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Neighborhood deprivation, race/ethnicity, and urinary metal concentrations among young girls in California

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ABSTRACT

Background: Although metals can adversely impact children's health, the distribution of exposures to many metals, particularly among vulnerable subpopulations, is not well characterized.
Objectives: We sought to determine whether neighborhood deprivation was associated with urinary concentrations of thirteen metals and whether observed relationships varied by race/ethnicity.
Methods: We obtained neighborhood characteristics from the 2005–2009 American Community Survey. Demographic information and urine samples from 400 healthy young girls in Northern California were obtained during a clinical visit. Urine samples were analyzed for metals using inductively-coupled plasma-mass spectrometry and levels were corrected for creatinine. We ran analysis of variance and generalized linear regression models to estimate associations of urinary metal concentrations with neighborhood deprivation and race/ethnicity and stratified multivariable models to evaluate possible interactions among predictors on metals concentrations.
Results: Urinary concentrations of three metals (barium, lead, antimony) varied significantly across neighborhood deprivation quartiles, and four (barium, lead, antimony, tin) varied across race/ethnicity groups. In models adjusted for family income and cotinine, both race/ethnicity ($F_{3,224} = 4.34$, $p = 0.01$) and neighborhood deprivation ($F_{3,224} = 4.32$, $p = 0.01$) were associated with antimony concentrations, but neither were associated with lead, barium, or tin, concentrations. Examining neighborhood deprivation within race/ethnicity groups, barium levels ($p_{interaction} < 0.01$) decreased with neighborhood deprivation among Hispanic girls ($p_{trend} < 0.001$) and lead levels ($p_{interaction} = 0.06$) increased with neighborhood deprivation among Asian girls ($p_{trend} = 0.04$).
Conclusions: Our results indicate that children's vulnerability to some metals varies by neighborhood deprivation quartile and race/ethnicity. These differential distributions of exposures may contribute to environmental health disparities later in life.

1. Introduction

Metals are ubiquitous chemical elements that arise from both natural and anthropogenic sources and can have adverse health effects at high levels of exposure (Agency for Toxic Substances & Disease Registry (ATSDR), 2008; Occupational Safety and Hazard Administration (OSHA)). Some metals have been identified as known, probable, or possible carcinogens (International Agency for Research on Cancer (IARC), 2013). Children may be particularly vulnerable to these environmental toxicants due to their smaller bodies and immature metabolic pathways (Landrigan and Miodovnik, 2011). Exposure during childhood to environmental chemicals may contribute to the health disparities observed among various racial and ethnic groups and thus merit research attention (Rosen and Imus, 2007).

General population studies suggest a range of health effects from metals exposure, including positive associations between urinary barium and indices of obesity (Padilla et al., 2010), cadmium and all-cause and cardiovascular mortality (Tellez-Plaza et al., 2012), and antimony, cadmium, cobalt, and tungsten exposure with cerebrovascular disease

Abbreviations: CYGNET, Cohort Study of Young Girls' Nutrition, Environment, and Transitions; KPNC, Kaiser Permanente of Northern California.

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(Agarwal et al., 2011). In addition to their classification as human carcinogens, cadmium and arsenic are also associated with kidney damage (Järup, 2003) and respiratory, dermal, reproductive, and neurological effects (Jomova et al., 2011). The limited epidemiological evaluations of tin exposure suggest a link with occupational lung disease (Chonan et al., 2007). In children specifically, metals exposure is most frequently associated with neurodevelopmental toxicity. Several studies report associations between antimony, cadmium, chromium, arsenic, lead, manganese, mercury, and/or nickel and autism spectrum disorder (Palmer et al., 2009; Roberts et al., 2013; Windham et al., 2006). Exposure to lead has also consistently been linked to neurocognitive deficits such as attention deficit disorder (Kim et al., 2013) and lowered IQ (Lanphear et al., 2005), as well as behavioral problems such as aggression and delinquency (Olympio et al., 2009). Adverse effects from manganese exposure include impairments in motor skills, intelligence, and olfactory function (Grandjean and Landrigan, 2014; Roels et al., 2012). For most heavy metals, exposures to which may be concomitant due to common sources, the published data suggest a lack of knowledge around factors driving individual susceptibility (Karagas et al., 2012).

Knowledge of the severe effects of lead and other metals on children's health has resulted in regulations to reduce exposure, including the removal of lead from gasoline and paint, and remediation of indoor sources, such as lead-based paint (Mauss, 1994; Needleman, 2004). Nevertheless, a primary source of childhood exposure to lead is ingestion of household lead-based paint chips or dust (ATSDR, 2007b; Levin et al., 2008). Household paint also contains other potentially toxic metals, such as cobalt and manganese (Fjelsted and Christensen, 2007; Mielke et al., 2001). Living in dilapidated housing has been linked to lead exposure (Hood, 2005), and the age of housing stock is the primary determinant of differential exposure (Needleman, 2004; Oyana and Margai, 2007). In addition to deteriorating paint, traffic and industrial activities have also been linked to the presence of lead and other trace metals in urban environments (Akkus and Ozdenerol, 2014; Wong et al., 2006).

Blood lead levels in the U.S. population have decreased dramatically over the past 40 years, but remain significantly higher among Black children and those living in poverty (Environmental Protection Agency (EPA), 2013). Levels of other metals such as antimony, thallium, cadmium, and mercury have also been found to be elevated in racial/ethnic minority populations (Belova et al., 2013; Said and Hernandez, 2015). Consideration not only of physical environmental hazards, but also of individual and social stressors, is critical to an enhanced understanding of the disparities in vulnerability to environmental exposures (Gee and Payne-Sturges, 2004; Morello-Frosch et al., 2011). Despite calls for increased attention to the social determinants of environmental health (Wright, 2009), very few quantitative studies (Hicken et al., 2012) have explored how socio-environmental processes contribute to the differential distribution of exposures associated with health disparities (Burger and Gochfeld, 2011; Schwartz et al., 2011). In this study, we sought to understand social patterns of metals exposure by exploring neighborhood deprivation and race/ethnicity as determinants.

2. Theory

Whereas traditional exposure pathway models focus on the source and routes of exposure, dose, and biological characteristics of the exposed population, the multilevel stress-exposure-disease model proposes that individual-level and community-level stressors contribute to differential distributions of exposures (i.e., vulnerability) and differential health impacts of exposures (i.e., susceptibility) (Gee and Payne-Sturges, 2004; Gochfeld and Burger, 2011; Payne-Sturges and Gee, 2006). This model is consistent with cumulative risk approaches that recognize the potential for social stressors to contribute to the adverse effects of environmental toxicants (EPA, 2003; Sexton and Linder, 2010).

Using the stress-exposure-disease model as a framework, the current study examines whether urinary biomarkers of exposure to thirteen metals vary across levels of a community-level stressor, neighborhood deprivation, and whether exposure vulnerability is similar for girls from various racial/ethnic groups. We do not address exposure-disease associations; rather, we focus on predictors of differential vulnerability to better understand the relationships between stressors and metals exposures. As shown in Fig. 1, neighborhood deprivation is conceptualized as impacting vulnerability through 1) direct associations with environmental hazards and exposures (e.g., paint, air pollution, food environment), and 2) indirect associations with internal and biologically-effective doses through psychosocial stress resulting from community and individual stressors. Psychosocial stress may link social conditions with environmental hazards by influencing internal dose through increased absorption and/or compromised defense systems (Gee and Payne-Sturges, 2004). Higher levels of stress, assessed by both self-report and physiological measures, have been documented among racial/ethnic populations (Borders et al., 2015; Byrd, 2012; Dowd et al., 2014; Juster et al., 2010; Merkin et al., 2009; Morello-Frosch et al., 2011; Peek et al., 2010). Thus, in this study, race/ethnicity serves as a proxy for individual-level stressors associated with being a member of a minority group. We hypothesized that urinary metals concentrations would be highest among girls living in areas with the highest levels of neighborhood deprivation, and that these associations would be most pronounced among Black and Hispanic girls.

3. Materials and methods

3.1. Participants and procedures

The Cohort Study of Young Girls' Nutrition, Environment, and Transitions (CYGNET) is a longitudinal study aimed at exploring the influence of environmental factors on pubertal development, in order to further understand the etiology of breast cancer within the Breast Cancer and the Environment Research Program (Hiatt et al., 2009). One of three similar studies, recruitment and enrollment details are described elsewhere (Biro et al., 2013). In brief, a Kaiser Permanente Northern California (KPNC) database was used to identify all current female members between the ages of 6–8 years in 2005–2006 who lived in Marin or San Francisco counties or select East Bay communities in Contra Costa and Alameda Counties, CA at the time of recruitment and at the time of birth. Girls who had been diagnosed with precocious puberty or other endocrinological conditions known to influence pubertal timing were excluded. Families of these girls received printed materials describing the study and inviting their participation; those who planned to move in the near future were ineligible. A total of 444 girls and their caregivers enrolled in the CYGNET study. The study received approval from Institutional Review Boards at the University of California San Francisco, the University of California at Berkeley, Kaiser Permanente Northern California, and the Centers for Disease Control and Prevention (CDC).

3.2. Data collection

Data and biospecimens were collected at study visits that occurred at one of three Kaiser Permanente clinic sites in Oakland, San Francisco, or San Rafael. At the first study visit, which occurred between June 2005 and August 2006, data were collected by interview with the primary caregiver, and through anthropometry and pubertal maturation assessment of the child participant as previously described (Biro et al., 2013). The primary caregiver (more than 90% were the child's mother) provided demographic information and an updated home address; the latter was used to derive neighborhood deprivation measures.

Urine was collected and stored using materials that were determined to be metal-free by the CDC. A urine sample was collected at the time of study visit and stored temporarily in a refrigerator. At the