well established,[17,18] few studies have investigated differences in CHD rates by maternal race/ethnicity and infant sex.[19,20] The purpose of the study was to determine the live birth prevalence of 11 major CHDs by race/ethnicity-sex and whether prevalence differs by race/ethnicity and sex after adjusting for covariates.

Methods

We used data from the Florida Birth Defects Registry (FBDR), a passive population-based surveillance system, to conduct a retrospective cohort study. Since 1998 the FBDR has monitored birth defects in Florida by merging data from (1) Florida Vital Statistics Birth Data; (2) Agency for Health Care Administration hospital discharge data; (3) Children's Medical Services (CMS)

CHD	Congenital heart defects
CMS	Children's Medical Services
FBDR	Florida Birth Defects Registry
ICD-9-CM	International Classification of Diseases, Ninth edition, Clinical Modification
NH	Non-Hispanic
PR	Prevalence ratio
RVOTO	Right ventricular outflow tract obstruction
VSD	Ventricular septal defect

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Regional Perinatal Intensive Care Center data; (4) CMS Early Steps data; and (5) the CMS Minimum Data Set. The FBDR uses a deterministic merging strategy to link source data sets to the birth vital record, with variables such as mother and child's names, social security numbers, dates of birth, and infant sex. All questionable links are manually reviewed. The success rate for the linkage process ranges from approximately 85% in the Agency for Health Care Administration inpatient data set to more than 95% in the Regional Perinatal Intensive Care Center data set. 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, (ICD-9-CM) diagnosis coding system.

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, 15 to 49 years old, were eligible for this study. CHDs were classified with select ICD-9-CM diagnosis codes in the 745.00 to 747.99 range, and affected infants were placed into categories on the basis of cardiac phenotype.[21] 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 ventricular outflow tract 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 ventricular outflow tract obstructive CHDs included hypoplastic left heart syndrome (746.7), aortic valve stenosis (746.3), and coarctation of the aorta (747.10). Septal CHDs included ventricular septal defect (VSD, 745.4); and atrioventricular septal defects (745.60, 745.61, or 745.69) were the final category.

Data on maternal race/ethnicity, age, education, and infant sex were obtained from the Florida Office of Vital Statistics live birth certificate. Maternal race/ethnicity was determined on the basis of 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. Maternal education was grouped, on the basis of years of education, as <12, 12, and 13+ years.

During the study period, 1 216 142 infants were live births to Florida residents; of these 10 027 had at least 1 of the selected CHD. 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 (2.5%) gestational age (1.8%), maternal education (0.4%), parity (0.03%), birth weight (0.03%), or prenatal maternal smoking (0.02%) were also excluded (numbers do not add up to 966 because some infants had more than 1 exclusion).

Race/ethnic- and sex-specific prevalence estimates were calculated for each type of CHD by dividing the number of cases of CHD by the number of live births during the study period for each racial/ethnic and sex group (per 10 000 live births). The exact 95% confidence interval (CI) for each prevalence was obtained by the Clopper-Pearson method.[22] We used the prevalence ratio (PR), the prevalence in 1 racial/ethnic-sex group divided by the prevalence in the reference group (NH-white), to assess the variation in prevalence by maternal race/ethnicity. For each PR, Bonferroni simultaneous CIs were obtained on the basis of the normal approximation on the log-scale.[23] By use of simultaneous CIs with an overall confidence coefficient of at least 95% for multiple testing, the family-wise error rate[24] is controlled to be no greater than 5%.

Multivariable Poisson regression modeling was used to determine the relative risk of the occurrence of each type of CHD for NH-blacks and Hispanics relative to NH-whites (reference group) after adjusting for covariates. Models were constructed for all CHD types combined and for each type of CHD separately. To examine the effects of infant sex and maternal race/ethnicity on the relative risk of CHD, the final adjusted model for each type of CHD included all covariates (maternal age, education, parity and maternal prenatal tobacco use), as well as important effect modification (interaction) terms.

Our initial statistical significance level was set at $P < .05$ for the main effect and $P < .10$ for interactive effects. However, because 48 hypotheses were tested simultaneously, 3 statistically significant results from our models would occur by chance alone at the $P < .05$ level. Hence, our P value for the multivariable models was adjusted for multiple hypothesis testing with the Bonferroni correction,[25] which reduces the statistical significance level of each individual test from $P < .05$ to a more conservative significance level ($P < .0001$) to control the family-wise error rate at 0.05.[24] All P values are 2-sided.

The research was conducted in accordance with the prevailing ethical principles and the Office of Research Integrity and Compliance, Institutional Review Board at the University of South Florida approved the study. The Florida Department of Health Institutional Review Board approved the study and the use of data from Florida birth records and FBDR data.

Results

After exclusions, our final study population included 8029 infants with CHDs, of which 4346 (54.1%) were born to NH-white, 1779 (22.2%) were born to NH-black, and 1904 (23.7%) were born to Hispanic women. The study population included 4010 (49.9%) males and 4019 (50.1%) females.

Overall, among males we found few racial/ethnic differences in the crude prevalence of specific types of CHD (**Table I**). We observed no statistically significant racial/ethnic differences among males with conotruncal CHD. We did find differences among males with right ventricular outflow tract obstruction (RVOTO) defects; compared with the prevalence for NH-white males, the prevalence of pulmonary valve atresia/stenosis (PR = 1.72; 95% CI: 1.30 - 2.29) was higher among NH-black males. We found no difference in prevalence between Hispanic and NH-white males with types

Table I. Number, percent, prevalence rates, unadjusted prevalence ratios and 95% simultaneous confidence intervals for male infants with congenital heart defects by maternal race/ethnicity, Florida Birth Defects Registry, 1998–2003 (n = 4010)

	Number of cases			Prevalence rate*†				Prevalence Rate Ratio‡	
CHD	NHW	NHB	Hispanic	NHW	NHB	Hispanic	Total	BW RR§	HW RR§
Conotruncal Defects									
Common truncus	32 (60.4%)	9 (17.0%)	12 (22.6%)	0.99 (0.67, 1.39)	0.65 (0.30, 1.24)	0.83 (0.42, 1.45)	0.87 (0.57, 1.28)	0.66 (0.21, 2.18)	0.84 (0.30, 2.44)
Transposition of great vessels	168 (58.7%)	57 (19.9%)	61 (21.3%)	5.18‖ (4.42, 6.02)	4.15‖ (3.13, 5.37)	4.22‖ (3.23, 5.42)	4.72 (3.96, 5.58)	0.80 (0.50, 1.30)	0.82 (0.51, 1.30)
Tetralogy of Fallot	182 (55.0%)	69 (20.9%)	80 (24.2%)	5.61‖ (4.82, 6.48)	5.02‖ (3.90, 6.35)	5.54‖ (4.39, 6.89)	5.46 (4.63, 6.38)	0.89 (0.58, 1.39)	0.99 (0.65, 1.50)
Right ventricular outflow tract obstructive defects									
Tricuspid valve atresia/stenosis	42 (47.7%)	28 (31.8%)	18 (20.4%)	1.29 (0.93, 1.75)	2.04‖ (1.35, 2.94)	1.25‖ (0.74, 1.97)	1.45 (1.05, 1.95)	1.57 (0.74, 3.37)	0.96 (0.41, 2.33)
Pulmonary valve atresia/stenosis	282 (45.4%)	206 (33.2%)	133 (21.4%)	8.69‖ (7.70, 9.77)	14.98‖ (13.00, 17.17)	9.21‖ (7.71, 10.91)	10.24 (9.10, 11.47)	1.72‖ (1.30, 2.29)	1.06 (0.76, 1.47)
Ebstein's Anomaly	21 (60.0%)	3 (8.6%)	11 (31.4%)	0.65‖ (0.40, 0.99)	0.22‖ (0.04, 0.64)	0.76 (0.38, 1.36)	0.58 (0.34, 0.92)	0.34 (0.06, 2.32)	1.18 (0.38, 3.76)
Right ventricular outflow tract obstructive defects									
Hypoplastic left heart syndrome	91 (52.6%)	43 (24.9%)	39 (22.5)	2.80‖ (2.26, 3.44)	3.13‖ (2.26, 4.21)	2.70‖ (1.92, 3.69)	2.85 (2.23, 3.53)	1.11 (0.63, 1.99)	0.96 (0.53, 1.76)
Aortic valve atresia/stenosis	77 (77.8%)	11 (11.1%)	11 (11.1%)	2.37‖ (1.87, 2.96)	0.80 (0.40, 1.43)	0.76 (0.38, 1.36)	1.63 (1.20, 2.16)	0.34‖ (0.13, 0.94)	0.32‖ (0.12, 0.89)
Coarctation of the aorta	211 (61.3%)	68 (19.8%)	65 (18.9%)	6.50‖ (5.65, 7.44)	4.95‖ (3.84, 6.27)	4.50‖ (3.47, 5.74)	5.67 (4.84, 6.60)	0.76 (0.49, 1.18)	0.69 (0.45, 1.08)
Septal defects									
Ventricular septal defect	1478 (55.6%)	536 (20.2%)	643 (24.2%)	45.55‖ (43.26, 47.93)	38.98‖ (35.75, 42.41)	44.52‖ (41.15, 48.09)	43.82 (41.42, 46.30)	0.86 (0.73, 1.00)	0.98 (0.84, 1.13)
Atrioventricular septal defect	130 (59.4%)	51 (23.3%)	38 (17.4%)	4.01‖ (3.34, 4.76)	3.71‖ (2.76, 4.88)	2.63‖ (1.86, 3.61)	3.61 (2.95, 4.37)	0.93 (0.56, 1.56)	0.66 (0.37, 1.17)
Total	2217 (55.3%)	885 (22.1%)	908 (22.6%)	68.33‖ (65.52, 71.22)	64.36‖ (60.20, 68.73)	62.87‖ (58.86, 67.08)	66.13 (62.90, 71.31)	0.94‖ (0.87, 1.02)	0.92‖ (0.85, 0.99)

*Prevalence is per 10 000 live births.
†Prevalence rates calculated by number of cases of CHD for each race/ethnic group divided by the number of live births during the study period.
‡Rate ratios calculated by prevalence rate for NHB and Hispanic divided by the prevalence rate for NHW.
§Referent group = non-Hispanic whites.
‖Statistically significant Bonferroni adjusted simultaneous 95% confidence intervals.

of RVOTO. Among infants with left ventricular outflow tract obstruction defects, the prevalence of aortic valve atresia/stenosis and coarctation of the aorta was lower for both NH-black and Hispanic males compared with NH-white males, but the differences were not statistically significant at $P < .0001$ for coarctation of the aorta. For infants with septal CHDs, NH-black males had lower prevalence of VSD compared with NH-white males (PR = 0.86; 95% CI: 0.73 - 1.00).

After adjusting for covariates, NH-black males had increased risk of pulmonary valve atresia/stenosis ($RR_a = 1.66$, $P < .0001$) but lower risk of aortic valve atresia/stenosis ($RR_a = 0.33$, $P = .0001$) and VSD ($RR_a = 0.78$, $P < .0001$) in comparison to NH-white males (**Table II**). Hispanic males had lower risk of aortic valve atresia/stenosis ($RR_a = 0.28$, $P < .0001$), coarctation of the aorta ($RR_a = 0.61$, $P = .0003$) and VSD ($RR_a = 0.79$, $P < .0001$) as compared with NH-white males.

We observed fewer racial/ethnic differences in crude CHD prevalence among females (**Table III**). We found no racial/ethnic differences in prevalence among females with conotruncal CHD. Among infants with RVOTO defects, NH-black females had statistically significant higher rates of pulmonary valve atresia/stenosis (PR = 1.64; 95% CI: 1.22 - 2.19) compared with NH-white females. We found no differences in prevalence between Hispanic and NH-white females with RVOTO. The prevalence of left ventricular outflow tract obstruction and septal defects did not differ between NH-whites, NH-blacks, or Hispanics.

After adjusting for covariates, compared with NH-white females, NH-black, and Hispanic females had decreased risk of VSD ($RR_a = 0.75$ and $RR_a = 0.84$, $P \leq .0001$, respectively) (**Table II**). Hispanic females also had lower risk of tetralogy of Fallot ($RR_a = 0.54$, $P = .0001$) and atrioventricular septal defect ($RR_a = 0.53$, $P = .0001$) compared to NH-whites.

Discussion

Our intent was to determine the prevalence of specific types of CHD by race/ethnicity and sex for NH-white, NH-black, and Hispanic infants and to determine whether prevalence varied by race/ethnicity and infant sex after adjusting for

Table II. Adjusted* relative risks from multivariate Poisson regression analyses for CHD by infant sex and maternal race/ethnicity

	Males (n = 4612)						Females (n = 4449)					
	NH-blacks† (n = 1017)			Hispanics† (n = 1072)			NH-blacks† (n = 987)			Hispanics† (n = 1134)		
CHD	n	RR	*P* value	n	RR	*P* value	n	RR	*P* value	n	RR	*P* value
Conotruncal defects												
Common truncus	9	0.70	.3423	12	0.77	.4407	21	1.64	.0994	10	0.62	.1932
Transposition of the great vessels	57	0.89	.4759	61	0.76	.0724	41	0.88	.4844	42	0.74	.1008
Tetralogy of Fallot	69	0.84	.2089	80	0.83	.1705	67	0.87	.3429	50	0.54‡	.0001
Right obstructive defects												
Tricuspid valve atresia/stenosis	28	1.82	.0204	18	0.91	.7451	19	1.57	.1453	13	0.84	.5998
Pulmonary valve atresia/stenosis	206	1.66‡	<.0001	133	0.90	.3392	193	1.46‡	.0001	119	0.75	.0103
Ebstein's anomaly	3	0.26	.0130	11	0.83	.6129	3	0.27	.0151	4	0.29	.0134
Left obstructive defects												
Hypoplastic left heart syndrome	43	1.09	.6607	39	0.83	.3309	38	1.45	.0917	19	0.61	.0611
Aortic valve atresia/stenosis	11	0.33‡	.0001	11	0.28‡	<.0001	13	0.58	.0726	18	0.67	.1455
Coarctation of the aorta	68	0.77	.0625	65	0.61‡	.0003	55	0.82	.2182	52	0.63	.0038
Septal defects												
Ventricular septal defect	536	0.78‡	<.0001	643	0.79‡	<.0001	573	0.75‡	<.0001	776	0.84‡	.0001
Atrioventricular septal defect	51	0.97	.8486	38	0.60	.0052	53	0.76	.0935	44	0.53‡	.0001

*Adjusted for maternal age, education, parity, prenatal maternal smoking, and number of birth defects.
†Reference group = non-Hispanic whites.
‡Statistically significant $P < .0001$.

potential confounders. In general, we found few racial/ethnic-sex differences in the prevalence of CHD. Although there is little information previously published on race/ethnic- or sex-specific prevalence of specific types of CHD to which we can directly compare our results,[19,20] there are published reports on race/ethnicity patterns in general. Canfield et al[8] reported a lower prevalence of tetralogy of Fallot for infants born to Hispanic women compared with infants born to NH-white women; in our study lower prevalence is statistically significant only for Hispanic females. Ferencz et al[26] reported a higher prevalence of both tricuspid valve atresia/stenosis and pulmonary valve atresia/stenosis for NH-blacks compared with NH-whites; in our study rate ratios for NH-black males and females, pulmonary valve atresia/stenosis– and tricuspid valve atresia/stenosis–affected infants were elevated compared with NH-whites. NH-black infants were also reported to have lower prevalence of aortic valve atresia/stenosis relative to NH-Whites,[9,12,26-28] but in our study it was statistically significant only for NH-black males. The lower prevalence of coarctation of the aorta for NH-black and Hispanic infants compared with NH-white infants suggested in the literature[9,20,29] is seen in our data to occur only among Hispanic males and females compared with NH-whites and although present for both black males and females, not statistically significant. Although reports of lower prevalence of ventricular septal defect for NH-blacks and Hispanics compared with NH-whites[14] was also seen in our study, the magnitude of the decrease was similar for both male and female NH-black and Hispanic infants compared with NH-whites. Our finding of lower prevalence of atrioventricular septal defect for Hispanic females compared with NH-white females has not been previously reported.

The explanations for our findings are not readily apparent. Many defects have deviations in the ratio of affected males versus affected females,[30] and there are differences in the sex ratios by ethnicity for infants with cleft lip,[31] but no explanations for these deviations have been forthcoming. It is possible that the racial/ethnic differences we observed may be the result of differential loss of CHD-affected pregnancies during the gestational period rather than true differences in the incidence of CHD. We report "prevalence at live birth" rather than incidence because incidence is a complex phenomenon affected by spontaneous miscarriages, fetal deaths, and elective terminations.[32] Racial/ethnic sex differences in loss of pregnancies affected by CHD may cause differences in prevalence at live birth. At present there is no information supporting racial/ethnic-sex differences in rates of spontaneous miscarriages or fetal deaths for CHD-affected pregnancies, thus it is unknown whether these losses affected our results. It is also possible that our results are due to racial/ethnic-sex differences in diagnosis of CHD after birth, but no published studies have documented differential access to diagnosis or care for infants with CHD. Although studies show racial/ethnic disparities in access to pediatric care and treatment received when care is obtained,[33-35] there are no statistically significant differences between blacks and whites in the age at diagnosis of CHD[36] and no reason to

Table III. Number, percent, prevalence rates, unadjusted prevalence ratios and 95% simultaneous confidence intervals for female infants with CHD by maternal race/ethnicity, Florida Birth Defects Registry, 1998–2003 (n = 4019)

	Number of cases			Prevalence rate*†				Prevalence Rate Ratio‡	
CHD	NHW	NHB	Hispanic	NHW	NHB	Hispanic	Total	BW RR§	HW RR§
Conotruncal defect									
Common truncus arteriosus	28 (47.5%)	21 (35.6%)	10 (16.9%)	0.91 (0.61, 1.32)	1.57 (0.97, 2.41)	0.72 (0.35, 1.33)	1.02 (0.68, 1.46)	1.72 (0.71, 4.22)	0.79 (0.26, 2.51)
Transposition of great vessels	103 (55.4%)	41 (22.0%)	42 (22.6%)	3.36‖ (2.74, 4.07)	3.07‖ (2.21, 4.17)	3.03‖ (2.18, 4.10)	3.23 (2.58, 3.94)	0.91 (0.51, 1.63)	0.90 (0.51, 1.60)
Tetralogy of Fallot	154 (56.8%)	67 (24.7%)	50 (18.4%)	5.03‖ (4.30, 5.89)	5.02‖ (3.89, 6.38)	3.61‖ (2.68, 4.76)	4.68 (3.91, 5.56)	1.00 (0.64, 1.58)	0.72 (0.43, 1.20)
Right ventricular outflow tract obstructive									
Tricuspid valve atresia/stenosis	28 (46.7%)	19 (31.7%)	13 (21.7%)	0.91 (0.61, 1.32)	1.42 (0.86, 2.22)	0.94 (0.50, 1.60)	1.04 (0.70, 1.48)	1.56 (0.63, 3.93)	1.03 (0.37, 2.94)
Pulmonary valve atresia/stenosis	271 (46.5%)	193 (33.1%)	119 (20.4%)	8.85‖ (7.82, 9.97)	14.47‖ (12.50, 16.66)	8.59‖ (7.11, 10.27)	10.08 (8.92, 11.34)	1.64‖ (1.22, 2.19)	0.97 (0.69, 1.37)
Ebstein's anomaly	19 (73.1%)	3 (11.5%)	4 (15.4%)	0.62‖ (0.37, 0.97)	0.22‖ (0.05, 0.66)	0.29‖ (0.08, 0.74)	0.45 (0.24, 0.77)	0.36 (0.07, 2.53)	0.47 (0.01, 2.61)
Right ventricular outflow tract obstructive									
Hypoplastic left heart syndrome	53 (48.2%)	38 (34.6%)	19 (17.3%)	1.73‖ (1.29, 2.26)	2.85‖ (2.02, 3.91)	1.37 (0.82, 2.14)	1.90 (1.42, 2.48)	1.65 (0.85, 3.20)	0.79 (0.35, 1.84)
Aortic valve atresia/stenosis	46 (59.7%)	13 (16.9%)	18 (23.4%)	1.50‖ (1.10, 2.00)	0.97 (0.52, 1.67)	1.30 (0.77, 2.05)	1.33 (0.94, 1.83)	0.65 (0.25, 1.75)	0.86 (0.37, 2.07)
Coarctation of the aorta	141 (56.9%)	55 (22.2%)	52 (21.0%)	4.60‖ (3.87, 5.43)	4.12‖ (3.11, 5.37)	3.75‖ (2.80, 4.92)	4.29 (3.55, 5.13)	0.90 (0.55, 1.48)	0.82 (0.49, 1.36)
Septal defects									
Ventricular septal defect	1522 (53.0%)	573 (20.0%)	776 (27.0%)	49.70‖ (47.24, 52.25)	42.97‖ (39.53, 46.63)	56.00‖ (52.14, 60.07)	49.66 (47.05, 52.37)	0.86 (0.74, 1.01)	1.13 (0.98, 1.29)
Atrioventricular septal defect	151 (60.9%)	53 (21.4%)	44 (17.7%)	4.93‖ (74.96, 81.22)	3.97‖ (2.98, 5.20)	3.18‖ (2.31, 4.26)	4.29 (3.55, 5.13)	0.81 (0.49, 1.33)	0.64 (0.38, 1.10)
Total	2129 (53.0%)	894 (22.2%)	996 (24.8%)	69.52 (66.60, 72.53)	67.04 (62.74, 71.57)	71.88 (67.50, 76.47)	69.46 (63.70, 75.89)	0.96 ‖ (0.89, 1.04)	1.03‖ (0.95, 1.11)

*Prevalence is per 10 000 live births.
†Prevalence rates calculated by number of cases of CHD for each race/ethnic group divided by the number of live births during the study period.
‡Rate ratios calculated by prevalence rate for NHB and Hispanic divided by the prevalence rate for NHW.
§Referent group = non-Hispanic whites.
‖Statistically significant Bonferroni adjusted simultaneous 95% confidence intervals.

believe that, if present, this would occur differentially by sex; however, differential access to care could explain observed differences within sex groups.

Our study should be interpreted in the context of some potential limitations. Compared with active surveillance systems, which ascertain cases through medical record abstraction, passive systems such as the FBDR, generally underestimate the number of infants with CHDs because case ascertainment relies heavily on data linkage. Therefore it is likely that we have underestimated the true number of CHD cases in Florida. Another important limitation is the use of ICD-9-CM codes for diagnosis of CHD. ICD-9-CM codes have poor diagnostic accuracy because they lack the specificity to distinguish significant CHD from minor conditions that are not considered structural defects.[37] In addition, birth defects registries that only ascertain cases of birth defects in the first year of life exclude infants who died shortly after birth without an autopsy, or diagnosed later in childhood. Moreover, our study only includes live births, whereas other studies[8,12] include data on stillbirths and terminations. Without medical record review, the FBDR data also lack information on case confirmation or case severity.

Nevertheless, we found racial/ethnic- and sex-specific differences in prevalence of specific types of CHD. More studies are needed to elucidate these differences and their importance in clinical practice. This knowledge may lead to improved understanding of these differences in prevalence of CHD and the potential causative genetic and environmental components. ■

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