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Prevalence and descriptive epidemiology of infantile hypertrophic pyloric stenosis in the United States: A multistate, population-based retrospective study, 1999–2010

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Abstract

Background: Antecedents for infantile hypertrophic pyloric stenosis (IHPS) vary across studies; therefore, we conducted a multistate, population-based retrospective study of the prevalence and descriptive epidemiology of IHPS in the United States (US).

Methods: Data for IHPS cases (n = 29,554) delivered from 1999–2010 and enumerated from 11 US population-based birth defect surveillance programs, along with data for live births (n = 14,707,418) delivered within the same birth period and jurisdictions, were analyzed using Poisson regression to estimate IHPS prevalence per 10,000 live births. Additional data on deliveries from 1999–2005 from seven of these programs were analyzed using multivariable logistic regression to estimate adjusted prevalence ratios (aPR)s and 95% confidence intervals (CI)s for selected infant and parental characteristics.

Results: Overall, IHPS prevalence from 1999–2010 was 20.09 (95% CI = 19.87, 20.32) per 10,000 live births, with statistically significant increases from 2003–2006 and decreases from 2007–2010. Compared to their respective referents, aPRs were higher in magnitude for males,

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preterm births, and multiple births, but lower for birth weights <2,500 g. The aPRs for all cases increased with decreasing parental age, maternal education, and maternal parity, but decreased for parental race/ethnicity other than non-Hispanic White. Estimates restricted to isolated cases or stratified by infant sex were similar to those for all cases.

Conclusions: This study covers one of the largest samples and longest temporal period examined for IHPS in the US. Similar to findings reported in Europe, estimates suggest that IHPS prevalence has decreased recently in the US. Additional analyses supported associations with several infant and parental characteristics.

Keywords

descriptive; epidemiology; infantile hypertrophic pyloric stenosis; population-based; prevalence

1 | INTRODUCTION

Infantile hypertrophic pyloric stenosis (IHPS) is characterized by muscular hypertrophy of the pyloric sphincter, causing obstruction of the gastric outlet and projectile vomiting in the newborn. This defect usually presents at 2–12 weeks after birth, often in a previously healthy infant, with peak onset at week 5 (Jobson & Hall, 2016). IHPS is one of the most frequently treated surgical conditions in children (El-Gohary, Abdelhafeez, Paton, Gosain, & Murphy, 2018). If left untreated, IHPS can lead to dehydration, weight loss, metabolic alkalosis and, in severe cases, death (Spicer, 1982). Pyloromyotomy is the standard method of treatment for IHPS and generally has a good prognosis and low fatality rate (<1%) (reviewed in MacMahon, 2006; Tigges and Bigham, 2012).

A previous review has suggested an increased predisposition for IHPS in populations living in temperate regions of North America and Western Europe compared to those in tropical countries (Spicer, 1982). Reported prevalence estimates (per 10,000 live births) for IHPS range from 17 to 50 in the United States (US) (Applegate & Druschel, 1995; Markel, Proctor, Ying, & Winchester, 2015; Schechter, Torfs, & Bateson, 1997; St Louis et al., 2017) and Western Europe (de Laffolie, Turial, Heckmann, Zimmer, & Schier, 2012; Hedback, Abrahamsson, Husberg, Granholm, & Oden, 2001; O'Donoghue et al., 1993; Pedersen et al., 2008; Sommerfield et al., 2008; Sule, Stone, & Gilmour, 2001). Some populations in Western Europe have reported declines in IHPS prevalence across different birth periods from the 1980s through the 2000s, although others have not (de Laffolie et al., 2012; O'Donoghue et al., 1993; Pedersen et al., 2008; Sommerfield et al., 2008). A US study reported a decline in prevalence in New York State from 1983–1990 (Applegate & Druschel, 1995) and a more recent study using data from several US population-based birth defect surveillance programs showed rather stable prevalence from 1999–2007 (St Louis et al., 2017).

The recurrence of IHPS in families suggests a genetic component for this defect (reviewed in MacMahon, 2006), and analyses of infant and parental characteristics suggest a role for non-inherited factors. Specifically, studies of infant characteristics report a fourfold or higher male excess of IHPS (Applegate & Druschel, 1995; Hedback et al., 2001; Krogh et al., 2012; Lammer & Edmonds, 1987; Markel et al., 2015; Schechter et al., 1997; To, Wajja, Wales, &

Langer, 2005; Vermes, Laszlo, Czeizel, & Acs, 2016; Wang, Waller, Hwang, Taylor, & Canfield, 2008) and largely positive associations with preterm birth (Krogh et al., 2012; Schechter et al., 1997; Stark, Rogers, Eberly, & Nylund, 2015; Svenningsson, Svensson, Akre, & Nordenskjold, 2014; Wang et al., 2008), but inconclusive findings for birth weight (Applegate & Druschel, 1995; Lammer & Edmonds, 1987; Schechter et al., 1997; Wang et al., 2008), multiple births (Applegate & Druschel, 1995; Markel et al., 2015; Rider, Stevenson, Rinsky, & Feldkamp, 2013; Schechter et al., 1997; Stark et al., 2015), and month or season of birth (Dodge, 1975; Kwok & Avery, 1967; Lammer & Edmonds, 1987; Schechter et al., 1997; Zamakhshary et al., 2011).

Studies of parental characteristics report varying associations with maternal (Applegate & Druschel, 1995; Krogh et al., 2012; Markel et al., 2015; Pedersen et al., 2008; Schechter et al., 1997; Svenningsson et al., 2014; Wang et al., 2008) and paternal (Archer, Langlois, Suarez, Brender, & Shanmugam, 2007; Grewal, Carmichael, Yang, & Shaw, 2012; McIntosh, Olshan, & Baird, 1995) age at delivery. Similarly, varying associations have been reported with maternal race/ethnicity with the highest risks observed among non-Hispanic White mothers and lower risks for non-Hispanic Blacks, Hispanics (Applegate & Druschel, 1995; Lammer & Edmonds, 1987; Markel et al., 2015; Schechter et al., 1997; Wang et al., 2008), and Asians (Markel et al., 2015; Schechter et al., 1997; Wang et al., 2008). Conversely, more consistent associations have been reported for maternal education at delivery with decreasing prevalence observed with increasing education (Applegate & Druschel, 1995; Markel et al., 2015; To et al., 2005; Wang et al., 2008) and for maternal parity with decreasing prevalence observed with increasing parity (Applegate & Druschel, 1995; Dodge, 1975; Krogh et al., 2012; Schechter et al., 1997; Svenningsson et al., 2014; Wang et al., 2008). Few previous studies of infant or parental characteristics examined associations stratified by IHPS phenotype (isolated vs. multiple birth defects) or by infant sex.

The differing temporal reports of IHPS prevalence and somewhat inconsistent findings from previous descriptive studies on infant and parental characteristics suggest the need for a large study among a racially/ethnically diverse population by pooling population-based data on cases enumerated using systematic surveillance methods and comparing to population-based data on live births. To accomplish this, we used retrospective data from 11 US population-based birth defect surveillance programs to conduct a comprehensive investigation of the prevalence and descriptive epidemiology of IHPS. Our findings offer an improved understanding of the prevalence and descriptive characteristics of this defect in a large, well-described US population.

2 | METHODS

2.1 | Case enumeration and classification

Data on IHPS diagnosis among live births delivered from 1999–2010 were obtained from 11 population-based birth defects surveillance programs, representing around 35% of the deliveries in the US. Of these 11 surveillance programs, eight used active case finding (Arkansas [AR], Arizona [AZ], Georgia [GA, Metropolitan Atlanta Surveillance Program operated by the Centers for Disease Control and Prevention], Hawaii [HI], Iowa [IA], North

Carolina [NC], Oklahoma [OK], and Texas [TX]), and three used passive case finding (Colorado [CO], Florida [FL], and New York State [NY]) to enumerate IHPS cases. All but three programs (birth period[s])–AZ (1999, 2000, 2003–2004), HI (1999–2005), and NC (2003–2010)--contributed data for each year of the 12-year birth period. IHPS cases were enumerated using either the *International Classification of Diseases, 9th Edition, Clinical Modification (ICD-9-CM)* diagnosis code 750.5 or the Centers for Disease Control and Prevention/British Pediatric Association code 750.51. These diagnosis codes also were used by each program to report IHPS cases for annual birth defect surveillance reports generated by the National Birth Defects Prevention Network (NBDPN). IHPS cases with out additional, unrelated birth defects of organ structure or function were classified as isolated cases with the remain der classified as multiple cases. To help ensure complete ascertainment of IHPS cases, each program contributing data to this report included live births followed to at least one-year of age.

2.2 | Birth data

Participating surveillance programs provided de-identified birth certificate data for the birth period(s) in which they enumerated IHPS cases. Birth certificate data were used to retrieve information on selected infant and parental characteristics for all live births.

2.3 | Infant and parental characteristics

For the birth period 1999–2005, de-identified birth certificate data for infant (sex, birth weight, clinical estimate of gestational age, season and year of birth, plurality), maternal (age at delivery, race/ethnicity, education at delivery, parity—calculated as the sum of number of previous live and nonlive births) and paternal (age at delivery, race/ethnicity) characteristics for IHPS cases and live births were obtained from participating surveillance programs. We were unable to examine gestational age and birth weight as continuous variables, because only categorical data were provided by the surveillance programs for each variable. Instead, we also created a combined gestational age (weeks) and birth weight (grams) variable with four categories: (a) term birth and normal birth weight (≥ 37 weeks and >2,500 g); (b) term birth and low birth weight (≥ 37 weeks and <2,500 g); (c) preterm and normal birth weight (<37 weeks and >2,500 g); and (d) preterm and low birth weight (<37 weeks and <2,500 g).

Overall, data were available for more than 99% of live births for each descriptive characteristic, except maternal race/ethnicity (80%), and parity (75%) and paternal race/ethnicity (67%) (data not shown). For the birth period 2006–2010, only data for birth year and maternal race/ethnicity for IHPS cases and live births were available. These limited data were available for eight of the 11 surveillance programs via data submitted to the NBDPN for its annual surveillance report. Data through 2010 are the most recent IHPS data submitted to the NBDPN. Data for birth year and race/ethnicity for IHPS cases and live births delivered from 1999 to 2010 were used to estimate IHPS prevalence. The additional data available for IHPS cases and live births for 1999–2005 were used to examine the descriptive epidemiology of IHPS. The study protocol was reviewed and approved by the institutional review boards (IRBs) for each participating surveillance program, where required, as well as the IRBs at The University of Iowa and Emory University.

2.4 | Prevalence analysis

We estimated IHPS prevalence (per 10,000 live births) as the ratio of the number of cases to the number of live births for the birth period 1999–2010 for each participating surveillance program. We also examined prevalence stratified by active and passive surveillance programs. Because three programs (AZ, HI, NC) were unable to provide data for all 12 years, we declined to apply some analytic approaches, such as Joinpoint regression, across the individual program data. Instead, data were pooled across programs to estimate IHPS prevalence by birth year. A Poisson regression model with a log link function was used to estimate the prevalence for each individual surveillance program or birth year along with a 95% confidence interval (CI) for the prevalence. A separate model was implemented for each surveillance program. In order to examine longitudinal trends in prevalence from 1999—2010, a generalized linear mixed effects model assuming a Poisson distribution with a log link function was fitted for the number of IHPS cases diagnosed each birth year. Birth year, treated as a continuous linear variable, and state were considered fixed effects. A random intercept for state was included to account for clustering. Prevalence analyses were conducted using PROC GLIMMIX in SAS version (SAS Institute Inc., © 2013).

2.5 | Descriptive analysis

The descriptive epidemiology of IHPS was generated using data provided by each of the 11 surveillance programs (Figure 1); data only were available for the years 1999–2005 to conduct descriptive analyses. Cases and live births were compared on the selected infant, maternal, and paternal characteristics listed in section 2.3 using the Pearson Chi square test. Analyses were conducted separately for all cases and isolated cases only. Crude and adjusted prevalence ratios ([cPR]s and [aPR]s, respectively) and their corresponding 95% CIs also were estimated to examine associations between IHPS and infant and parental characteristics using multivariable logistic regression analysis. Adjusted analyses were limited to data from seven states (AR, AZ, GA, IA, NC, OK, and TX) that provided data for all variables. Covariables included in the model were selected if a statistically significant bivariable association ($p<.05$) was observed between IHPS and the covariable or if the covariable was shown to be associated with IHPS in previous studies. Analyses for cPRs and aPRs were conducted separately for all cases and isolated cases, and those for aPRs were stratified by infant sex. Descriptive analyses were conducted using SAS version 9.4 (SAS Insititute Inc., 2013).

3 | RESULTS

3.1 | Prevalence analysis

Our study included 14,707,418 live births and 29,554 IHPS cases from 11 US population-based birth defect surveillance programs for all or selected years from 1999–2010. Of these 11 surveillance programs, the lowest prevalence (per 10,000 live births) was observed in HI (5.52; 95% CI = 4.53, 7.00), and the highest prevalence was observed in OK (33.28; 95% CI = 31.88, 34.74) (Table 1). Combining available data across the 11 surveillance programs, the overall estimated prevalence for IHPS was 20.09 (95% CI = 19.87, 20.32). The combined prevalence estimate from the eight surveillance programs that contributed data for the entire 12-year birth period remained stable from 1999–2002, but changed significantly ($p<.05$)

from 2003–2010, showing an increase from 2003–2006, followed by a decline from 2007–2010. The combined prevalence by birth year was highest in 2006 at 25.06 and lowest in 2010 at 17.29 (Figure 2). Further stratification of data by programs that conducted active (eight programs) or passive (three programs) surveillance yielded similar trends as observed when using data across all programs (data not shown).

3.2 | Descriptive analysis

Descriptive data for the 8,390,584 live births and 16,320 IHPS cases, including 13,922 (85.3% of total) isolated cases, were available for the birth period 1999–2005 and pooled across 11 surveillance programs to analyze infant and parental characteristics (Table 2). Among all cases, the cPRs were higher for males, infants delivered at <37 weeks (preterm births), and infants from multiple gestations, but lower for those with birth weight <2,500 g; all estimates for season of birth were near unity. The cPRs also were higher for mothers or fathers <20 years of age at delivery than those 20–34 years of age, mothers with <12 years of education at delivery than those with 12 years of education, and for first-born children. Conversely, the cPRs for mothers or fathers ≥35 years of age or mothers >12 years of education at delivery were lower than their respective referents. Additionally, cPRs for mothers or fathers who were non-Hispanic Black, Hispanic, or other race/ethnicity (includes Asian/Pacific Islander, Native American, and other race/ethnicity) were lower than those who were non-Hispanic White. Findings for infant and parental characteristics for isolated cases tended to be similar to those for all cases.

The aPRs for several infant and parental characteristics tended to parallel their respective cPRs with an increase in magnitude persisting for male sex, plurality, first-born children, and mothers or fathers <20 years of age at delivery and a decrease in magnitude persisting for increasing maternal education and age at delivery, although the estimates for parity, maternal education and parental age at delivery were attenuated (Table 3). The direction of the associations for all low birth weight and all preterm gestational age did not differ from those for low birth weight among term gestational age infants and preterm gestational age among normal birth weight infants, respectively. The association for low birth weight among preterm gestational age infants was near unity (data not shown). A decrease in magnitude for aPRs persisted for birth weight <2,500 g and parental race/ethnicity other than non-Hispanic White, although the estimates for Hispanic parents were near unity. Findings for isolated cases tended to parallel those for all cases. Findings for aPRs stratified by sex were similar to findings from the main analysis (Figure 3).

4 | DISCUSSION

Our multistate, population-based retrospective study pooled data from 11 US birth defect surveillance programs to estimate the prevalence of IHPS from 1999–2010. Prevalence for all cases during this birth period was 20.09 per 10,000 live births and ranged from 5.52 in HI to 33.28 in OK. The difference in prevalence observed across surveillance programs may reflect the population demographics in these programs. As examples, the HI surveillance program monitors a higher proportion of Asian residents than the other programs included in our analyses, whereas the OK surveillance program monitors a high proportion of non-

Hispanic Whites. IHPS prevalence estimates among offspring of Asian parents have been observed to be considerably lower than those among offspring of other parental races/ethnicities (Schechter et al., 1997; Tiao, Tsai, Kuo, & Yang, 2011; Wang et al., 2008). In comparing estimates by birth year, we observed that prevalence was relatively stable through 2002, followed by statistically significant increases from 2003–2006 and then significant decreases from 2007–2010. The cPRs estimated for our descriptive analysis using data from a subset of the birth period (1999–2005) and from surveillance programs with complete information on all selected characteristics suggested a higher prevalence of IHPS among males, preterm births, multiple births, and first-born children, as well as infants born to mothers or fathers <20 years of age at delivery or whose mothers had less than a high school education at delivery. These findings tended to persist in adjusted analyses, although estimates for parental age <20 years were attenuated. Findings restricted to isolated cases and those stratified by sex were similar to findings for all cases.

Our IHPS prevalence estimates differ from another US study which reported an overall prevalence (per 10,000 live births) of 15.8 (95% CI = 15.61, 16.04) from 1999–2007, but no significant change in prevalence during this birth period (St Louis et al., 2017). Although our study and that of St Louis et al. (2017) included mostly overlapping time frames and data from multiple surveillance programs in the US, only six programs (AZ, CO, FL, GA, NY, and TX) overlapped. Differences in the racial/ethnic distributions of cases and live births included between the two studies may have contributed to the variable findings for IHPS prevalence.

Our observation of a male predominance for IHPS was consistent with previous population-based studies from the US (Applegate & Druschel, 1995; Lammer & Edmonds, 1987; Schechter et al., 1997) and European countries (Hedback et al., 2001; Krogh et al., 2012; Markel et al., 2015; Vermes et al., 2016). The inverse association we observed for birth weight <2,500 g was not directly comparable with previous studies because of differences in study methods. Conversely, the higher PRs we observed for preterm infants were consistent with previous studies that examined all IHPS cases (Krogh et al., 2012; Schechter et al., 1997) and isolated IHPS cases (Schechter et al., 1997). Even so, the seemingly paradoxical findings we observed for low birth weight and preterm birth were observed when grouping birth weight and gestational age into meaningful categories considering small and large for gestational age. Additionally, our null findings for season of birth contrast with similar previous studies that reported seasonal variation in IHPS (Kwok & Avery, 1967; Zamakhshary et al., 2011), and our finding of increased prevalence among multiple births was consistent with two other recent studies (Rider et al., 2013; Stark et al., 2015). The results of a Texas study which used data that overlapped with those used in our study generally reported similar associations for infant sex and gestational age, although the Texas study did not observe the lower prevalence in IHPS with low birth weight observed in our study, but this may be because of differences in low birth weight categorizations used between studies (Wang et al., 2008).

With regard to parental characteristics, we observed increased IHPS prevalence among infants born to mothers <20 years of age at delivery, which supported some previous studies (Markel et al., 2015; Pedersen et al., 2008). Similarly, our observation of increased

prevalence among nulliparous mothers, supported some previous studies (Applegate & Druschel, 1995; Schechter et al., 1997) as did our observation of a decreased prevalence among infants born to mothers ≥35 years of age at delivery (Applegate & Druschel, 1995; Pedersen et al., 2008; Schechter et al., 1997). Additionally, the decreased prevalence observed for mothers with race/ethnicity other than non-Hispanic White was similar to those reported in three population-based studies (Applegate & Druschel, 1995; Lammer & Edmonds, 1987; Schechter et al., 1997). The association with maternal education at delivery in our study was consistent with three previous studies (Applegate & Druschel, 1995; Markel et al., 2015; To et al., 2005). The study by Wang et al. (2008) that used data which overlapped with our study tended to report similar associations between maternal characteristics (i.e., age, race/ethnicity, education, and parity) and IHPS.

Our observation of increased IHPS prevalence associated with younger paternal age was consistent with the findings of one previous study, although the results may not be directly comparable because of differences in paternal age categorization (Grewal et al., 2012). Associations with paternal age at delivery reported in a Texas study with data that overlapped those used in our study were similar for fathers ≥35 years of age, but not for those <20 years of age at delivery (Archer et al., 2007). Additionally, the decreased prevalence we observed among infants of fathers with a race/ethnicity other than non-Hispanic White is the first examination of IHPS prevalence by paternal race/ethnicity.

Our study has several strengths. It represents one of the largest population-based studies of IHPS to date in the US, examining approximately 35% of US deliveries during the birth period included in the study. Our study also provides the most recent and longest temporal examination of IHPS prevalence estimates in the US. Along with these strengths, our study included a racially/ethnically diverse population, improving our ability to generalize findings to the US population. Other strengths include use of active case finding approaches at most of the participating surveillance programs; systematic approaches for record abstraction, including data on co-occurring birth defects; and population-based birth data from the corresponding jurisdictions for each surveillance program.

A limitation of our study was that data obtained from birth certificates may not always be reliable because of misclassification of information for gestational age (Barradas et al., 2014; Dietz et al., 2014) and race/ethnicity (Mason, Nam, & Kim, 2014), and inconsistencies that may exist in how information is collected and reported on birth certificates by different states. Another limitation was the lack of IHPS data for the entire study period from three of the 11 participating surveillance programs. Also, we lacked data on selected infant and parental characteristics from some surveillance programs, as well as data on maternal behavioral and medication exposures that may contribute to IHPS, including maternal cigarette smoking during pregnancy (Krogh et al., 2012; Leite, Albieri, Kjaer, & Jensen, 2014; Markel et al., 2015; Sorensen, Norgard, Pedersen, Larsen, & Johnsen, 2002; Svenningsson et al., 2014) and maternal use of medications during pregnancy, such as any macrolide (Ludvigsson, Lundholm, Ortqvist, & Almqvist, 2016; Lund et al., 2014), erythromycin (Cooper et al., 2002; Louik, Werler, & Mitchell, 2002), azithromycin (Eberly, Eide, Thompson, & Nylund, 2015), decongestants (Yau, Mitchell, Lin, Werler, & Hernandez-Diaz, 2013), and bendectin (Eskenazi & Bracken, 1982).

Nonetheless, more than 99% of data were available for each selected infant or parental characteristic, except maternal race/ethnicity, parity, and paternal race/ethnicity, for which only 80%, 75%, and 67% of data, respectively, were available.

Using multistate population-based data covering one of the largest sample sizes and longest temporal period examined for IHPS in the US, our findings suggest that the prevalence of this birth defect appears to have decreased in the US during 2007–2010. Our descriptive analyses supported several previously reported findings, contributing to an increased understanding of selected infant and parental antecedents of IHPS. The suggestion of a decrease in IHPS prevalence warrants its continued monitoring to see if this pattern continues and to examine additional risk factors for IHPS to help explain the changing prevalence of this defect.

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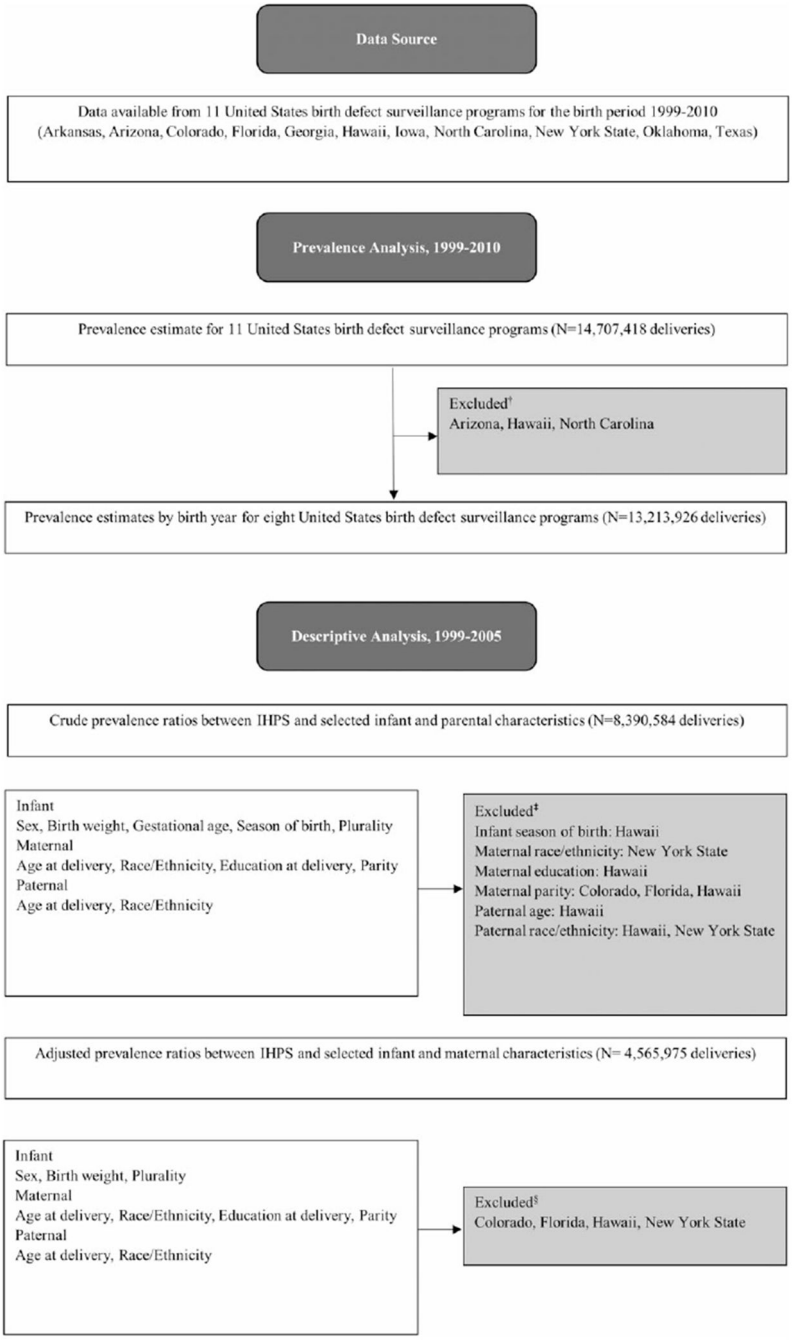

FIGURE 1.

Inclusion of state-specific population-based birth defect surveillance program data for prevalence and descriptive analysis of infantile hypertrophic pyloric stenosis, 1999–2010.†Surveillance programs excluded because of no available data for one or more birth years. ‡Surveillance programs excluded because of no available data for one or more infant or parental characteristics. §Surveillance programs excluded because of no available data for one or more infant or maternal characteristics included in the adjusted model

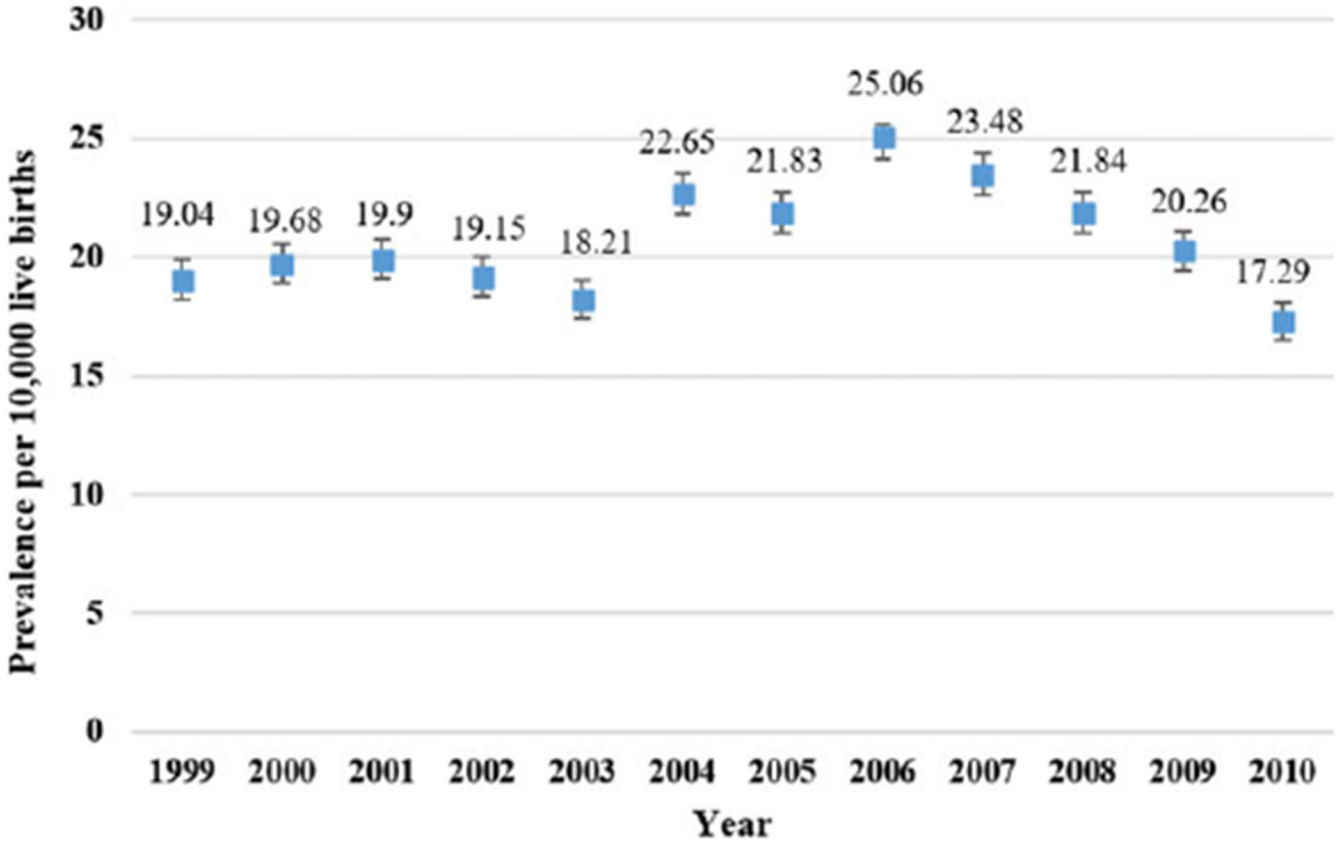

FIGURE 2.

Prevalence (per 10,000 live births) of infantile hypertrophic pyloric stenosis across eight United States population-based birth defect surveillance programs, 1999–2010.Vertical bars represent 95% confidence intervals around the prevalence estimates

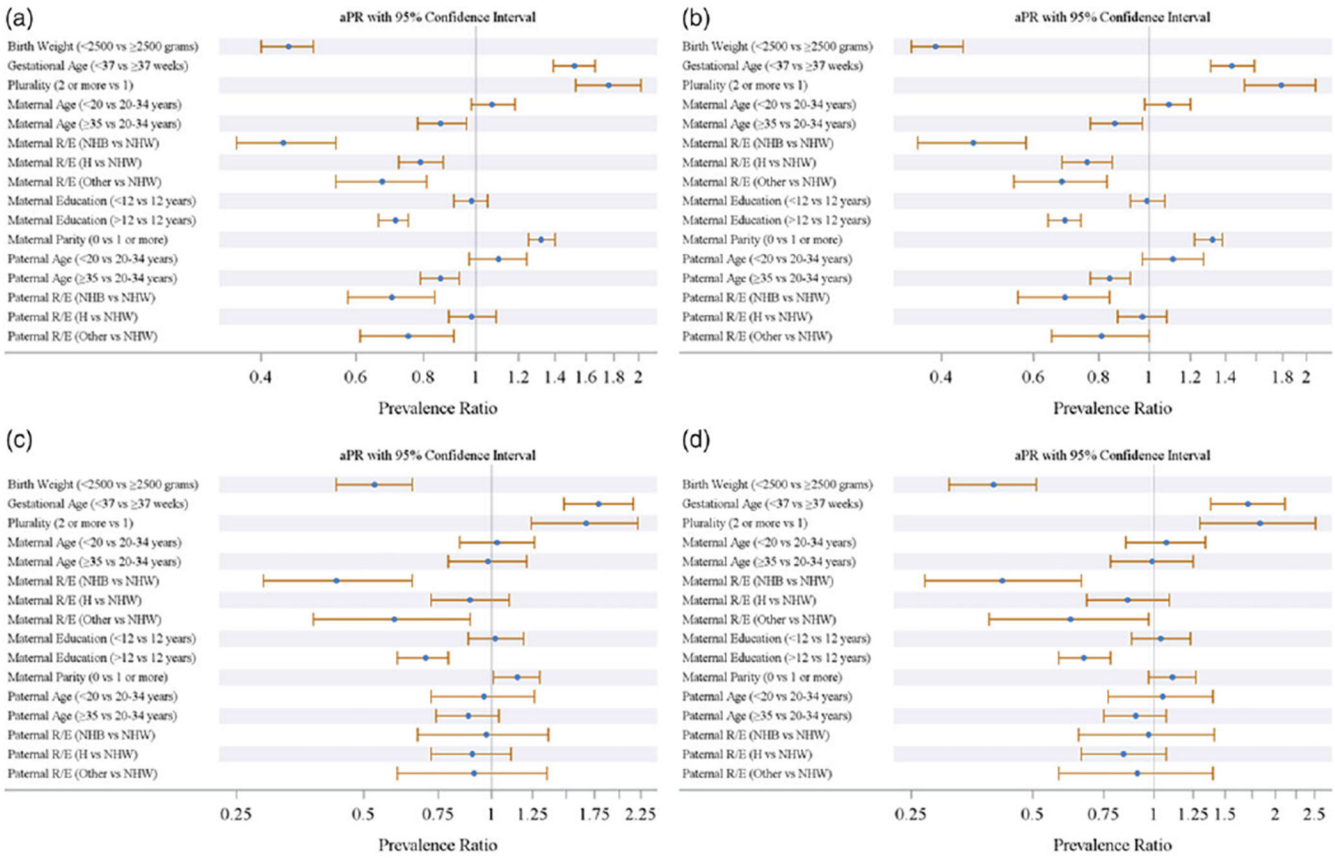

FIGURE 3.

Adjusted prevalence ratios and 95% confidence intervals for associations between selected infant and parental characteristics and (a) all male cases, (b) isolated male cases, (c) all female cases, and (d) isolated female cases of infantile hypertrophic pyloric stenosis in seven United States population-based birth defect surveillance programs[†], 1999–2005.

aPRs = adjusted prevalence ratios; H = Hispanic; NHB = Non-Hispanic Black; NHW = Non-Hispanic White; R/E = race/ethnicity. [†]includes data from Arkansas, Arizona, Georgia, Iowa, North Carolina, Oklahoma, and Texas

TABLE 1

Prevalence of infantile hypertrophic pyloric stenosis in 11 United States population-based birth defect surveillance programs, 1999–2010

Surveillance program	First birth year of data	Final birth year of data	Total years of data	Average number of IHPS cases/year	Average number of live births/year	Prevalence per 10,000 live births (95% CI)
Arkansas	1999	2010	12	49	38,484	12.80 (11.81, 13.87)
Arizona	1999	2004	4	160	92,693	17.29 (16.00, 18.68)
Colorado	1999	2010	12	104	68,081	15.29 (14.46, 16.16)
Florida	1999	2010	12	556	217,101	25.61 (25.00, 26.23)
Georgia	1999	2010	12	65	51,792	12.52 (11.67, 13.43)
Hawaii	1999	2005	7	10	17,605	5.52 (4.53, 7.00)
Iowa	1999	2010	12	111	38,951	28.58 (27.09, 30.16)
North Carolina	2003	2010	8	221	124,935	17.67 (16.86, 18.51)
New York State	1999	2010	12	480	248,433	19.32 (18.83, 19.83)
Oklahoma	1999	2010	12	173	51,936	33.28 (31.88, 34.74)
Texas	1999	2010	12	718	386,384	18.59 (18.20, 19.00)
Total						20.09 (19.87, 20.32)

Abbreviations. CI = confidence interval; IHPS = infantile hypertrophic pyloric stenosis.

TABLE 2

Infant and parental characteristics of all live births and those with infantile hypertrophic pyloric stenosis in 11 United States population-based birth defect surveillance programs, 1999–2005

Characteristics	All live births (*n* = 8,390,584)		All IHPS cases (*n* = 16,320)			Isolated IHPS cases (*n* = 13,922)		
	n	%	*n*	%	cPR (95% CI)	*n*	%	cPR (95% CI)
Infant								
Sex								
Male	4,298,604	51.23	13,236	81.66	4.24 (4.08, 4.41)	11,295	81.13	4.27 (4.09, 4.45)
Female	4,091,779	48.77	2,970	18.33	Referent	2,520	18.16	Referent
Missing	201		114			107		
Birth weight (grams)								
<2,500	935,890	11.17	1,334	8.23	0.71 (0.67, 0.75)	970	6.97	0.60 (0.56, 0.64)
≥2,500	7,443,360	88.71	14,875	91.77	Referent	12,848	92.30	Referent
Missing	11,334		111			104		
Gestational age (weeks)								
<37	863,739	10.29	1,910	11.70	1.17 (1.11, 1.22)	1,470	10.72	1.04(0.98, 1.10)
≥37	7,481,029	89.16	14,180	86.89	Referent	12,247	89.30	Referent
Missing	45,816		230			205		
Season of birth[a]								
Winter	2,018,116	24.05	3,834	24.49	0.98 (0.94, 1.02)	3,261	23.68	0.98 (0.94, 1.03)
Spring	2,047,277	24.40	3,979	24.65	Referent	3,402	24.71	Referent
Summer	2,206,353	26.30	4,334	26.83	1.01 (0.97, 1.05)	3,668	26.64	1.00(0.96, 1.06)
Fall	2,118,657	25.25	3,994	24.78	0.97 (0.93, 1.02)	3,438	24.97	0.98 (0.93, 1.02)
Missing	181		179			153		
Plurality								
1	8,077,511	96.26	15,619	96.39	Referent	13,341	96.52	Referent
2 or more	261,259	3.13	585	3.61	1.16(1.07, 1.26)	473	3.42	1.10(1.00, 1.20)
Missing	52,155		116			108		
Maternal								

Characteristics	All live births (*n* = 8,390,584)		All IHPS cases (*n* = 16,320)			Isolated IHPS cases (*n* = 13,922)		
	n	**%**	***n***	**%**	**cPR (95% CI)**	***n***	**%**	**cPR (95% CI)**
Age at delivery (years)								
<20	989,683	11.80	2,514	15.51	1.33 (1.27, 1.39)	2,142	15.50	1.32(1.26, 1.39)
20–34	6,262,742	74.65	11,983	73.95	Referent	10,235	74.09	Referent
≥35	1,136,782	13.55	1,708	10.53	0.78 (0.75, 0.83)	1,437	10.30	0.77 (0.73, 0.82)
Missing	1,377		115			108		
Race/Ethnicity[b]								
Non-Hispanic White	3,223,485	48.80	9,503	58.95	Referent	8,193	59.62	Referent
Non-Hispanic Black	916,161	13.87	1,206	7.49	0.45 (0.42, 0.47)	1,027	7.47	0.44 (0.41, 0.47)
Hispanic	2,090,796	31.65	4,926	30.51	0.80 (0.77, 0.83)	4,082	29.70	0.77 (0.74, 0.80)
Other[c]	375,261	5.68	492	3.05	0.44 (0.41, 0.49)	440	3.20	0.46 (0.42, 0.51)
Missing	1,784,881		193			180		
Education (years)								
<12	2,025,815	24.45	4,630	29.01	1.07 (1.03, 1.12)	3,948	29.00	1.08 (1.03, 1.12)
12	2,566,792	31.98	5,456	34.19	Referent	4,645	34.13	Referent
>12	3,693,245	44.49	5,874	36.80	0.75 (0.72, 0.78)	5,017	36.86	0.75 (0.72, 0.78)
Missing	104,732		360			312		
Parity[d]								
0	2,096,559	33.83	4,957	47.52	1.43 (1.38, 1.48)	4,189	42.20	1.42 (1.36, 1.48)
1 or more	4,100,012	66.17	6,775	57.48	Referent	5,775	57.68	Referent
Missing	2,194,013		4,588			3,985		
Paternal								
Age at delivery (years)								
<20	306,101	4.22	804	5.89	1.32(1.22, 1.42)	693	5.96	1.33 (1.23, 1.43)
20–34	4,998,022	68.89	9,965	73.13	Referent	8,524	73.27	Referent
≥35	1,950,938	26.89	2,861	20.98	0.73 (0.70, 0.77)	2,416	20.77	0.72 (0.69, 0.75)
Missing	1,135,523		2,690			2,289		
Race/ethnicity[b]								

Characteristics	All live births (*n* = 8,390,584)		All IHPS cases (*n* = 16,320)			Isolated IHPS cases (*n* = 13,922)		
	n	**%**	***n***	**%**	**cPR (95% CI)**	***n***	**%**	**cPR (95% CI)**
Non-Hispanic White	2,859,121	51.15	7,958	59.25	Referent	6,870	59.86	Referent
Non-Hispanic Black	666,213	11.92	1,013	7.54	0.55 (0.51, 0.58)	864	7.52	0.54 (0.50, 0.58)
Hispanic	1,756,268	31.42	4,096	30.44	0.84 (0.81, 0.87)	3,406	29.68	0.81 (0.77, 0.84)
Other[c]	308,180	5.51	373	2.77	0.43 (0.39, 0.48)	336	2.93	0.45 (0.41, 0.51)
Missing	2,800,802		2,880			2,446		

Abbreviations. CI = confidence interval; cPR = crude prevalence ratio; IHPS = infantile hypertrophic pyloric stenosis.

Percentages may not total to 100 because of rounding.

Percentages may not total to 100 because of rounding.

[a]Excludes data from Hawaii.

[b]Excludes data from New York State.

[c]Includes Asian/Pacific islanders, Native Americans, and other race/ethnic groups.

[d]Includes previous live and previous non-live births from all states except Colorado, Florida, and Hawaii.

TABLE 3

Adjusted prevalence ratios and 95% confidence intervals for associations between selected infant and parental characteristics and infantile hypertrophic pyloric stenosis in seven United States population-based birth defect surveillance programs[a], 1999–2005

Characteristics	All cases aPR (95% CI)	Isolated cases aPR (95% CI)
Infant		
Sex		
Male	4.37 (4.11, 4.64)	4.39 (4.11, 4.71)
Female	Referent	Referent
Birth weight (grams)		
<2,500	0.47 (0.43, 0.51)	0.40 (0.35, 0.44)
≥2,500	Referent	Referent
Gestational age (weeks)		
<37	1.57 (1.45, 1.69)	1.49 (1.36, 1.63)
≥37	Referent	Referent
Plurality		
1	Referent	Referent
2 or more	1.75 (1.54, 1.98)	1.80 (1.56, 2.06)
Maternal		
Age at delivery (years)		
<20	1.06 (0.98, 1.16)	1.08 (0.99, 1.19)
20–34	Referent	Referent
≥35	0.89 (0.81, 0.97)	0.88 (0.80, 0.98)
Race/ethnicity		
Non-Hispanic White	Referent	Referent
Non-Hispanic Black	0.44 (0.36, 0.53)	0.45 (0.37, 0.55)
Hispanic	0.81 (0.74, 0.88)	0.78 (0.70, 0.86)
Other[b]	0.66 (0.55, 0.78)	0.67 (0.55, 0.81)
Education (years)		
<12	0.99 (0.93, 1.05)	1.00 (0.93, 1.08)
12	Referent	Referent
>12	0.70 (0.66, 0.74)	0.69 (0.64, 0.73)
Parity[c]		
0	1.29 (1.23, 1.36)	1.26 (1.19, 1.34)
1 or more	Referent	Referent
Paternal		
Age at delivery (years)		
< 20	1.07 (0.96, 1.20)	1.10 (0.97, 1.24)
20–34	Referent	Referent

Characteristics	All cases aPR (95% CI)	Isolated cases aPR (95% CI)
≥ 35	0.86 (0.80, 0.93)	0.85 (0.79, 0.92)
Race/ethnicity		
Non-Hispanic White	Referent	Referent
Non-Hispanic Black	0.75 (0.64, 0.88)	0.74 (0.61, 0.88)
Hispanic	0.97 (0.89, 1.06)	0.94 (0.85, 1.04)
Other[b]	0.78 (0.65, 0.92)	0.83 (0.68, 1.00)

Abbreviations. aPR = adjusted prevalence ratio; CI = confidence intervals.

[a]Includes data from Arkansas, Arizona, Georgia, Iowa, North Carolina, Oklahoma, and Texas.

[b]Includes Asians/Pacific islanders, Native Americans, and other race/ethnic groups.

[c]Includes previous live and previous nonlive births.