Role of Health Insurance on the Survival of Infants With Congenital Heart Defects

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Objectives. We examined the association between health insurance and survival of infants with congenital heart defects (CHDs), and whether medical insurance type contributed to racial/ethnic disparities in survival.

Methods. We conducted a population-based, retrospective study on a cohort of Florida resident infants born with CHDs between 1998 and 2007. We estimated neonatal, post-neonatal, and infant survival probabilities and adjusted hazard ratios (AHRs) for individual characteristics.

Results. Uninsured infants with critical CHDs had 3 times the mortality risk (AHR = 3.0; 95% confidence interval = 1.3, 6.9) than that in privately insured infants. Publicly insured infants had a 30% reduced mortality risk than that of privately insured infants during the neonatal period, but had a 30% increased risk in the post-neonatal period. Adjusting for insurance type reduced the Black–White disparity in mortality risk by 50%.

Conclusions. Racial/ethnic disparities in survival were attenuated significantly, but not eliminated, by adjusting for payer status. (*Am J Public Health.* 2014;104: e62–e70. doi:10.2105/AJPH.2014.301969)

Major health care reform efforts have sought to improve access to health care by reducing barriers associated with the lack of or insufficient insurance coverage.[1,2] Although literature exists on the impact of insurance coverage on health care utilization, there is a relative dearth of population-based evidence on whether insurance coverage is associated with significant health outcomes, particularly among the most medically vulnerable groups, which includes children with birth defects.

Infants born with congenital heart defects (CHDs), the most common birth defect and leading cause of death among those born with birth defects, often require timely specialized surgical and medical care[3–5]; therefore, access to care and service utilization may be important predictors of survival. Although recent advances in surgical interventions have resulted in improved survival rates among infants born with CHDs, mortality remains a significant public health problem, and unexplained racial/ethnic disparities add health equity concerns.[6–9] These racial/ethnic disparities in survival suggest that identification of contributing factors could potentially lead to effective strategies to reduce CHD-related infant and childhood mortality, which has been identified as a national public health priority by *Healthy People 2020*.[10]

Some hospital-based studies have found positive associations between insurance type and postoperative mortality of infants with CHDs.[6,8,11–14] Population-based birth defects surveillance programs provide the most complete ascertainment of infants born with major birth defects in a population that, when linked with vital records, provide a more complete source of case data for survival studies. However, most published population-based studies have had only a limited ability to examine factors associated with survival.[15–22] Despite the high sensitivity and accuracy of surveillance data,[23] payer information is not typically available beyond that reported on the birth certificate.

We used population-based birth defects surveillance data, which were linked with data for each hospitalization, to obtain information on the type of health insurance used for hospitalizations initiated during the first year of life. Using these unique data, we examined the association between survival and health insurance type, and the association of health insurance type on racial/ethnic disparities in survival of infants born with CHDs.

METHODS

Our study was a retrospective, population-based cohort study of infants born in Florida from January 1, 1998, through December 31, 2007. Eligible infants were those born alive to a Florida resident mother during the study period and identified by the Florida Birth Defects Registry (FBDR) as having a CHD as determined by *International Classification of Disease, Ninth Revision, Clinical Modification* codes 745.0–747.49.[24] All those infants without a matched death certificate were assumed to be alive at the end of the study. Age at death (days) was determined by the number of days from birth date to death date on a death certificate, determined by subtracting the birth date from the date of death. Information about each infant's hospitalizations was collected and reported by participating hospitals to the Florida Agency for Health Care Administration (AHCA) as required by Florida law. The relevant AHCA data included inpatient hospital discharge information, including demographic characteristics, diagnostic coding, procedural codes, and principal payer information.[25–27] Exclusion criteria for the FBDR included out-of-state deliveries, and any adopted and prospective adopted infants. Because gestational age at less than 23 weeks often results in high mortality regardless of medical intervention, we excluded these infants from the analyses. Similarly, we also excluded those with chromosomal abnormalities because of the high fatality rate, with the exception of those with Down syndrome. Survival of infants with Down syndrome has improved significantly in recent years, particularly among those with CHDs, and the survival of infants with co-occurring Down syndrome and CHDs is similar to that for infants with only CHDs.[16,28]

In our cohort, infants with Down syndrome had a 1-year survival similar to those with isolated CHDs (95% vs 97%, respectively) and was better than those with a CHD and non-chromosomal birth defects (88%).

Variables

We determined the primary independent variable, health insurance payer type, by the reported expected principal payer for any inpatient admission during infancy and classified it into 3 categories: (1) private, including military coverage (CHAMPUS/TriCare); (2) public, including Medicare, Medicaid, KidCare, and veterans benefits; and (3) no insurance, self-pay, or underinsured, which was defined as no third party coverage or less than 30% estimated insurance coverage. For brevity, the uninsured, underinsured, and self-pay group is hereafter referred to as uninsured. We determined final insurance status for each infant by assessing changes to the payer type across all admissions during infancy and classified insurance status in 1 of 4 insurance coverage categories: private only, public only, uninsured only, or a mix (more than 1 type of payer). We determined the level of neonatal care at the birth hospital from the AHCA data and classified it as levels I to III, with level III representing the highest level of intensity and specialization of care.[29] For those infants who were transferred directly from the birth hospital to another facility, we analyzed the facility with the higher level of care.

FBDR matched case data with Florida vital records to obtain the demographic characteristics, which included maternal race/ethnicity (non-Hispanic [NH] White, NH Black, Hispanic, and other); maternal nativity, age, and education; the Adequacy of Prenatal Care Utilization Index, which is a measure of the adequacy of both initiation of and the of receipt prenatal care services (inadequate vs adequate)[30]; and infant sex, birth weight, gestational age, and plurality.

We used clinical information from the FBDR to identify the presence of CHDs and any additional noncardiac birth defects. Infants with critical CHDs require surgical or catheter intervention in the first year of life and are at risk for cardiovascular collapse or death if discharged from the birth hospital without a critical CHD diagnosis.[31] To examine whether the association between insurance and survival was stronger among those requiring more medical services, we classified infants with any of the following subtypes of CHD as having at least 1 critical CHD: hypoplastic left heart syndrome, pulmonary atresia, complete transposition of the great arteries, truncus arteriosus, tricuspid atresia, tetralogy of Fallot, total anomalous pulmonary venous return, coarctation of the aorta, doublet outlet right ventricle, Ebstein anomaly, interrupted aortic arch, and single ventricle.[31]

Analysis

We estimated infant survival probabilities by the Kaplan-Meier product-limit method,[32] and Greenwood's method was used to calculate the estimated survival probability variance and 95% confidence intervals (CIs).[33] We used a bivariate log-rank test to determine whether the survival probabilities were significantly different among nonmissing levels of maternal race/ethnicity, insurance status, and other covariates described previously.[34] Kaplan-Meier curves were visually examined to assess the survival distribution across infancy. We used Cox proportional hazard models to estimate unadjusted and adjusted hazard ratios (AHRs) for possible factors for the neonatal period (< 28 days) and post-neonatal (28–364 days) period, which were estimated assuming survival through the neonatal period.[34] Because maternal nativity was correlated with race/ethnicity and prenatal care had a high number of missing values, we dropped these variables from the model. All other variables had < 2% missing data. We also examined potential trends in the survival estimates across the study period. To identify confounders with the most influence, we grouped covariates into clinical, demographic, and health care categories. Separate models were run for each category. The effect of secular trends on the association between survival and payer type were examined. We used SAS-PC version 9.2 (SAS Institute, Cary, NC) for the computations.

RESULTS

Of the 45 144 infants identified with CHDs, 917 infants (2%) were unable to be linked with AHCA data, and 816 infants were excluded because of a gestational age of less than 23 weeks or chromosomal abnormalities other than Down syndrome. Of the final study population (n = 43 411), 46.1% of infants had private insurance, 44.3% had public insurance, 3.3% were uninsured, and 6.3% had a mix of payers during the first year of life (Table 1). Infants with at least 1 critical CHD were 8.5% (n = 3683) of the total cohort (Table 2).

Survival Estimates

The overall 1-year survival of infants with any CHD was 96.7% (95% CI = 96.5, 96.8) and was 85.3% (95% CI = 84.1, 86.4) in those with a critical CHD. Overall infant survival varied by maternal race/ethnicity, with Hispanics having the best survival (98.1%), followed by other race/ethnicity (96.9%), NH Whites (96.5%), and NH Blacks (95.3%; $P < .001$; Table 2). The Kaplan-Meier survival curves by race/ethnicity indicated that racial/ethnic disparities increased with increasing age through infancy, and racial/ethnic disparities were greater in infants with critical CHDs compared with those with noncritical CHDs (Figure A, available as a supplement to the online version of this article at http://www.ajph.org).

Infant survival varied by insurance coverage in the bivariate analysis ($P < .001$), and the Kaplan-Meier survival curves showed that the impact of insurance coverage on survival differed by CHD type and infant's age (Figure 1). Although they represented approximately 3% of the entire cohort, uninsured infants had poorer survival in early infancy, and this survival disadvantage was considerably greater among infants with critical CHDs (Figure 1).

Because the distribution of insurance type varied by race/ethnicity (i.e., non-Whites were more likely to be on public insurance), racial/ethnic disparities were examined within each insurance category. In both the private and public insurance categories, NH Blacks had poorer survival compared with Hispanic and NH Whites ($P < .004$); however, NH Whites had the lowest survival among uninsured infants (Figure B, available as a supplement to the online version of this article at http://www.ajph.org). Hispanics had the best survival for all categories of insurance, and a subanalysis showed moderate yet statistically significant ($P < .001$; $\alpha = 0.05$) differences in infant survival in the Hispanic subgroup, with Cubans having the highest (99.0%) and Mexicans having the lowest (96.8%) survival (data not shown).

TABLE 1—Selected Demographic, Clinical, and Hospital Characteristics by Insurance Status for Children With Congenital Heart Defects (CHDs): Florida, 1998–2007

		Critical CHD					Noncritical CHD				
	Total No.	Private, No. (%)	Public, No. (%)	Uninsured, No. (%)	Mix, No. (%)	P[a]	Private, No. (%)	Public, No. (%)	Uninsured, No. (%)	Mix, No. (%)	P[a]
Total	43 411	1492 (40.5)	1706 (46.3)	30 (0.8)	455 (12.4)		18 520 (46.6)	17 508 (44.1)	1399 (3.5)	2301 (5.8)	
Mother's race/ethnicity											
White	18 624	1039 (52.7)	714 (36.2)	10 (0.5)	207 (10.5)		9801 (58.9)	5591 (33.6)	438 (2.6)	824 (4.9)	
Black	10 858	166 (20.4)	515 (63.3)	8 (1.0)	124 (15.3)		2742 (27.3)	6049 (60.2)	453 (4.5)	801 (8.0)	
Hispanic	12 603	234 (29.3)	443 (55.4)	11 (1.4)	112 (14.0)	< .001	5223 (44.3)	5508 (46.7)	455 (3.9)	617 (5.2)	< .001
Other	1176	49 (53.3)	31 (33.7)	1 (1.1)	11 (12.0)		683 (63.0)	304 (28.0)	47 (4.3)	50 (4.6)	
Unknown/missing	150	4 (50.0)	3 (37.5)	0 (0.0)	1 (12.5)		71 (50.0)	56 (39.4)	6 (4.2)	9 (6.3)	
Maternal nativity											
United States	30 211	1182 (42.5)	1267 (45.6)	13 (0.5)	318 (11.4)		12 976 (47.3)	12 140 (44.3)	685 (2.5)	1630 (5.9)	
Foreign	13 161	309 (34.4)	436 (48.6)	17 (1.9)	136 (15.1)	< .001	5541 (45.2)	5343 (43.6)	711 (5.8)	668 (5.4)	< .001
Missing	39	1 (20.0)	3 (60.0)	0 (0.0)	1 (20.0)		3 (8.8)	25 (73.5)	3 (8.8)	3 (8.8)	
Maternal age, y											
< 24	14 708	215 (16.5)	884 (67.7)	13 (1.0)	194 (14.9)		2760 (20.6)	9110 (68.0)	510 (3.8)	1022 (7.6)	
25–34	21 041	931 (52.7)	642 (36.3)	9 (0.5)	185 (10.5)	< .001	11 106 (57.6)	6554 (34.0)	652 (3.4)	962 5.0)	< .001
≥ 35	7662	346 (56.7)	180 (29.5)	8 (1.3)	76 (12.5)		4654 (66.0)	1844 (26.1)	237 (3.4)	317 (4.5)	
Maternal education											
< 12 y	8190	61 (7.7)	613 (77.6)	10 (1.3)	106 (13.4)		815 (11.0)	5585 (75.5)	406 (5.5)	594 (8.0)	
HS or GED	14 743	358 (29.2)	691 (56.4)	11 (0.9)	165 (13.5)	< .001	4467 (33.0)	7579 (56.1)	540 (4.0)	932 (6.9)	< .001
≥ some college	20 210	1069 (65.1)	381 (23.2)	9 (0.5)	182 (11.1)		13 159 (70.9)	4207 (22.7)	438 (2.4)	765 (4.1)	
Missing	268	4 (14.8)	21 (77.8)	0 (0.0)	2 (7.4)		79 (32.8)	137 (56.8)	15 (6.2)	10 (4.1)	
Birth weight, g											
< 1500	3633	53 (34.4)	79 (51.3)	2 (1.3)	20 (13.0)		1191 (34.2)	1971 (56.7)	51 (1.5)	266 (7.6)	
1500–2499	5835	194 (34.4)	306 (54.3)	1 (0.2)	63 (11.2)	.004	2175 (41.3)	2539 (48.2)	154 (2.9)	403 (7.6)	< .001
≥ 2500	33 930	1244 (42.0)	1321 (44.6)	27 (0.9)	372 (12.6)		15 150 (48.9)	12 993 (42.0)	1194 (3.9)	1629 (5.3)	
Missing	13	1 (100.0)	0 (0.0)	0 (0.0)	0 (0.0)		4 (33.3)	5 (41.7)	0 (0.0)	3 (25.0)	
Additional defects											
No	40 694	1348 (41.4)	1485 (45.6)	28 (0.9)	396 (12.2)	.02	17 713 (47.3)	16 318 (43.6)	1373 (3.7)	2033 (5.4)	< .001
Yes	2717	144 (33.8)	221 (51.9)	2 (0.5)	59 (13.8)		807 (35.2)	1190 (51.9)	26 (1.1)	268 (11.7)	
Prenatal care											
Inadequate	2824	57 (15.1)	255 (67.5)	4 (1.1)	62 (16.4)		568 (23.2)	1605 (65.6)	101 (4.1)	172 (7.0)	
Adequate	35 913	1345 (44.4)	1299 (76.1)	23 (76.7)	360 (79.1)	< .001	16 809 (51.1)	13 233 (40.2)	1005 (3.1)	1839 (5.6)	< .001
Missing	4674	90 (32.4)	152 (54.7)	3 (1.1)	33 (11.9)		1143 (26.0)	2670 (60.7)	293 (6.7)	290 (6.6)	
NICU level[b]											
I	5023	120 (35.5)	150 (44.4)	3 (0.9)	65 (19.2)		1902 (40.6)	2293 (48.9)	180 (3.8)	310 (6.6)	
II	11 007	195 (46.2)	167 (39.6)	6 (1.4)	54 (12.8)	< .001	5751 (54.3)	3959 (37.4)	396 (3.7)	479 (4.5)	< .001
III	27 318	1175 (40.3)	1388 (47.6)	21 (0.7)	335 (11.5)		10 844 (44.4)	11 223 (46.0)	820 (3.4)	1512 (6.2)	
Missing	63	2 (50.0)	1 (25.0)	0 (0.0)	1 (25.0)		23 (39.0)	33 (55.9)	3 (5.1)	0 (0.0)	

Note. GED = general equivalency diploma; HS = high school; NICU = neonatal intensive care unit.
[a] χ^2 test of homogeneity across strata ($\alpha = 0.05$).
[b] Level of neonatal care at the birth hospital.

Hazard Models

Compared with private insurance, public insurance was associated with a 30% lower risk of death during the neonatal period and a 30% higher mortality risk in the post-neonatal period (Table 3). Uninsured infants with critical and noncritical CHDs had approximately 3 times and 2 times the increased neonatal mortality risk, respectively, compared with infants with private insurance. The 3 times increased neonatal mortality risk in infants with critical CHDs was largely driven by the 5 times and 6 times increased neonatal mortality among NH White and Hispanic infants, respectively. The mortality risk of those with mixed insurance was

TABLE 2—Infant Survival Probabilities of Infants With Critical and Noncritical Congenital Heart Defects (CHDs) by Selected Maternal and Infant Characteristics: Florida, 1998–2007

	Critical CHDs				Noncritical CHDs				All CHDs			
	No. of Births	No. of Deaths	Survival Probabilities, % (95% CI)	P[a]	No. of Births	No. of Deaths	Survival Probabilities, % (95% CI)[a]	P[a]	No. of Births	No. of Deaths	Survival Probabilities, % (95% CI)[a]	P[a]
Total	3683	541	85.3 (84.1, 86.4)		39 728	902	97.7 (97.6, 97.9)		43 411	1443	96.7 (96.5, 96.8)	
Mother's race/ethnicity				< .001				< .001				< .001
White	1970	266	88.5 (84.9, 87.9)		16 654	378	97.7 (97.5, 97.9)		18 624	644	96.5 (96.3, 96.8)	
Black	813	167	79.5 (76.5, 82.1)		10 045	345	96.6 (96.2, 96.9)		10 858	512	95.3 (94.9, 95.7)	
Hispanic	800	91	86.6 (85.0, 88.0)		11 803	153	98.7 (98.5, 98.9)		12 603	244	98.1 (97.8, 98.3)	
Other	92	17	81.5 (72.0, 88.1)		1084	20	98.2 (97.2, 98.8)		1176	37	96.9 (95.7, 97.7)	
Maternal nativity				.99				< .001				< .001
United States	2780	406	85.4 (84.0, 86.6)		27319	687	97.5 (97.3, 97.7)		30 211	1099	96.4 (96.1, 96.6)	
Foreign	898	132	85.3 (82.8, 87.4)		12 223	199	98.4 (98.1, 98.6)		13 161	332	97.5 (97.2, 97.7)	
Maternal age, y				.31				< .001				
< 24	1306	207	84.2 (82.1, 86.1)		13 402	395	97.1 (96.8, 97.4)		14 706	602	95.9 (95.6, 96.2)	
25–34	1767	253	85.7 (84.0, 87.3)		19 274	353	98.2 (98.0, 98.4)		21 041	606	97.1 (96.9, 97.3)	
≥ 35	610	81	86.7 (83.7, 89.1)		7052	154	97.8 (97.5, 98.1)		7662	235	96.9 (96.5, 97.3)	
Maternal education				.1				< .001				< .001
< 12 y	790	131	83.4 (80.6, 85.8)		7400	231	96.9 (96.5, 97.3)		8190	362	95.6 (95.1, 96.0)	
HS or GED	1225	186	84.8 (82.7, 86.7)		13 518	336	97.5 (97.3, 97.8)		14 743	522	96.5 (96.1, 96.7)	
≥ some college	1641	218	86.7 (84.9, 88.2)		18 569	321	98.3 (98.1, 98.5)		20 210	539	97.3 (97.1, 97.5)	
WIC[b]				.04				< .001				< .001
No	759	110	85.5 (82.8, 87.8)		11 239	216	98.1 (97.8, 98.3)		12 019	301	97.5 (97.2, 97.8)	
Yes	780	85	89.1 (86.7, 91.1)		8225	220	97.3 (97.0, 97.7)		8984	330	96.3 (95.9, 96.7)	
Infant sex				.1				.39				.33
Male	2130	295	86.2 (84.7, 87.6)		20 763	484	97.7 (97.5, 97.9)		22 893	779	96.6 (96.4, 96.8)	
Female	1553	246	84.2 (82.3, 86.0)		18 965	418	97.8 (97.6, 98.0)		20 518	664	96.8 (96.5, 97.0)	
Birth weight, g				< .001				< .001				< .001
< 1500	154	50	67.5 (59.5, 74.4)		3479	329	90.5 (89.5, 91.5)		3633	379	89.6 (88.5, 90.5)	
1500–2499	564	137	75.7 (72.0, 79.0)		5271	214	95.9 (95.4, 96.4)		5835	351	94.0 (93.3, 94.6)	
≥ 2500	2964	354	88.1 (86.9, 89.2)		30 996	357	98.9 (98.7, 99.0)		33 930	711	97.9 (97.7, 98.1)	
Gestational age, wk				< .001				< .001				< .001
20–31	162	62	62.6 (54.8, 69.6)		3787	318	91.6 (90.7, 92.4)		3953	380	90.4 (89.4, 91.3)	
32–36	577	116	79.9 (76.4, 82.9)		7257	219	97.0 (96.6, 97.4)		7834	335	95.7 (95.3, 96.2)	
37–44	2940	363	87.7 (86.4, 88.8)		28 683	365	98.7 (98.6, 98.9)		31 623	728	97.7 (97.5, 97.9)	
Plurality				.52				.001				< .001
Singleton	3567	522	85.4 (84.2, 86.5)		38 595	851	97.8 (97.6, 97.9)		42 343	1388	96.7 (96.5, 96.9)	
Multiple	115	19	83.5 (75.3, 89.1)		947	35	93.3 (94.9, 97.3)		1067	55	94.8 (93.3, 96.0)	
Additional defects				.002				< .001				< .001
No	3257	453	86.1 (84.9, 87.2)		37 437	663	98.2 (98.1, 98.4)		40 694	1116	97.3 (97.1, 97.4)	
Yes	426	88	79.3 (75.1, 82.8)		2291	239	89.6 (88.2, 90.8)		2717	327	88.0 (86.7, 89.1)	
Prenatal care				.05				< .001				< .001
Inadequate	3027	421	86.1 (84.8, 87.3)		32 886	656	98.0 (97.8, 98.2)		35 913	624	95.6 (94.9, 96.2)	
Adequate	378	67	82.3 (78.0, 85.8)		2446	118	96.9 (96.3, 97.4)		2824	638	97.0 (96.8, 97.2)	
NICU level[c]				< .001				< .001				< .001
I	338	22	93.5 (90.3, 95.7)		4685	34	99.3 (99.0, 99.5)		5023	56	98.9 (98.6, 99.1)	
II	422	24	94.3 (91.7, 96.2)		10 585	85	99.2 (99.0, 99.4)		11 007	109	99.0 (98.8, 99.2)	
III	2919	494	83.1 (81.7, 84.4)		24 399	783	96.8 (96.6, 97.0)		27 318	1277	95.3 (95.1, 95.6)	

Continued

TABLE 2—*Continued*

Payer status				.03				< .001				< .001
Private only	1492	207	86.1 (84.3, 87.8)		18 520	321	98.3 (98.1, 98.4)		20 012	528	97.4 (97.1, 97.6)	
Public only	1706	275	83.9 (82.1, 85.6)		17 508	484	97.2 (97.0, 97.5)		19 214	759	96.1 (95.8, 96.3)	
Uninsured	30	7	76.7 (57.2, 88.1)		1399	30	97.9 (96.9, 98.5)		1429	37	97.4 (96.4, 98.1)	
Combination	455	52	88.6 (85.2, 91.1)		2301	67	97.1 (96.3, 97.7)		2756	119	95.7 (94.9, 96.4)	

Note. CI = confidence interval; GED = general equivalency diploma; HS = high school; NICU = neonatal intensive care unit; WIC = Women, Infants, and Children.
[a]χ^2 test of homogeneity across strata (α = 0.05).
[b]Enrollment in Supplemental Nutrition Program for Women, Infants, and Children.
[c]Level of neonatal care at the birth hospital.

very similar to those on public insurance, a finding that was largely consistent across CHD types and racial/ethnic categories. The public–private survival difference was most notable among NH Blacks, particularly among NH Blacks with critical CHDs for whom the private–public mortality risk difference was greatest between the neonatal and post-neonatal periods. Among NH Black infants with critical CHDs, those with public insurance had a 70% reduced risk of death in the neonatal period compared with privately insured infants, but they also had a 2 times increased risk in the post-neonatal period. No change in survival over time was observed among noncritical CHDs; however, infant survival improved among critical CHDs from 84.7% in 1998 to 88.9% in 2007 (data not shown). A post hoc analysis included the addition of a variable for birth year in the models, and this inclusion did not change the mortality risk associated with insurance type.

After adjustment for factors for which survival varied in the crude survival analysis, infants born to NH Black mothers had a 20% higher mortality risk during the post-neonatal period than that of infants born to NH White mothers. There was no Black–White survival disparity in the neonatal period. Infants born to Hispanic mothers were 40% less likely to die in the post-neonatal period than infants born to NH White mothers. This difference in post-neonatal mortality was not notably attenuated by adjustment for potential confounders (data available as a supplement to the online version of this article at http://www.ajph.org). Adjusting only for factors related to health care (birth hospital level of care and insurance status) had the greatest reduction of the observed crude post-neonatal Black–White disparity, attenuating the excess Black mortality risk by 63% (data available as a supplement to the online version of this article at http://www.ajph.org).

DISCUSSION

The type of health insurance coverage of infants born with CHDs was associated with differences in infant survival, and the magnitude of the association varied by maternal race/ethnicity, CHD type, and infancy period. The most vulnerable group was infants with critical CHDs who had no insurance or who were underinsured. Although small in numbers, this group was 3 times more likely to die in the neonatal period than privately insured infants with critical CHDs. Infants with public insurance had a modestly reduced risk of death during the neonatal period compared with infants with private insurance, but experienced an increasing excess mortality risk throughout the post-neonatal period. Type of health insurance and level of neonatal care use accounted for more of the observed racial differences in mortality between NH Blacks and NH Whites than did other demographic characteristics and clinical factors. However, a higher infant mortality risk among NH Blacks persisted even after accounting for potential confounders. Also, for each type of insurance, Hispanics had the lowest infant mortality.

A surprising and somewhat paradoxical finding was the changing public–private mortality risk across infancy periods. Infants with public insurance had lower mortality risk during the neonatal period and an increased mortality risk in the post-neonatal period. Children on public insurance were shown to have poorer access to specialty care compared with privately insured children, and would be expected to be at greater risk,[35] so reasons why publicly insured infants with CHDs had better neonatal survival are not clear. One potential explanation could be that publicly insured women with CHD-affected fetuses were considered high risk and were more vigilantly referred to specialty care. If true, it be might be expected that this effect would be observed among only or more strongly for infants with critical CHDs, which were more likely to be detected prenatally.[36] Yet, the survival benefit in our study was present nearly equally among infants with critical and noncritical CHDs.

Although approximately 3% of the cohort was uninsured, this group had the greatest risk for neonatal mortality compared with infants with private insurance, and this risk difference was highest among infants with critical CHDs. A published review of the literature that examined access to medical care by children with special health care needs documented a consistent negative association between having no insurance and accessing specialty care both in terms of frequency of use and delays in obtaining care.[35] Perlstein et al. found that uninsured infants with CHDs were referred to pediatric cardiologists later than those with insurance,[37] and Chang et al. found an increasing trend in the age of surgical repair of select CHDs for infants with private, managed care, and public insurance.[38] Although lack of insurance was shown to be associated with mortality among infants of very low birth weight,[39] ours was the first study that observed that association among infants with CHDs.

The Black–White disparities in survival observed in our study were consistent with most infant[7,9,40] and in-hospital mortality studies,[6,8,41] but not all.[42] We corroborated previous work that demonstrated that racial/ethnic disparities were more apparent in the

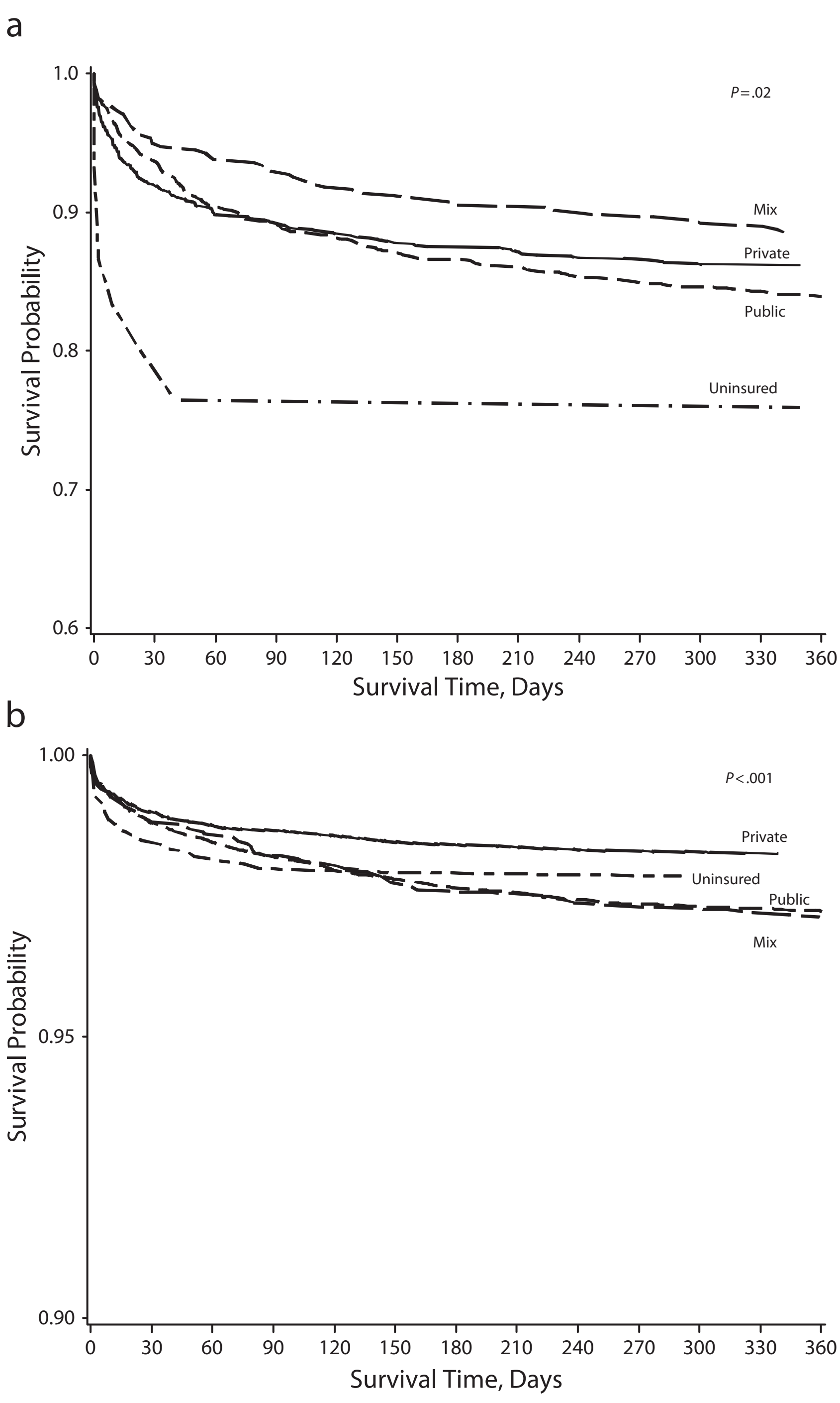

FIGURE 1—Kaplan-Meier survival curves by insurance category for infants with (a) critical congenital heart defects and (b) noncritical congenital heart defects: Florida, 1998–2007

post-neonatal period and widened with increasing age,[16] suggesting that studies that presented only overall infant survival might mask racial/ethnic disparities because a large proportion of deaths occur in the neonatal period, during which disparities were not as evident. Survival differences between Hispanics and NH Whites were less consistently reported. Although we observed improved infant survival among Hispanics, previous studies did not note a survival advantage.[7,42] A subanalysis of our population found statistically significant heterogeneity across Hispanic groups, possibly because of the unique composition of the Hispanic population in Florida, which might limit the generalizability to other states.

Our study found that indicators related to access to care, whether potential (health insurance) or realized (birth in hospitals with more specialized services),[43] accounted for more of the racial/ethnic disparities than other measured indicators. Health insurance and other access to care indicators accounted for approximately two thirds of the excess mortality risk among NH Blacks, yet some unexplained increased risk remained. Additional work to examine referral patterns and the potential that NH Blacks might be referred to hospitals with a record of higher mortality might further explain the observed racial/ethnic disparities in survival.[39,44,45]

Study Limitations

Although we used population-based birth defects surveillance data, several limitations should be considered. In contrast to active ascertainment that involves program staff actively searching data sources and abstracting information from medical records, the FBDR used passive case ascertainment that relied on administrative data from multiple data sources to identify infants with CHDs and other birth defects. These methods were less comprehensive than active ascertainment used by a limited number of other state-based programs,[46] and might produce less accurate birth prevalence estimates.[47–49] In addition, the 2% of infants with CHDs identified by FBDR that did not match with inpatient records were more likely to die during infancy and were also more likely to be born to Hispanic and NH Black mothers. Insurance status was determined by the expected payer listed at hospital discharge, because no information was available on the actual payer or whether there were multiple payers.

Another limitation of our study was the lack of information related to prenatal diagnoses, elective terminations, and clinical care during infancy. A prenatal diagnosis provides more time to plan optimal in utero and urgent postnatal surgical management, although the positive impact of a prenatal diagnosis has not been

TABLE 3—Adjusted Hazard Ratios for Insurance Status Among Infants With Congenital Heart Defects (CHDs) by Race/Ethnicity for Live Born Infants With CHDs: Florida, 1998–2007

		Total, AHR (95% CI)		Non-Hispanic White, AHR (95% CI)		Non-Hispanic Black, AHR (95% CI)		Hispanic, AHR (95% CI)	
Insurance Status	No.	< 28 Days	28–364 Days	< 28 Days	28–364 Days	< 28 Days	28–364 Days	< 28 Days	28–364 Days
All CHDs									
Public only	19 214	0.7 (0.6, 0.8)	1.3 (1.1, 1.6)	0.6 (0.5, 0.8)	1.0 (0.8, 1.4)	0.6 (0.4, 0.8)	1.7 (1.2, 2.4)	1.0 (0.6, 1.6)	1.3 (0.8, 2.1)
Uninsured	1429	1.9 (1.2, 2.8)	0.7 (0.3, 1.3)	1.8 (0.9, 3.6)	0.4 (0.1, 1.8)	1.6 (0.8, 3.0)	1.0 (0.4, 2.5)	2.6 (1.1, 6.4)	0.8 (0.2, 3.5)
Mix	2756	0.6 (0.4, 0.8)	1.2 (0.9, 1.6)	0.6 (0.4, 1.1)	1.0 (0.7, 1.6)	0.5 (0.3, 0.9)	1.6 (1.0, 2.5)	0.5 (0.2, 1.3)	1.0 (0.5, 2.1)
Critical									
Public only	1706	0.6 (0.4, 0.8)	1.3 (1.0, 1.8)	0.6 (0.4, 1.0)	0.9 (0.6, 1.4)	0.3 (0.2, 0.6)	1.9 (1.0, 3.7)	0.8 (0.4, 1.9)	1.6 (0.7, 3.6)
Uninsured	30	3.0 (1.3, 6.9)	0.7 (0.1, 5.3)	5.1 (1.6, 16.5)	. . .[a]	0.8 (0.1, 6.0)	2.0 (0.3, 15.5)	6.4 (1.3, 32.2)	. . .[a]
Mix	455	0.5 (0.3, 0.8)	1.0 (0.7, 1.5)	0.5 (0.3, 1.1)	0.8 (0.4, 1.5)	0.3 (0.2, 0.7)	1.3 (0.6, 2.8)	0.5 (0.1, 1.8)	0.7 (0.2, 2.4)
Noncritical									
Public only	17 508	0.8 (0.6, 1.0)	1.3 (1.0, 1.7)	0.6 (0.4, 0.9)	1.2 (0.8, 1.7)	0.8 (0.5, 1.2)	1.6 (1.0, 2.3)	1.1 (0.6, 1.9)	1.1 (0.6, 2.0)
Uninsured	1399	1.9 (1.2, 3.0)	0.7 (0.3, 1.5)	1.5 (0.4, 1.5)	0.6 (0.1, 2.3)	2.1 (1.0, 4.2)	0.9 (0.3, 2.5)	2.2 (0.8, 6.7)	0.9 (0.2, 4.1)
Mix	2301	0.7 (0.5, 1.1)	1.4 (1.0, 2.0)	0.8 (0.4, 1.5)	1.2 (0.7, 2.2)	0.7 (0.3, 1.3)	1.7 (0.9, 3.0)	0.7 (0.2, 2.2)	1.3 (0.5, 3.3)

Note. AHR = adjusted hazard ratio; CI = confidence interval. AHRs were adjusted for birth weight, maternal age, maternal education, plurality, hospital level of care, and presence of other birth defects. Private only was the reference insurance type for each comparison.
[a]Too few deaths to produce stable estimates.

firmly established.[50–52] Infants with more severe types of CHDs might be more likely to be electively terminated when prenatally diagnosed [53–55]; thus, differential access to prenatal care because of health insurance type and cultural predisposition toward prenatal testing and pregnancy termination might affect the survival likelihood of the live birth cohort.[56,57] Although not available for this analysis, the quality of hospital and surgical care played an important role in survival. Because health insurance also influenced access and use of specific facilities and health care providers, caution should be used against overadjustment of the quality of care when considering the association between survival and insurance because of its role as an intermediate variable in the causal pathway.[58]

Our study improved on previous studies in 4 important areas. First, the period of observation for each infant was not restricted to a single encounter within the health care system; therefore, we were able to observe the impact of health insurance status on the entire infant survival experience. Second, a more robust categorization of insurance status was determined because we had information on all Florida hospitalizations during infancy and were able to identify those infants who changed insurance types during the first year of life. Third, the data used in our study were unique in that they combined individually linked and de-duplicated data from a state-wide, population-based birth defects surveillance program with linked, longitudinal hospitalization data. Fourth, the large population size and racial/ethnic diversity allowed for more detailed stratifications that revealed complex relations between insurance type and survival. We also illustrated how birth defects surveillance data, in combination with other administrative data sets, could be used to examine survival among infants with CHD, including critical and noncritical CHDs.

Conclusions

In our state-wide study population of infants with CHDs, those with no or insufficient health care coverage had a significantly higher risk of neonatal death, indicating that lack of health insurance was a potential barrier to appropriate and life-saving medical treatment. Although efforts to enroll uninsured infants with CHDs into public insurance plans might reduce mortality, public insurance was associated with a greater post-neonatal mortality risk compared with private insurance. This factor deserves greater scrutiny to identify potential coverage gaps or other barriers to quality and timely care for publicly insured infants. Although health insurance appeared to have a role in the Black–White disparity in survival, the growing racial disparities throughout infancy among both the privately and publicly insured groups is concerning. Additional examination of the role of insurance type and race/ethnicity on referral patterns and of socioeconomic indicators is warranted to better understand what points of intervention will close the survival gap between insurance types and shed better light on the yet unexplained racial/ethnic disparities in survival. ■

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Contributors

J. E. Kucik conceptualized and designed the study, carried out the analyses, drafted the initial article, and approved the final article as submitted. C. H. Cassell, P. Donohue, C. S. Minkovitz, T. Burke, and R. S. Kirby contributed to the conceptualization and study design, reviewed and revised the article, and approved the final article as submitted. C. J. Alverson advised on and replicated the statistical analyses, reviewed and revised the article, and approved the final article as submitted. C. H. Cassell, J. P. Tanner, and J. Correia contributed to data collection and linkages, contributed to and reviewed the article, and approved the final article as submitted.

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This study received institutional review board approvals from the University of North Carolina at Charlotte, the Florida Department of Health, and the University of South Florida.

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