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Autism spectrum disorder prevalence and proximity to industrial facilities releasing arsenic, lead or mercury

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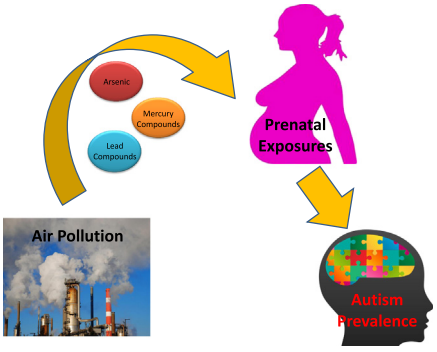
HIGHLIGHTS

- We examined associations between autism prevalence and proximity to pollutant sources.
- We found that tracts in the closest 10th percentile had higher autism prevalence.
- We found that results were still significant after adjusting for socioeconomic status.

GRAPHICAL ABSTRACT



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ABSTRACT

Prenatal and perinatal exposures to air pollutants have been shown to adversely affect birth outcomes in offspring and may contribute to prevalence of autism spectrum disorder (ASD). For this ecologic study, we evaluated the association between ASD prevalence, at the census tract level, and proximity of tract centroids to the closest industrial facilities releasing arsenic, lead or mercury during the 1990s. We used 2000 to 2008 surveillance data from five sites of the Autism and Developmental Disabilities Monitoring (ADDM) network and 2000 census data to estimate prevalence. Multi-level negative binomial regression models were used to test associations between ASD prevalence and proximity to industrial facilities in existence from 1991 to 1999 according to the US

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Environmental Protection Agency Toxics Release Inventory (USEPA-TRI). Data for 2489 census tracts showed that after adjustment for demographic and socio-economic area-based characteristics, ASD prevalence was higher in census tracts located in the closest 10th percentile compared of distance to those in the furthest 50th percentile (adjusted RR = 1.27, 95% CI: (1.00, 1.61), $P = 0.049$). The findings observed in this study are suggestive of the association between urban residential proximity to industrial facilities emitting air pollutants and higher ASD prevalence.

1. Introduction

1.1. Autism spectrum disorder

Autism spectrum disorder (ASD) is defined by the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5)* as a persistent impairment in social interaction and communication across multiple contexts that presents in early development and causes clinically significant social, educational, and occupational deficits (American Psychiatric Association, 2013). Recent surveillance studies estimate the prevalence of ASD in U.S. children to be about 1–2% (Autism and Developmental Disabilities Monitoring Network Surveillance Year, 2010 Principal Investigators, 2014; Blumberg et al., 2013). The etiology of ASD is poorly understood, but it has been hypothesized that exposure to environmental factors may trigger or enhance genetic risk (Volk et al., 2014).

1.2. Air pollutants and birth outcomes

Prenatal and perinatal exposures to air pollutants, such as carbon monoxide, nitrogen dioxide, and particulate matter, have been shown to adversely affect birth outcomes (Bell et al., 2010; Calderon-Garciduenas et al., 2011; Ezziane, 2013; Freire et al., 2010; Lakshmi et al., 2013; Munroe and Gauvain, 2012; Padula et al., 2013; Tang et al., 2014). Associated complications include developmental delay (Tang et al., 2014), congenital heart defects (Padula et al., 2013), low birth weight (Bell et al., 2010; Ezziane, 2013), cognitive deficits (Calderon-Garciduenas et al., 2011; Freire et al., 2010; Munroe and Gauvain, 2012), and mortality (Ezziane, 2013; Lakshmi et al., 2013). Prior research has shown residential proximity to point source pollution to be positively associated with congenital malformations, including chromosomal anomalies (Brender et al., 2008) and neural tube defects (Suarez et al., 2007), increased allergen-specific immunoglobulin-E in children (Patel et al., 2011), adverse birth outcomes (i.e. fetal death, preterm birth, and low birth weight) (Brender et al., 2011), and childhood brain cancer (Choi et al., 2006).

Both long-term and short-term exposures to ambient air pollutants have been shown to stimulate oxidative stress and inflammation in humans, which may also affect neurologic development (Block and Calderon-Garciduenas, 2009; Calderon-Garciduenas et al., 2009). Studies have also shown that inflammation may contribute to the pathogenesis of ASD (Enstrom et al., 2009; Li et al., 2009). Thus, inflammation may serve as a link between ASD risk and ambient air pollutant exposure. In addition, lead (Jarup, 2003; Sanders et al., 2009; Zheng et al., 2003), mercury (Aschner and Aschner, 1990; Jarup, 2003; Zheng et al., 2003), and arsenic (Jarup, 2003) are well-established neurotoxicants known to cross the blood–brain barrier and effect neurodevelopment. Mercury has been shown to have harmful effects including intellectual and developmental disabilities (Counter et al., 2002) while studies have also indicated that higher arsenic levels are associated with decreased cognitive abilities including decreased attention, comprehension, and language skills (Calderon et al., 2001), reduced intelligence quotient (IQ) scores (Wang et al., 2007; Wasserman et al., 2004; Wright et al., 2006), and diminished verbal learning and memory (Wright et al., 2006). Furthermore, lead can have adverse effects on health of children, causing behavioral and neurological problems (Bellinger, 2008; Ha et al., 2009) and reduction in IQ scores (Canfield et al., 2003).

1.3. Air pollutants and ASD

Some recent studies have investigated the relationship between ASD and exposure to ambient air pollutants (Blanchard et al., 2011; Kalkbrenner et al., 2010, 2014; Ming et al., 2008; Palmer et al., 2009; Roberts et al., 2013; Volk et al., 2011, 2014; Windham et al., 2006). Several of these studies have demonstrated associations between ASD and prenatal or perinatal air concentrations of various air pollutants, including particulate matter (Becerra et al., 2013; Kalkbrenner et al., 2010, 2014; Roberts et al., 2013; Talbott et al., 2015; Windham et al., 2006). Additionally, proximity to sources of airborne pollutants, including industrial facilities (Palmer et al., 2009), agricultural pesticides (Shelton et al., 2014), and high-traffic roadways (Volk et al., 2011), have been associated with ASD diagnosis and school-reported administrative prevalence, respectively. Based on results from these studies, observed relationships should be further investigated on a larger scale using highly reliable data. For the current study, we used surveillance data from multiple states to evaluate the association between ASD prevalence of 8-year old children at the census tract level and proximity of tract centroids to point source industrial facilities with air releases of well-known and frequently released neurotoxic substances from waste facilities, arsenic, lead, and/or mercury, during 1991 to 1999.

2. Materials and methods

2.1. Data sources

We used data from the Autism and Developmental Disabilities Monitoring (ADDM) Network, a multi-state public health surveillance system for ASD and other developmental disabilities established by the CDC in 2000 to measure ASD prevalence among 8-year-old children in 2000, 2002, 2004, 2006, and 2008. Details of ASD case definition and ascertainment have been described previously (Rice et al., 2007; Van Naarden et al., 2007); a synopsis of the ADDM methodology follows. School and health sources are queried for children who have special education exceptionalities and/or diagnoses that trigger further evaluation for ASD. ASD case status of 8-year-old children is determined through a systematic review of records from healthcare and education sources such as primary care clinics, hospitals, schools, and diagnostic and treatment centers. These records are reviewed by expert clinician reviewers to determine if behaviors are described in the abstracted data which meet the number and pattern required for an ASD diagnosis based on the *Diagnostic and Statistical Manual of Mental Disorders, 4th edition, Text Revision* (DSM-IV-TR) (American Psychiatric Association, 2000). Data for 8-year-old children identified with ASD during the ADDM surveillance years (even years) spanning from 2000 through 2008 were obtained through a data sharing agreement with the CDC for the following five participating sites: Arizona, Maryland, New Jersey, South Carolina, and Utah. These sites provided de-identified data aggregated by census tract, including total number of identified children with ASD along with race and sex distributions. It is important to note that for race distributions, virtually all Hispanics did not report race for this data source. Thus, Black and White categories exclude almost all Hispanic individuals.