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Residential proximity to waste sites and industrial facilities and chromosomal anomalies in offspring

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Abstract

A few studies have found chromosomal anomalies in offspring associated with a maternal residence near waste sites, but did not examine the effect of living near industrial facilities, and most combined specific anomalies into heterogeneous groups. With a case–control study design, we investigated whether maternal residential proximity to hazardous waste sites or industrial facilities with chemical air emissions was associated with chromosomal anomalies in births. Maternal residences of 2099 Texas births with chromosomal anomalies and 4368 control births without documented malformations were related to boundaries of hazardous waste sites and street addresses of industrial facilities through geographic information systems. With adjustment for maternal age, race/ethnicity, and education, maternal residence within 1 mile of a hazardous waste site (relative to farther away) was not associated with chromosomal anomalies in offspring except for Klinefelter variants among Hispanic births (odds ratios (OR) 7.9, 95% confidence interval (CI) 1.1–42.4). Women 35 years or older who lived within 1 mile of industries with emissions of heavy metals were two times more likely (95% CI 1.1–4.1) than women living farther away to have offspring with chromosomal anomalies including trisomies 13, 18, or 21 or sex chromosome abnormalities. Among women 40 years or older, maternal residence within a mile of industries with solvent emissions was associated with chromosomal anomalies in births (OR 4.8, 95% CI 1.2–42.8). Study findings suggest some relation between residential proximity to industries with emissions of solvents or heavy metals and chromosomal anomalies in births to older mothers.

Keywords: Chromosomal anomalies; Hazardous waste sites; Industrial pollution; Residential exposure; Geographic information system

Background

Contaminants at waste sites and in solid waste leachates are known to induce chromosomal aberrations and DNA damage in several animal models including

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wild rats (Eckl and Riegler, 1997) and mice (Chandra et al., 2006; Tewari et al., 2006). Results from epidemiologic studies have also suggested increased risks of chromosomal anomalies in offspring of women who lived near hazardous waste sites (Geschwind et al., 1992; Dodds and Seviour, 2001; Orr et al., 2002; Vrijheid et al., 2002), although Cordier et al. (2004) found no association between such anomalies and maternal residence in communities with municipal waste incinerators.

Most of these studies have grouped chromosomal anomalies into a limited single category such as trisomy 21 or into categories so broad that they conflate heterogeneous conditions. Furthermore, previous studies have focused on residential proximity to waste sites and not on the potential risk associated with living near more general sources of industrial emissions. Yet, results of several studies indicated a relation between exposure to industrial pollution and chromosomal aberrations in both experimental animal models (Somers et al., 2002) and human populations (Major et al., 1998; Michalska et al., 1999).

In this study, we examined the relation between maternal residential proximity to hazardous waste sites and industrial air emissions, and chromosomal anomalies in offspring. We categorized chromosomal anomalies by type and also studied the effects of several classes of contaminants including solvents, heavy metals, and polycyclic aromatic hydrocarbons (PAHs).

Methods

Data for this study came from a project examining the relation between residential proximity to environmental hazards and selected congenital malformations in offspring (Brender et al., 2006a). Case and control births were selected from 1996 to 2000 births to Texas (USA) state residents. The Texas Birth Defects Registry (TBDR) conducts active birth defect surveillance by reviewing medical facility log books, hospital discharge lists, and other records. For the years 1996–1998, only selected public health regions in Texas were included in this birth defects surveillance system; since 1999, the TBDR has covered affected births occurring to all mothers residing in Texas. Although the TBDR includes spontaneous abortions and elective terminations with eligible defects in the surveillance system, we restricted this study to live births and fetal deaths (unless the termination had a vital record) because of the availability of vital records with information about maternal addresses as well as demographic characteristics.

In the TBDR, abstracts of cases with chromosomal anomalies undergo additional review by a clinical geneticist to assure appropriate classification.

Cases were included in this study if they had one or more of the British Pediatric Association (BPA) Classification of Diseases codes 758.000–758.990. Through vital record numbers supplied by the TBDR, registry cases were linked to their respective computerized live birth or fetal death records. Overall, we captured 81% of births during 1996–2000 that were identified by the registry as having chromosomal anomalies. A total of 206 live births, 66 spontaneous fetal deaths, and 258 induced terminations were excluded; these cases either did not have a live birth or fetal death certificate (induced terminations or spontaneous fetal deaths less than 20 weeks gestation) or linkage to their respective vital records was unsuccessful (live births or fetal deaths 20-weeks gestation or greater).

Chromosomal anomalies were categorized into specific diagnostic groups including trisomy 21 (BPA codes 758.000–758.090), trisomy 13 (BPA codes 758.100–758.190), trisomy 18 (BPA codes 758.200–758.295), autosomal deletion syndromes (BPA codes 758.300–758.390), balanced autosomal translocation in normal individuals (BPA code 758.400), other conditions due to autosomal anomalies (BPA codes 758.500–758.590), monosomy X variants (BPA codes 758.600–758.690), Klinefelter variants (BPA codes 758.700–758.790), and other sex chromosome anomalies (BPA codes 758.800–758.890).

A total of 4965 control births without documented congenital malformations were randomly selected for the entire project from the computerized live birth certificate files for births occurring during 1996–2000. These control records were frequency-matched to the entire sample of congenital malformations by year of birth and public health region of maternal residence. The Institutional Review Boards of the Texas Department of State Health Services, Texas State University, and Texas A&M University approved the research protocol.

Sources and methods related to environmental data are described in detail by Brender et al. (2006a). Briefly, we obtained information about National Priority List (NPL) hazardous waste sites in Texas including site characteristics and contaminants from the Agency for Toxic Substances and Disease Registry (2005) online Hazardous Substance Release/Health Effects Database (HazDat). Study staff abstracted information about state superfund sites from paper and microfilmed files stored at the Texas Commission on Environmental Quality in Austin, Texas. A total of 43 NPL sites and 70 state superfund sites were active (undergoing assessment and remediation) at the beginning of the study period.

We obtained information about industrial facilities, their locations, chemical air releases by year, and type of industry from the online Toxic Release Inventory (TRI) databases (United States Environmental Protection