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Distribution of Congenital Anomalies by Race/Ethnicity and Geospatial Location in Oklahoma, 1997–2009

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Abstract

Introduction: Congenital anomalies were the leading cause of infant mortality, responsible for 23% and 21% of deaths in Oklahoma and the US, respectively, in 2016. We aimed to determine the prevalence by race/ethnicity and spatial distribution of congenital anomalies to identify geographic and racial/ethnic disparities, particularly among American Indian/Alaska Natives (AI/AN).

Methods: We evaluated the prevalence of anomalies by type and race/ethnicity among 648,074 live births in Oklahoma from 1997–2009. Prevalence proportion ratios (PPR) and 95% confidence intervals (CI) were calculated using Poisson regression. We used Moran's I and Getis-Ord Gi* to evaluate spatial clustering for neural tube defects, critical congenital heart defects (CCHD), and oral clefts among births whose residence geocoded to the ZIP code or finer level.

Results: Overall prevalence of anomalies among live births was 3.9%. Non-Hispanic (NH) African American (PPR: 0.87, 95% CI: 0.83, 0.91), Asian/Pacific Islander (PPR: 0.70, 95% CI: 0.63, 0.78), and Hispanic (PPR: 0.87, 95% CI: 0.83, 0.91) children had a lower prevalence of anomalies compared to NH whites. The prevalence in NH AI/AN children was similar to NH whites (PPR: 1.01, 95% CI: 0.97, 1.05). However, differences in specific types of anomalies were observed by race/ethnicity. We observed no spatial autocorrelation for CCHD and oral clefts. Neural tube defects demonstrated spatial autocorrelation ($p < 0.0001$). Local hot-spots varied by anomaly.

Discussion: The prevalence of anomalies by race/ethnicity and geography differed by race/ethnicity and region, though this varied by anomaly. Additional research is needed to identify behavioral or environmental factors to target for prevention.

Keywords

congenital anomalies; prevalence; spatial analysis; race/ethnicity; American Indian/Alaska Native

Introduction

Congenital anomalies were the leading cause of infant mortality, responsible for 23% and 21% of deaths in Oklahoma and the US, respectively, in 2016 (United States Department of Health and Human Services, 2019). Recent studies have evaluated the prevalence of major congenital anomalies among American Indian/Alaska Natives (AI/AN) and identified increased prevalence for certain central nervous system (CNS), ear, orofacial, musculoskeletal, gastrointestinal, and chromosomal anomalies compared to whites (Canfield et al., 2014; Le, Shumate, Hoyt, Wilkinson, & Canfield, 2019; Mai et al., 2015; Marengo et al., 2018). Only one study included data from Oklahoma, though it did not provide a detailed comparison of anomalies by race/ethnicity for Oklahoma children (Mai et al., 2015). Studies have identified spatial clusters of oral clefts in Texas (Cech, Burau, & Walston, 2007) and Poland (Materna-Kiryluk et al., 2014); congenital heart defects in Wisconsin (Cronk, Gangnon, Cossette, McElroy, & Pelech, 2011), Maryland/District of Columbia (Kuehl & Loffredo, 2006), and China (Ma et al., 2014); and neural tube defects in China (Liao, Wang, Li, Guo, & Zheng, 2009; Liao et al., 2016).

To date, no studies have evaluated the prevalence of congenital anomalies in Oklahoma by both race/ethnicity and geospatial location. Because Oklahoma has the second largest AI/AN population in the US (U.S. Census Bureau, 2019b), evaluating anomalies among this population will advance the understanding of health in AI/ANs as sample sizes are frequently too small for analyses in other states.

Methods

Study Population.

Our study was comprised of all live births in Oklahoma, which included all live born children with and without birth defects who linked with an Oklahoma birth certificate from 1997–2009 (N=648,074). Children with anomalies were identified from the Oklahoma Birth Defects Registry (OBDR) (N=25,565). The OBDR is an active surveillance system that identifies children with anomalies using International Classification of Disease, Ninth Revision discharge codes 740.0–759.9 reported by all birthing hospitals in Oklahoma, including Tribal facilities. We obtained the population of live births from Oklahoma birth certificates to determine the denominator for prevalence estimates. We linked the OBDR and birth certificate datasets using first and last name and date of birth using Registry Plus™ Link Plus software v. 2.0, using both probabilistic and deterministic methods, with 95% of children linking (CDC, Atlanta, GA). The birth years (1997–2009) were selected as these were the most recent linked OBDR and birth certificate data available for analysis. We obtained information on race and ethnicity using mother's race documented on the birth certificate for all included live births. We geocoded maternal residence at delivery from the birth certificate in ArcGIS v. 10.6 using Texas A&M Geocoding Service (Texas A&M University, 2016). Institutional Review Board approval was obtained from the University of Oklahoma Health Sciences Center and the Oklahoma State Department of Health.

Statistical Analysis.

We calculated prevalence proportions, and 95% confidence intervals, of congenital anomalies per 10,000 live births overall and for specific anomalies by race/ethnicity. Similar to previous estimates, we included the following categories: central nervous system, ear, cardiovascular, orofacial, gastrointestinal, genitourinary, musculoskeletal, and chromosomal (Canfield et al., 2014; St. Louis et al., 2017). Race/ethnicity categories included non-Hispanic (NH) white (N=431,972 live births), NH African American (N=60,896), NH AI/AN (N=68,072), NH Asian/Pacific Islanders (PI) (N=13,206), and Hispanic (N=70,723). We excluded children with unknown race/ethnicity (N=3,205). We calculated prevalence as the number of anomalies among live births divided by the total number of live births in the population. Because the birth certificate included address at birth and maternal race/ethnicity information for all births, we only included live births in our prevalence estimates. We calculated prevalence proportion ratios (PPR) comparing each race/ethnic category to NH whites, and 95% confidence intervals (CI), using Poisson regression. In a sub-analysis, we also evaluated the PPRs adjusted for maternal age. We used SAS v. 9.4 for all analyses.

Spatial Analysis.

We conducted spatial analyses of congenital anomalies at the census tract level and included all singleton births whose residence at birth geocoded to the ZIP code or finer level (N=629,915). We evaluated clustering of critical congenital heart defects (CCHD), NTDs, and oral clefts based on the prevalence proportion by census tract (Table S1). CCHDs are a subset of cardiovascular anomalies identified by the OBDR, which are often life-threatening within the first few weeks of life and are more readily identified at birth than non-CCHDs (Mahle et al., 2009; Olney, Ailes, & Sontag, 2015; Tanner et al., 2015).

To determine if there was spatial autocorrelation of anomalies at the census tract level, we used Moran's I. This statistic detects clustering over the entire study area, but does not identify cluster location. We evaluated local clustering using Getis-Ord G_i^* to perform hot-spot analysis, which calculated a statistic for each individual feature based on how similar the feature was to neighboring features, adjusted for multiple testing. We generated a spatial weights matrix using the K nearest neighbors (n=8 neighbors) to conceptualize the spatial relationships of the data, which was used in both spatial autocorrelation and hot-spot analysis. This method reduces the effect of border issues in our analysis since census tracts near borders will likely have fewer neighbors (Mitchell, 2009). We used census tracts from the 2000 US Census and excluded the panhandle for our primary spatial analysis because of concerns for bias in our spatial analyses (Stewart Fotheringham & Rogerson, 1993). We also performed several sensitivity analyses: 1) including only addresses geocoded at either parcel or street segment levels; 2) including census tracts in the panhandle of Oklahoma; and 3) including only addresses geocoded at either parcel or street segment levels and including census tracts in the panhandle. We used ArcMap 10.6 for all spatial analyses.

Results

Prevalence.

The prevalence of all anomalies among live births in Oklahoma was 394 per 10,000 (95% CI: 390, 399), or 3.9%, in 1997–2009. When stratified by race/ethnicity, NH African Americans (PPR: 0.87, 95% CI: 0.83, 0.91), NH Asian/PI (PPR: 0.70, 95% CI: 0.63, 0.78), and Hispanics (PPR: 0.87, 95% CI: 0.83, 0.91) had a lower prevalence of anomalies compared to NH whites (Table 1). The prevalence in NH AI/AN children was similar to that in NH whites (PPR: 1.01, 95% CI: 0.97, 1.05). For specific types of anomalies, we observed a lower prevalence for NH African Americans for aortic stenosis, cleft lip with or without cleft palate, pyloric stenosis, hypospadias, diaphragmatic hernia, gastroschisis, and Trisomy 21 (Down syndrome) compared to NH whites. NH AI/ANs also had a lower prevalence of hypospadias compared to NH whites. However, Hispanics had a higher prevalence of anotia/microtia (ear anomalies) and Trisomy 21 but a lower prevalence of coarctation of the aorta, esophageal atresia/tracheoesophageal fistula, pyloric stenosis, and hypospadias. Although not statistically significant, there were several PPRs that were elevated for NH African Americans (common truncus and omphalocele) and NH AI/ANs (multiple types of congenital heart defects) compared to NH whites. We observed little difference in the PPRs after adjusting for maternal age (Table S2).

Spatial Analysis.

Overall, 154,322 (24%) addresses were geocoded to a street address or parcel level; 343,973 (55%) to the street segment level; and 131,620 (21%) to the ZIP code level. We observed no significant spatial autocorrelation for NTD ($p=0.46$), oral clefts ($p=0.30$), or CCHDs ($p=0.75$) in our primary analysis excluding Oklahoma panhandle census tracts (Table 2). However, when excluding addresses that geocoded to the ZIP centroid, we observed significant spatial autocorrelation for NTDs ($p<0.0001$). There was no autocorrelation when including panhandle data for any anomaly types.

In the hot-spot analysis, we observed statistically significant hot-spots for NTDs in southwestern Oklahoma at the 95% confidence level (Figure S1, Map A), CCHDs in south central Oklahoma at the 95% confidence level (Figure S2, Map A), and oral clefts in northeastern Oklahoma at the 95% and 99% confidence level (Figure S3, Map A) when excluding the panhandle. However, variation in local hot-spots was observed in our sensitivity analyses (Figures S1–S3, Maps B–D).

Discussion

Overall, minority racial/ethnic groups had lower prevalence of any anomaly compared to NH whites, though this differed for specific anomaly types. While NH AI/ANs had a similar prevalence to NH whites for anomalies overall, we observed an elevated prevalence for many congenital heart defects. Though these results were not statistically significant, the comparisons of specific anomalies were limited by small numbers of events. We also observed that the spatial distribution differed by type of anomaly in Oklahoma. Canfield et al. (2014) evaluated the prevalence of congenital anomalies by race/ethnicity from 12 birth

defects registries in the US from 1999–2007, which did not include Oklahoma. Their results were restricted to live births, as in our study, and adjusted for maternal age. Similar statistically significant results were observed for hypospadias, cleft lip/cleft palate, pyloric stenosis, and gastroschisis among NH African Americans; anotia/microtia and hypospadias for NH AI/AN; pyloric stenosis and hypospadias for NH Asian/PI; and hypospadias, anotia/microtia, coarctation of the aorta, esophageal atresia, pyloric stenosis, and Down syndrome for Hispanics compared to NH whites. To allow for comparison of our results with Mai et al. (2015), we estimated the PPR and 95% confidence intervals using the prevalence estimates reported by race/ethnicity. Our results were similar for hypospadias among Hispanics (PPR: 0.19, 95% CI: 0.12, 0.31) and NH AI/ANs (PPR: 0.72, 95% CI: 0.54, 0.98) and increased total anomalous pulmonary venous connection among NH AI/ANs (PPR: 2.31, 95% CI: 1.06, 5.01) compared to NH whites. However, the study by Mai et al. (2015) did not restrict prevalence estimates to live births and obtained maternal race/ethnicity directly from hospital birth records rather than birth certificates.

In spatial analyses excluding ZIP level geocodes, we observed significant spatial autocorrelation for NTDs with hot spots in western Oklahoma. While the reason for this result is unclear, the Hispanic population is larger in Western Oklahoma and Hispanics were reported to have lower red blood cell folate concentrations associated with NTDs in the US (Tinker, Hamner, Qi, & Crider, 2015; U.S. Census Bureau, 2019a). However, since we did not observe a significantly elevated prevalence of NTDs for Hispanics, perhaps due to the small number of events, this finding requires further investigation.

Including ZIP level geocodes may result in misclassification and identification of clusters based on Post Office (PO) box locations rather than true clusters of disease (Hurley, Saunders, Nivas, Hertz, & Reynolds, 2003). However, excluding these records may result in selection bias (Hurley et al., 2003). Due to confidentiality issues, we were unable to contact postmasters throughout the state to determine the physical address of PO Box holders, as done in Hurley et al. (2003). In future studies, we will consider geoimputation to further evaluate potential misclassification and selection bias due to inclusion/exclusion of PO Box addresses.

There are several limitations to our study that should be considered. We were restricted to live births only, which may result in an underestimate of the prevalence of specific anomalies if there are differences in fetal deaths by race/ethnicity or geographic location. The small numbers for specific anomaly types limited our analysis, particularly for NH Asian/PIs. Although the percentage of children who linked between the birth certificates and OBDR databases was high (95%), the true prevalence may be underestimated. In addition, while variations in reporting of congenital anomalies by different hospitals can result in spurious associations of spatial relationships (Forand, Talbot, Druschel, & Cross, 2002), we reduced the potential for this bias in analysis of heart defects by only including CCHDs. There is also a potential for residual confounding due to factors such as maternal diabetes status and body mass index, which were not included as covariates and likely vary by race/ethnicity and geography. Furthermore, given the number of comparisons across race/ethnicity groups and types of anomalies, results should be interpreted cautiously, bearing in mind the potential for an inflated Type I error rate.

Another limitation is the modifiable areal unit problem (MAUP) (Flowerdew, Manley, & Sabel, 2008). MAUP is a source of statistical bias that occurs when point data are aggregated to a geographic unit (i.e., specific location of birth residence to a census tract), which can result in different conclusions about relationships depending on the chosen unit of analysis. We used the prevalence proportion in each census tract to minimize this potential bias. In addition, the uncertain geographic context problem (UGCP), which states that we cannot assume how individuals interact with their surroundings in different contexts, is a potential limitation (Flowerdew et al., 2008).

In conclusion, we observed differences in the prevalence of specific congenital anomalies by race/ethnicity and by geographic location. This was the first study to evaluate racial/ethnic and geographic distribution of congenital anomalies in Oklahoma and is unique in having a large proportion of AI/ANs compared to the US (16% v. 2%) (U.S. Census Bureau, 2019b). In the future, we will collaborate with other states to better understand factors associated with the prevalence of congenital anomalies and the relationship with other health outcomes, including childhood cancer (Lupo et al., 2019).

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Prevalence per 10,000 of major congenital anomalies and prevalence ratio (95% confidence interval) by race/ethnicity in Oklahoma, 1997–2009.

Table 1.

Anomaly Type	Non-Hispanic White				Non-Hispanic African American				Non-Hispanic American Indian				Non-Hispanic Asian/Pacific Islander				Hispanic			
	N	Prev [†]	Prevalence Ratio (95% CI) [‡]	Referent	N	Prev [†]	Prevalence Ratio (95% CI) [‡]	Referent	N	Prev [†]	Prevalence Ratio (95% CI) [‡]	Referent	N	Prev [†]	Prevalence Ratio (95% CI) [‡]	Referent	N	Prev [†]	Prevalence Ratio (95% CI) [‡]	Referent
Any Type of Anomaly	17612	407.7			2149	352.9	0.87 (0.83, 0.91)		2810	412.8	1.01 (0.97, 1.05)		378	286.2	0.70 (0.63, 0.78)		2505	354.2	0.87 (0.83, 0.91)	
Central Nervous System																				
Anencephalus	52	1.20	Referent		6	0.99	0.82 (0.35, 1.91)		<6	0.73	0.61 (0.24, 1.53)		<6	1.51	1.26 (0.31, 5.17)		10	1.41	1.17 (0.60, 2.31)	
Encephalocele	36	0.83	Referent		6	0.99	1.18 (0.50, 2.81)		6	0.88	1.06 (0.45, 2.51)		<6	1.51	1.82 (0.44, 7.55)		<6	0.42	0.51 (0.16, 1.65)	
Spina Bifida	164	3.80	Referent		14	2.30	0.61 (0.35, 1.05)		18	2.64	0.70 (0.43, 1.13)		<6	2.27	0.60 (0.19, 1.87)		31	4.38	1.15 (0.79, 1.69)	
Ear																				
Anotia/microtia	58	1.34	Referent		<6	0.82	0.61 (0.25, 1.52)		12	1.76	1.31 (0.71, 2.44)		<6	2.27	1.69 (0.53, 5.40)		18	2.55	1.90 (1.12, 3.22)	
Cardiovascular																				
Coarctation of aorta	225	5.21	Referent		26	4.27	0.82 (0.55, 1.23)		27	3.97	0.76 (0.51, 1.14)		10	7.57	1.45 (0.77, 2.74)		23	3.25	0.62 (0.41, 0.96)	
Common truncus [§]	35	0.81	Referent		7	1.15	1.42 (0.63, 3.19)		8	1.18	1.45 (0.67, 3.13)		<6	0.76	0.93 (0.13, 6.82)		6	0.85	1.05 (0.44, 2.49)	
Double outlet right ventricle [§]	24	0.56	Referent		<6	0.49	0.89 (0.27, 2.94)		6	0.88	1.59 (0.65, 3.88)		<6	0.76	1.36 (0.18, 10.07)		8	1.13	2.04 (0.91, 4.53)	
Ebstein anomaly [§]	33	0.76	Referent		0	0	N/A		<6	0.73	0.96 (0.38, 2.46)		<6	0.76	0.99 (0.14, 7.25)		<6	0.42	0.56 (0.17, 1.81)	
Hypoplastic left heart syndrome [§]	89	2.06	Referent		14	2.3	1.12 (0.64, 1.96)		20	2.94	1.43 (0.88, 2.32)		<6	1.51	0.74 (0.18, 2.99)		14	1.98	0.96 (0.55, 1.69)	
Interrupted aortic arch [§]	38	0.88	Referent		<6	0.66	0.75 (0.27, 2.09)		8	1.18	1.34 (0.62, 2.86)		0	0	N/A		8	1.13	1.29 (0.60, 2.76)	
Pulmonary valve atresia and stenosis [§]	38	0.88	Referent		<6	0.66	0.75 (0.27, 2.09)		<6	0.59	0.67 (0.24, 1.87)		<6	2.27	2.58 (0.80, 8.37)		7	0.99	1.13 (0.50, 2.52)	
Single ventricle [§]	50	1.16	Referent		11	1.81	1.56 (0.81, 3.00)		12	1.76	1.52 (0.81, 2.86)		<6	2.27	1.96 (0.61, 6.29)		10	1.41	1.22 (0.62, 2.41)	

Anomaly Type	Non-Hispanic White				Non-Hispanic African American				Non-Hispanic Indian				Non-Hispanic Asian/ Pacific Islander				Hispanic			
	N	Prev [†]	Prevalence Ratio (95% CI) [‡]		N	Prev [†]	Prevalence Ratio (95% CI) [‡]		N	Prev [†]	Prevalence Ratio (95% CI) [‡]		N	Prev [†]	Prevalence Ratio (95% CI) [‡]		N	Prev [†]	Prevalence Ratio (95% CI) [‡]	
Tetralogy of Fallot [§]	182	4.21	Referent		23	3.78	0.90 (0.58, 1.38)		34	4.99	1.19 (0.82, 1.71)		<6	3.79	0.90 (0.37, 2.19)		32	4.52	1.07 (0.74, 1.56)	
Total anomalous pulmonary venous connection [§]	58	1.34	Referent		8	1.31	0.98 (0.47, 2.05)		16	2.35	1.75 (1.01, 3.04)		<6	0.76	0.56 (0.08, 4.07)		15	2.12	1.58 (0.90, 2.79)	
Transposition of the great arteries [§]	109	2.52	Referent		10	1.64	0.65 (0.34, 1.24)		23	3.38	1.34 (0.85, 2.10)		<6	2.27	0.90 (0.29, 2.83)		17	2.4	0.95 (0.57, 1.59)	
Tricuspid valve atresia and stenosis [§]	29	0.67	Referent		8	1.31	1.96 (0.89, 4.28)		<6	0.44	0.66 (0.20, 2.16)		<6	0.76	1.13 (0.15, 8.28)		7	0.99	1.47 (0.65, 3.37)	
Orofacial																				
Cleft lip +/- cleft palate	571	13.22	Referent		40	6.57	0.50 (0.36, 0.68)		105	15.42	1.17 (0.95, 1.44)		16	12.12	0.92 (0.56, 1.51)		80	11.31	0.86 (0.68, 1.08)	
Cleft lip only (without cleft palate)	190	4.40	Referent		19	3.12	0.71 (0.44, 1.14)		27	3.97	0.90 (0.60, 1.35)		6	4.54	1.03 (0.46, 2.33)		20	2.83	0.64 (0.41, 1.02)	
Gastrointestinal																				
Esophageal atresia/ tracheoesophageal fistula	132	3.06	Referent		18	2.96	0.97 (0.59, 1.58)		14	2.06	0.67 (0.39, 1.17)		<6	0.76	0.25 (0.03, 1.77)		11	1.56	0.51 (0.28, 0.94)	
Pyloric stenosis	1512	35.00	Referent		95	15.60	0.45 (0.36, 0.55)		263	38.64	1.10 (0.97, 1.26)		11	8.33	0.24 (0.13, 0.43)		207	29.27	0.84 (0.72, 0.97)	
Rectal and large intestinal atresia/ stenosis	310	7.18	Referent		32	5.25	0.73 (0.51, 1.05)		48	7.05	0.98 (0.73, 1.33)		<6	3.79	0.53 (0.22, 1.28)		48	6.79	0.95 (0.70, 1.28)	
Genitourinary																				
Hypospadias	1083	48.85	Referent		125	40.10	0.82 (0.68, 0.99)		104	30.08	0.62 (0.50, 0.75)		12	17.69	0.36 (0.21, 0.64)		56	15.53	0.32 (0.24, 0.42)	
Musculoskeletal																				
Diaphragmatic hernia	176	4.07	Referent		7	1.15	0.28 (0.13, 0.60)		29	4.26	1.05 (0.71, 1.55)		<6	1.51	0.37 (0.09, 1.50)		26	3.68	0.90 (0.60, 1.36)	
Gastroschisis	243	5.63	Referent		20	3.28	0.58 (0.37, 0.92)		45	6.61	1.18 (0.86, 1.62)		<6	2.27	0.40 (0.13, 1.26)		31	4.38	0.78 (0.54, 1.13)	
Limb deficiencies (reduction defects)	202	4.68	Referent		28	4.60	0.98 (0.66, 1.46)		30	4.41	0.94 (0.64, 1.38)		<6	2.27	0.49 (0.16, 1.52)		34	4.81	1.03 (0.71, 1.48)	
Omphalocele	78	1.81	Referent		16	2.63	1.46 (0.85, 2.49)		9	1.32	0.73 (0.37, 1.46)		<6	0.76	0.42 (0.06, 3.01)		10	1.41	0.78 (0.41, 1.51)	

Anomaly Type	Non-Hispanic White				Non-Hispanic African American				Non-Hispanic American Indian				Non-Hispanic Asian/Pacific Islander				Hispanic			
	N	Prev [†]	Prevalence Ratio (95% CI) [‡]		N	Prev [†]	Prevalence Ratio (95% CI) [‡]		N	Prev [†]	Prevalence Ratio (95% CI) [‡]		N	Prev [†]	Prevalence Ratio (95% CI) [‡]		N	Prev [†]	Prevalence Ratio (95% CI) [‡]	
Reduction deformity, lower limbs	71	1.64	Referent		<6	0.82	0.50 (0.20, 1.24)		9	1.32	0.80 (0.40, 1.61)		<6	0.76	0.46 (0.06, 3.32)		11	1.56	0.95 (0.50, 1.79)	
Reduction deformity, upper limbs	151	3.50	Referent		22	3.61	1.03 (0.66, 1.62)		22	3.23	0.92 (0.59, 1.45)		<6	0.76	0.22 (0.03, 1.55)		29	4.10	1.17 (0.79, 1.75)	
Chromosomal																				
Trisomy 13	40	0.93	Referent		<6	0.82	0.89 (0.35, 2.25)		6	0.88	0.95 (0.40, 2.25)		<6	0.76	0.82 (0.11, 5.95)		7	0.99	1.07 (0.48, 2.39)	
Trisomy 18	61	1.41	Referent		11	1.81	1.28 (0.67, 2.43)		8	1.18	0.83 (0.40, 1.74)		<6	0.76	0.54 (0.07, 3.87)		8	1.13	0.80 (0.38, 1.67)	
Trisomy 21 (Down Syndrome)	526	12.18	Referent		50	8.21	0.67 (0.50, 0.90)		71	10.43	0.86 (0.67, 1.10)		19	14.39	1.18 (0.75, 1.87)		119	16.83	1.38 (1.13, 1.69)	

[†]Prev: Prevalence per 10,000 live births

[‡]95% CI: 95% Confidence Interval

[§]Critical congenital heart defects

Table 2.

Spatial autocorrelation (Moran's I) of specific congenital anomaly types, 2000 Census Tracts, Oklahoma, 1997–2009. Data source: U.S. Census Bureau TIGER; Oklahoma Birth Defects Registry and Oklahoma Birth Certificates, Oklahoma State Department of Health.

Type of congenital anomaly	Addresses	Z score	p-value
Neural tube defects	Excluding panhandle, including ZIP level geocodes	0.75	0.46
	Excluding panhandle and ZIP level geocodes	4.89	<0.0001
	Including panhandle and ZIP level geocodes	0.90	0.37
	Including panhandle, excluding ZIP level geocodes	4.97	<0.0001
Oral clefts	Excluding panhandle, including ZIP level geocodes	1.04	0.30
	Excluding panhandle and ZIP level geocodes	0.20	0.84
	Including panhandle and ZIP level geocodes	0.86	0.39
	Including panhandle, excluding ZIP level geocodes	−0.50	0.62
Critical congenital heart defects	Excluding panhandle, including ZIP level geocodes	−0.31	0.75
	Excluding panhandle and ZIP level geocodes	−0.84	0.40
	Including panhandle and ZIP level geocodes	0.03	0.97
	Including panhandle, excluding ZIP level geocodes	−0.61	0.54