Obesity rates in the United States have increased sharply during the past 20 years and remain at an all-time high. Current estimates suggest that more than one third of adults (approximately 39 percent) and approximately 18 percent of children in the United States are obese (Centers for Disease Control and Prevention 2017). More important, recent research suggests that the high level of obesity prevalence among adults and children has remained unchanged since 2003 and that obesity affects virtually all ages, races, sexes and socioeconomic groups (Newbold 2010; Ogden et al. 2014). These alarming figures indicate a “health epidemic” among the American population that has resulted in the rise of obesity-related illnesses such as type 2 diabetes, cardiovascular diseases, and hypertension (Flegal et al. 2012).

The cause of the obesity epidemic has traditionally been framed as an individual problem stemming from a person’s inability to regulate the balance between energy intake and expenditure. This energy balance theory suggests that obesity in the United States has increased because (1) caloric intake, especially of saturated fats, has increased, and (2) physical activity has decreased (Trasande et al. 2009). Contemporary research has broadened our understanding of obesity, and we now know that obesity is most likely caused by complex interactions among behavioral, genetic, environmental, and social factors (Hyman 2010).

Sociologists and public health experts have been instrumental in uncovering social determinants of health and illness (Link and Phelan 1995; Marmot and Wilkinson 1999). With regard to obesity, social science scholars have established strong associations between an individual’s weight and his or her social status, specifically socioeconomic status (Wilkinson and Marmot 2003), race and ethnicity (Laveist 2002; Chang, Hiller, and Metha 2009), and sex and gender (Rosengren and Lissner 2008; Zhang and Wang 2004).

Integrating Sociological Perspectives into Obesogenic Research: Associations between Air Pollution Exposure and Obesity Prevalence across U.S. Metropolitan Statistical Areas

Jessica L. Eckhardt¹

Abstract
Obesogenic theories suggests that obesity risk can be influenced by exposure to toxic chemicals present in built and natural environments. Although physical scientists have been on the forefront of obesogenic research, social science perspectives have been absent in understanding the relationship between environmental pollution and obesity risk. To address such gaps, the author uses a sociological perspective to explore the way in which exposure to a specific class of obesogens, endocrine disruptors, influences adult obesity prevalence. Using air pollution emissions data from the National Air Toxics Assessment and health data from the Behavioral Risk Factor Screening Survey, the author assesses the association between emission exposure and obesity risk across metropolitan areas in the United States. Although the nonsignificant findings do not support obesogenic hypotheses, this research demonstrates the need for obesogenic investigation using large, nationally represented data sets that can be stratified to identify inequalities in pollution exposure and associated obesity risk.

Keywords
environmental health, obesogens, air pollution, obesity, environmental inequality

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Additional obesity drivers have been identified by contemporary research on environmental inequality (Gray et al. 2018), the political economy (Fox et al. 2018), and contextual environments, such as neighborhoods (Kawachi and Kim 2016). With strong evidence that social conditions are risk factors for obesity, health sociologists are beginning to study the efficacy of obesity interventions (Ditlevsen 2018), how public policy influences body weight (Carey et al. 2017; Harder 2017), and the complex interactions between social contexts, genetics, and the natural environment (Liu and Guo 2015). The aim of this study is to further our understanding of how social conditions and environmental drivers work in concert to influence body weight. Using a nascent theoretical perspective known as obesogenics, I examine how environmental factors, specifically air pollution, may contribute to adult obesity prevalence in the United States.

The Obesogenic Hypothesis

A new, growing body of research suggests that chemical toxins play a leading role in the etiology of obesity; these chemicals are referred to as obesogens and can induce increased fat mass in humans (Grun and Blumberg 2009). In a landmark paper, Baillie-Hamilton (2002) argued that exposure to chemicals correlates with rising obesity rates (NCD Risk Factor Collaboration 2017) by showing that the global obesity epidemic coincides with significant increases of industrial chemicals in the environment over the past 40 years. Expanding on this work, Grun and Blumberg (2009) proposed the “environmental obesogens” hypothesis, which espouses that environmental pollutants can disrupt and interfere with the body’s metabolic and fat storage processes. Specifically, exposure to obesogens “promotes adiposity by altering programming of fat cell development [and] increasing energy storage in fat tissue” (Janesick and Blumberg 2016:556). These two potential pathways link exposure to chemicals found in the built and natural environment to excess fat storage and obesity in humans.

The first pathway involves adipogenesis, which is the process that creates fat cells. Fat cells are one of the body’s largest energy reserves, and they play a crucial function in keeping energy available and balanced within the body. In humans, adipogenesis begins in utero and remains high during adolescence but begins to taper off during the life course (Spaulding et al. 2008). Obesogens have been found to interfere with and misregulate the critical pathways involved in adipogenesis. In their review, Grun and Blumberg (2009) highlighted research demonstrating how chemicals such as bisphenol-A (BPA) inhibit nuclear hormone receptor signaling pathways during adipogenesis, which can cause an increase in fat cell production and, subsequently, obesity. In another review, Janesick and Blumberg (2016) concluded that “increased adipogenesis during early development permanently establishes an elevated fat cell number in adulthood” (p. 563). If this pathway exists, whereby obesogenic exposure early in the life course permanently increases the fat cell number within an individual, its implications are serious; diet, exercise, and surgery cannot reduce the number of fat cells one accumulates during the early stages of life.

Although the number of fat cells within the body is a risk factor for obesity, research suggests that the size of fat cells may be more important in determining weight than the number of fat cells. Spaulding et al. (2008) found that adult weight gain and loss are largely a result of changes in fat cell size and that the number of fat cells is largely independent of body mass index (BMI). This supports prior research suggesting that obesity mechanisms are complex and that by and large, obesity is a product of both increased adipose cell number and increased cell size (Salans et al. 1973). The same obesogenic pathway mentioned above involving environmental chemical exposure interfering with fat cell creation may also interfere with energy storage within fat cells, producing larger fat cells and, consequently, obesity (Heindel et al. 2015). Because obese individuals have greater numbers of fat cells and larger fat cells, the implications of obesogenic mechanisms are profound and a very real public health threat (Janesick and Blumberg 2016; Salans et al. 1973).

Endocrine-disrupting Chemicals as Obesogens

Obesogenic research has identified a subclass of chemicals that affect the body’s metabolic functions by interfering with endocrine system processes. The endocrine system produces hormones that regulate how the energy in fat cells is used and stored. Endocrine-disrupting chemicals (EDCs) are toxins capable of interfering with hormone signaling processes throughout the body, which can lead to diabetes mellitus (Diamanti-Kandarakis et al. 2009) and other endocrine-related diseases (reviewed in Heindel, Newbold, and Schug 2015; Janesick and Blumberg 2016). As obesogens, EDCs are thought to increase the risk for obesity in individuals by the aforementioned pathways: by increasing the number of fat cells and/or the storage of fat in existing cells (Holc9tamp 2012). Additionally, EDCs have been shown to modify metabolic rate (Heindel 2003), interfere with hormones that signal hunger and satiety (La Merrill and Birnbaum 2011), and alter digestive bacteria that promote food storage in the gut (Snedeker and Hay 2012), all of which can increase obesity risk.

Obesogens are not limited to endocrine disrupters specifically, but to date, the most conclusive toxicological findings establishing a causal link between chemical exposure and metabolic disruption have involved endocrine disruptors (Navas-Acien et al. 2008; Tang-Péronard et al. 2008). EDCs are ubiquitous in both natural and built environments, and common EDCs include industrial chemicals such as BPA and polychlorinated biphenyls (PCBs), herbicides such as atrazine, pervasive environmental pollutants including particulate matter, and naturally occurring heavy metals such as mercury and lead (Ahearn 2012; Schug et al. 2011). Because EDCs
exist in many molecular forms, such as plasticizers, fuels, chemicals, and pesticides, they are often used to produce common consumer goods, and over the past few decades, exposure to EDCs has become increasingly widespread (Diamanti-Kandarakis et al. 2009).

Concern about exposure to EDCs is also becoming more widespread as evidence connecting EDCs to health issues related to fertility, cancer, metabolism, and obesity mounts. Within the past decade, there has been a push by consumers and health advocates to increase regulations on EDCs in effort to protect human health. One of the most well known examples of consumers demanding industry changes involves the EDC BPA in plastic water bottles. As a plasticizer agent used to produce polycarbonate plastics and epoxy resin, BPA is one of the most commonly produced chemicals in the world (Vandenberg et al. 2007). Our everyday lives are full of polycarbonate plastics products such as baby bottles, water bottles, plastic cutlery, and plastic toys (Centers for Disease Control and Prevention 2012). Additionally, epoxy resin is used to line and seal aluminum in canned foods and to produce dental sealants. It has been found that BPA can leach into the contents of the plastic product (Vandenberg et al. 2007) or be absorbed through the skin (World Health Organization 2013). BPA leaching is so ubiquitous that in a study of 2,517 participant urine samples, the Centers for Disease Control and Prevention (CDC) (2012) found detectable levels of BPA in nearly every participant, which “indicates widespread exposure to BPA in the U.S. population” (p. 186). In response to findings such as these, consumers demanded that plastic water bottles be made BPA free, and in the past decade, many producers began making BPA-free baby bottles, water bottles, and canned food products.

**Airborne EDCs**

As noted in the above example of BPA leaching, there are different routes through which toxic chemicals such as EDCs can enter the body and produce disease susceptibility. There are three major routes of chemical exposure in humans: through the skin, the digestive tract, and the respiratory tract (Lauwerys and Hoet 2001). Exposure to EDCs via dermal absorption and ingestion has been the focus of most epidemiological research to date. For example, several studies have detected EDCs, including EDC substances banned in the European Union, in everyday cosmetic products that contact the skin, such as lotions, deodorants, hair shampoo, and conditioner (Gimeno et al. 2012; Llompart et al. 2013). Scholars have also found strong associations between the migration of EDCs in food packaging products to food items themselves, which can be ingested by the consumer (Pérez-Palacios et al. 2012; Suciu et al. 2013).

Inhalation is the exposure pathway that has been the least studied. Some EDCs possess molecular traits that enable them to exist in the atmosphere and be absorbed through inhalation (Teil et al. 2016). The size of these particles is directly linked to their potential to cause health problems. Small particles, 10 µm in diameter or less, have the ability to cause the most damage because they can migrate deep into the lungs and potentially into the bloodstream (Salgueiro-González et al. 2015). Once in the bloodstream, inhaled EDCs act similarly to those absorbed through the skin or ingestion by interfering with endocrine processes. In their extensive review, Giulivo et al. (2016) suggest that phthalates, a class of EDCs that includes polyvinyl chloride, are among the most common outdoor and indoor airborne EDCs. This is significant because the few studies linking EDCs to obesity have largely focused on phthalates.

For example, Buser, Murray and Scinicariello (2014) found positive associations between phthalate concentrations and obesity among children, adolescents, and adults. The authors found sex and age differences between metabolic concentrations of certain phthalates and obesity prevalence; specifically, men had generally higher odds of obesity compared with women with similar EDC exposure levels. Zhang et al. (2014) also found different effects of phthalate exposure on obesity for girls and boys in China; a positive association between EDC urine concentration and obesity was found among male children, whereas EDC exposure was negatively associated with girls’ obesity. Although not solely focused on phthalates, this study examines airborne EDCs and obesity prevalence.

**Integrating Sociological Perspectives into Obesogenic Research**

Although considerable progress has been made in understanding the role toxic chemical exposure plays in obesity, obesogenic research must be considered in the context of two significant limitations. First, obesogenic research has been conducted largely by natural scientists. Toxicology and endocrinology have been the leading fields of research probing how chemicals affect weight maintenance mechanisms in the body (Newbold 2010). Establishing the causal link between chemical exposure and disrupted bodily systems has been crucial, but social science has been absent in exploring how social processes, such as socioeconomic status, race, and ethnicity, mediate the effect chemical exposure has on human health (Vafeiadi et al. 2015).

Second, large-sample population comparisons are noticeably absent in obesogenic research. Comparing the association between chemical exposure and obesity prevalence among different populations could reveal patterns and relationships important to our understanding of obesogenics. Specifically, analyzing pollution exposure at multiple levels and across populations would allow researchers to identify under what conditions localized pollution exposure is more important than general, broad-scale pollution exposure and vice versa.
Sociology is uniquely equipped to address these gaps in the literature. Methodologically, environmental and health sociologists have a rich tradition of undertaking large comparative study designs to examine issues related to pollution exposure (Downey, Crowder, and Kemp 2016; Downey and Hawkins 2008; Zwickl, Ash, and Boyce 2014; Ringquist 2005; Mohai and Saha 2007), environmental racism (Bowen 2002; Grady 2012; Pellow 2000), and obesity (Sobal, Rauschenbach, and Frongillo 2003). Theoretically, foundational theories in environmental inequality and health disparities research can help explain obesogenic patterns regarding both pollution exposure and differential weight outcomes associated with exposure. Pellow’s (2000) framework of environmental inequality could be especially helpful in shedding light on social conditions that place certain populations at a higher risk for exposure to obesogenic chemicals. Additionally, Marmot’s (Marmot and Wilkinson 1999) social determinates of health and Link and Phelan’s (1995) fundamental causes of disease theories could help elucidate why certain groups (e.g., men vs. women, whites vs. non-whites) experience differential obesity outcomes with the same degree of obesogenic exposure.

Using a sociological lens, in the present study I address gaps in obesogenic research by using multilevel analyses to examine the association between airborne EDC exposure and adult obesity prevalence across U.S. metropolitan areas. This large-scale comparative study is, to my knowledge, the first of its kind. On the basis of findings from previous obesogenic research, I develop the following hypotheses:

**Hypothesis 1:** Increased exposure to annual concentrations of airborne EDCs is associated with greater probability of being obese.

**Hypothesis 2:** Increased exposure to annual concentrations of airborne EDCs is associated with greater probability of being morbidly obese.

**Hypothesis 3:** The effects of exposure to annual concentrations of airborne EDCs on obesity and morbidity obesity will differ by gender.

**Methods**

**Data**

**Behavioral Risk Factor Surveillance System.** Individual-level demographic and health data for the present study were collected from the 2005 Behavioral Risk Factor Surveillance System (BRFSS) (Centers for Disease Control and Prevention 2014). The BRFSS is a longitudinal survey project randomly administered via telephone to households across the United States by the CDC. The BRFSS collects individual-level data for adults 18 years and older in all 50 states. Survey questions are designed to ascertain preventive health practices and risk behaviors associated with infectious and chronic diseases. The CDC weighted the data to provide estimates that are representative of each state’s population. The design and characteristics of BRFSS are described in greater detail elsewhere (Centers for Disease Control and Prevention 2005). To protect participant privacy, publicly assessable 2005 BRFSS data are aggregated to the level of metropolitan statistical area (MSA) and denoted as the BRFSS SMART data set. MSAs are geographic areas mapped every 10 years by the Office of Management and Budget in preparation for the decennial census. MSAs consist of two or more adjacent counties that share an urban core, have a population greater than 50,000 each, or have a high degree of economic and social integration (U.S. Census Bureau 2016). For example, the 2005 Salt Lake-Utah MSA was composed of both Salt Lake County and Toole County.

**National Air Toxics Assessment.** Air pollution emissions data was obtained from the Environmental Protection Agency’s (EPA) (2016) online database National Air Toxics Assessment (NATA). The NATA emissions data are compiled from a variety of sources, including state and local air pollutant inventories, the EPA’s Toxic Release Inventory database, and emissions estimates from the EPA’s Office of Transportation and Air Quality. This compiled National Emissions Inventory is then used to model and estimate annual ambient concentrations of air toxics for each county; dispersion modeling developed by the EPA uses emissions and meteorological data to simulate the behavior and movement of air toxics in the atmosphere (modeling methodology detailed in Environmental Protection Agency 2011). In 2005, NATA estimated 177 of 187 air toxins listed under the 1990 Clean Air Act Amendment. Last, NATA data are detailed by two source types; point-source and non-point-source emissions. Point-source emissions are derived from a stationary location such as a factory smokestack or sewage treatment plant. Non-point-source emission sources are mobile sources that include automobiles, wildfire smoke, and sediment kick-up from mining and construction areas (Environmental Protection Agency 2012). County-level ambient air toxin concentration estimates were aggregated to the level of MSA to match data obtained from the BRFSS.

**Sample**

**BRFSS.** In total, 202,904 records were initially retrieved from the 2005 BRFSS SMART data set. Because of a lack of exposure data from NATA (see the following subsection), observations from Wyoming and Alaska were excluded from the final sample. Additionally, women pregnant during the survey period were excluded from the initial BRFSS sample selection criteria, because their body weight may be more sensitive to the effects of pollution exposure (Fudvoye, Bourguignon, and Parent 2014), and gestational weight gain is a known confounder in obesity studies (Snijder et al. 2012). Individuals who were underweight with BMI less than 18.5 kg/m² (n = 3,312) or extremely obese with BMI >
toxic estimations for 153 MSAs were used in this analysis. Point-source and non-point-source ambient air mates were added together to produce more robust exposure concentrations were so small that all seven concentration estimates (micrograms per cubic meter) for the seven EDCs were collected and aggregated to the MSA level for all states except Alaska and Wyoming, which lacked pollution exposure data (detailed later). An analytical sample of 188,252 individuals was generated.

**NATA.** Because prior obesogenic research has found strong associations between EDCs and obesity, estimated ambient concentration of air toxins was limited to seven known EDCs (Arner et al. 2010; Grun and Blumberg 2009; Janesick and Blumberg 2011; Vandenberg et al. 2012): five insecticides or fungicides (chlordane, hexachlorobenzene, hexachlorocyclohexane, methoxychlor, and toxaphene), one polychlorinated organic compound (PCBs), and one phthalate (bis(2-ethylhexyl)phthalate (DEHP)). Characteristics of each toxin are summarized in Table 1. County-level annual ambient concentration estimates (micrograms per cubic meter) for the seven EDCs were collected and aggregated to the MSA level for all states except Alaska and Wyoming, which lacked sufficient emissions data for NATA modeling processes. Additionally, individual estimates of ambient air toxic concentrations were so small that all seven concentration estimates were added together to produce more robust exposure variables. Point-source and non-point-source ambient air toxic estimations for 153 MSAs were used in this analysis.

**Measures**

**Dependent Variables.** BMI was computed as self-reported weight in kilograms divided by the square of self-reported height in meters. Obesity was measured by a categorical variable indicating obesity status as either obese (BMI ≥ 30 kg/m²) or morbidly obese (BMI ≥ 35 kg/m²), consistent with CDC (2015) guidelines and previous studies (Fan, Wen and Kowaleski-Jones 2016; Li et al. 2015; Ogden et al. 2014).

**MSA EDC Exposure Variables.** Airborne EDC pollution exposure was assessed using annual emission concentration estimates (micrograms per cubic meter) for point-source, non-point-source, and total (point-source plus non-point-source) emission sources. These total estimates are a summation of concentration estimates for the seven EDCs discussed earlier and are measured as continuous variables. Exposure measures were standardized to account for nonlinearity and heteroscedasticity (Long and Freese 2006). Analyzing non-point-source and point-source pollution estimates separately can be helpful in assessing cumulative impacts of air pollution exposure (Morello-Frosch, Pastor, and Sadd 2001; Morello-Frosch et al. 2011) and potentially identify which environmental hazards pose the most health risks, which has profound policy implications (Linder et al. 2008).

**Individual-level Control Variables.** On the basis of past research, individual demographic covariates used in the analysis include age and its square (Flegal et al. 2012), sex (Wang and Beydoun 2007; Grun and Blumberg 2009), and marital status (Sobal et al. 2003). Socioeconomic status was measured with two indicators, annual household income and level of education. Binary-coded household income categories include less than $15,000, $15,001 to $25,000, $25,001 to $35,000, $35,001 to $50,000 and more than $50,000. Approximately 12 percent of the sample did not report income, and unknown income is used as the reference group in these analyses. Education variables were also binary coded and include less than high school, high school graduate, some college, and college degree or more (reference category). Race and ethnicity was a categorical variable comparing whites (reference group), Hispanics, blacks, Asians, Native Hawaiians/Pacific Islanders, American Indians/Alaskan Natives, and individuals who identify as unlisted races.

**Analyses**

A multilevel modeling approach was used to explore the association between individual obesity status and airborne EDC pollution exposure at the metropolitan level. Multilevel logistic regression is often used by scholars examining the etiology of obesity by incorporating neighborhood characteristics and individual-level risk factors (Xu, Wen, and Wang 2015; Wang, Wen, and Xu 2013; Wen and Maloney 2011). The hierarchical structure of the data has two levels: individuals (level 1, n = 188,252) nested within counties (level 2, n = 153). Two-level random-intercept logistic regression analyses were performed using Stata version 13 (StataCorp, College Station, TX; Rabe-Hesketh and Skrondal 2008).

**Results**

Table 2 shows the descriptive statistics for the full sample. Approximately 24 percent of adults included in the sample are classified as obese, while 8 percent are classified as morbidly obese. Roughly 76 percent of the sample is white, 6 percent is Hispanic, and 8 percent is black. Asians, Native

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**Table 1.** List of Endocrine-disrupting Chemicals Analyzed in Multiple Regression.

| Chemical                          | Commercial Use                          |
|----------------------------------|-----------------------------------------|
| Chlordane                        | Insecticide and fungicide               |
| Hexachlorocyclohexane            | Insecticide and fungicide               |
| Methoxychlor                     | Insecticide and fungicide               |
| Toxaphene                        | Insecticide and fungicide               |
| Hexachlorobenzene                | Fungicide                               |
| Polychlorinated biphenyls        | Electronics, plasticizer                |
| Bis(2-ethylhexyl)phthalate(DEHP) | Plasticizer                             |

60 kg/m² (n = 9,457) or had missing BMI data (n = 418) were excluded from the sample (Brown et al. 2013). Listwise deletion on relevant covariates was conducted, and 517, 749, and 419 observations were dropped from the sample because of missing data for level of education, marital status, and BMI, respectively. Finally, individuals from Alaska and Wyoming (n = 222) were excluded from the sample because of missing pollution exposure data (detailed later). An analytical sample of 188,252 individuals was generated.
Hawaiians/Pacific Islanders, American Indians/Alaskan Natives, multiracial individuals, and those who listed “other race” each make up less than 3 percent of the sample. The median annual household income of respondents in the sample is $48,329, and approximately 63 percent attended college. At the MSA level, the non-point-source estimated ambient concentration of EDCs ranges from −0.67 to 6.22 µg/m³ and point-source estimated ambient concentration ranges from −0.23 to 7.21 µg/m³.

Table 3 presents odds ratios (ORs) from the multilevel models for EDC concentrations on individual risk for obesity for non-point-source and point-source pollution concentration estimates. Both non-point-source and point-source airborne pollution concentrations are nonsignificant but negatively associated with obesity (OR = 0.999 and OR = 0.998 respectively, \( p > 0.05 \)). The effects of all individual-level variables are consistent across all models. Age is positively associated with the odds of obesity, while the square of age is negative and significant, suggesting that the age-obesity trend reverses after reaching a certain age (Xu et al. 2015). Each level of education and income category is significant and positively associated with obesity. With regard to race, being black (OR = 1.81, \( p < .001 \)), Hispanic (OR = 1.12, \( p < .001 \)), Native Hawaiian or Pacific Islander (OR = 1.56, \( p < .001 \)), or American Indian/Alaskan Native (OR = 1.33, \( p < .001 \)) is associated with higher odds of obesity, while being Asian (OR = 0.38, \( p < .001 \)) is associated with a lower risk for obesity compared with whites. Marital status is statistically nonsignificant, but being female is a significant predictor of lower obesity odds (OR = 0.957, \( p < .001 \)).

Table 4 shows odds ratios for morbid obesity and EDC concentration estimates for both pollution source types. Although statistically nonsignificant, non-point-source estimates are negatively associated with morbid obesity, while point-source concentration estimates are positively associated with morbid obesity. Individual-level results from Table 3 assessing obesity are very similar to findings using morbid obesity as the dependent variable, with a few
notable exceptions. First, marital status is significant, and being married or in a partnership (OR = 0.918, \(p < .001\)) is associated with lower odds of morbid obesity compared to single individuals. Additionally, the significantly positive association between being Hispanic and obesity is rendered insignificant when assessing morbid obesity.

Tables 5 and 6 report gender-specific results for obesity. When models were estimated separately by gender, the findings showed that non-point-source ECD concentration estimates were not significantly associated with obesity for women or men. Although nonsignificant, point-source pollution estimates were negatively associated with obesity for women and positively associated with obesity for men. Age, level of education, and income were generally positively associated with obesity for both genders. One difference of note is that earning $50,000 a year or more is significant and positively associated with obesity risk for men (OR = 1.14, \(p < .001\)), but not for women. Being married was significant and negatively associated with obesity for women but positively associated with obesity for men. In addition, being black is significantly associated with higher obesity for women and men, although the odds are higher for women compared with men. Identifying as Hispanic was a positive predictor for obesity risk among women (OR = 1.14, \(p < .001\)) but was a nonsignificant predictor for men. All other racial/ethnic variables besides “other race” were positive and significantly correlated with obesity odds for both sexes. In gender-stratified models for morbid obesity, nonsignificant associations for key pollution exposure predictors were found (results not shown), and those models are available on request.

### Discussion and Conclusions

The purpose of this research was to ascertain the effect MSA-level EDC emission estimates have on individual-level risk for obesity and morbid obesity while controlling for socioeconomic factors, race and ethnicity, and health characteristics of the individual. Furthermore, emission source types were examined to determine if different causes of airborne emissions affect obesity risk differently. In these analyses, airborne EDC emissions were not associated with obesity, regardless of pollution source type. This finding rejects hypothesis 1, which predicts that higher exposure levels

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### Table 3. Adjusted Odds Ratios (95 Percent Confidence Intervals) of the Multilevel Logistic Model for Odds of Obesity (Body Mass Index \(\geq 30\) kg/m\(^2\)).

| Variable                                  | Non-point-source Estimated Airborne EDC Concentration | Point-source Estimated Airborne EDC Concentration |
|-------------------------------------------|-------------------------------------------------------|--------------------------------------------------|
| **MSA-level variables**                   |                                                        |                                                  |
| EDC exposure                              | 0.999 (0.972–1.026)                                    | 0.998 (0.967–1.031)                               |
| **Individual-level variables**            |                                                        |                                                  |
| Age                                       | 1.106*** (1.101–1.110)                                 | 1.106*** (1.101–1.110)                            |
| Age\(^2\)                                  | 0.999*** (0.998–0.999)                                 | 0.999*** (0.98–0.999)                             |
| Married                                   | 0.987 (0.936–1.011)                                    | 0.985 (0.963–1.011)                               |
| Female                                     | 0.957*** (0.936–0.979)                                 | 0.956*** (0.936–0.979)                            |
| No high school degree                     | 1.779*** (1.703–1.858)                                 | 1.779*** (1.703–1.858)                            |
| High school degree                        | 1.534*** (1.490–1.580)                                 | 1.534*** (1.490–1.580)                            |
| Some college                              | 1.457*** (1.415–1.499)                                 | 1.457*** (1.415–1.499)                            |
| Annual household income ($\))             |                                                        |                                                  |
| <15,000                                    | 1.567*** (1.490–1.580)                                 | 1.567*** (1.493–1.644)                            |
| 15,001–25,000                             | 1.398*** (1.339–1.460)                                 | 1.398*** (1.339–1.460)                            |
| 25,001–35,000                             | 1.327*** (1.267–1.389)                                 | 1.327*** (1.267–1.389)                            |
| 35,001–50,000                             | 1.278*** (1.223–1.335)                                 | 1.278*** (1.223–1.335)                            |
| >50,001                                    | 1.066** (1.024–1.109)                                  | 1.066** (1.024–1.109)                             |
| Black                                     | 1.813*** (1.746–1.882)                                 | 1.813*** (1.746–1.882)                            |
| Hispanic                                  | 1.124*** (1.074–1.177)                                 | 1.124*** (1.074–1.177)                            |
| Asian                                     | 0.381*** (0.344–0.423)                                 | 0.382*** (0.344–0.423)                            |
| Native Hawaiian/Pacific Islander          | 1.569*** (1.313–1.874)                                 | 1.569*** (1.313–1.874)                            |
| American Indian/Alaskan Native            | 1.418*** (1.286–1.563)                                 | 1.417*** (1.286–1.563)                            |
| Multiple races                            | 1.379*** (1.282–1.483)                                 | 1.379*** (1.283–1.483)                            |
| Other race                                | 1.061 (0.924–1.218)                                   | 1.061 (0.924–1.218)                               |
| **n**                                     | 188,252                                                | 188,252                                          |

*Note: Endocrine-disrupting chemical (EDC) exposure variables used in multilevel analyses were subsequently standardized to a mean of zero and a standard deviation of one. MSA = metropolitan statistical area.*

**p < .01. ***p < .001.
should be associated with a greater risk for obesity on the basis of the obesogenic hypothesis. This result largely counters the limited number of studies examining the direct link between EDC exposure and obesity prevalence. In one such study, Vafeiadi et al. (2015) found a correlation between exposure to two EDCs in utero, hexachlorobenzene and dichlorodiphenyldichloroethane, and increased BMI in children at age 4 in a longitudinal cohort study conducted in Greece. Tang-Péronard et al. (2014) also found an association between elevated BMI in female children 7 years of age and prenatal exposure to PCBs and dichlorodiphenyldichloroethane; associations for male children were nonsignificant. Wang et al. (2012) found that urinary BPA concentrations were significantly associated with increasing BMI in school-aged children in China.

In one of the only such studies analyzing adults, Hatch et al. (2010) found positive correlations between phthalate exposure and obesity in both men and women. However, a portion of their analysis found that higher levels of a particular phthalate, mono-2-ethylhexyl, was associated with lower BMI in adolescent girls and women aged 20 to 59 years. The authors hypothesized that EDC exposure may reduce hormone levels in the body, which “could help explain the inverse relationship between mono-2-ethylhexyl and BMI” (p. 6). In other words, exposure to EDCs may alter hormonal and metabolic pathways in such a way that promotes weight loss instead of weight gain; a theory supported by these research findings.

My results also did not support hypothesis 2, as the associations between EDC exposure and morbid obesity were found to be nonsignificant. This finding suggests that if correlations between EDC exposure and excessive weight do exist, there may be a threshold effect. Additionally, not all studies examining the relationship between EDC exposure and obesity found significant effects (Buckley et al. 2016). Hypothesis 3 regarding gender differences was also not supported by these results, as non-point-source and point-source EDC emissions were not significant predictors of obesity risk. Although nonsignificant, the direction of gendered associations is surprising, as it too does not support the obesogenic hypothesis. Uncovering gender differences in obesity risk is consistent with the sociological

### Table 4. Adjusted Odds Ratios (95 Percent Confidence Intervals) of the Multilevel Logistic Model for Odds of Morbid Obesity (Body Mass Index $\geq 35$ kg/m$^2$).

| Variable                          | Non-point-source EDC Pollution Exposure Concentration | Point-source EDC Pollution Exposure Concentration |
|----------------------------------|-------------------------------------------------------|--------------------------------------------------|
| EDC exposure                     | 0.999 (0.967–1.033)                                   | 1.001 (0.963–1.040)                               |
| Demographic variables            |                                                       |                                                  |
| Age                              | 1.131*** (1.123–1.138)                                 | 1.131*** (1.123–1.138)                            |
| Age$^2$                          | 0.998*** (0.998–0.998)                                 | 0.998*** (0.998–0.998)                            |
| Married                          | 0.918*** (0.884–0.952)                                 | 0.918*** (0.884–0.952)                            |
| Female                           | 1.281*** (1.237–1.327)                                 | 1.288*** (1.237–1.326)                            |
| Socioeconomic variables          |                                                       |                                                  |
| No high school degree            | 1.852*** (1.737–1.975)                                 | 1.852*** (1.737–1.975)                            |
| High school degree               | 1.496*** (1.429–1.566)                                 | 1.496*** (1.429–1.566)                            |
| Some college                     | 1.488*** (1.423–1.557)                                 | 1.488*** (1.423–1.557)                            |
| Annual household income ($)      |                                                       |                                                  |
| <15,000                          | 1.817*** (1.693–1.950)                                 | 1.817*** (1.693–1.950)                            |
| 15,001–25,000                    | 1.479*** (1.385–1.581)                                 | 1.479*** (1.385–1.581)                            |
| 25,001–35,000                    | 1.339*** (1.248–1.437)                                 | 1.339*** (1.248–1.437)                            |
| 35,001–50,000                    | 1.211*** (1.131–1.296)                                 | 1.211*** (1.131–1.296)                            |
| > 50,000                         | 0.956*** (0.856–0.982)                                 | 0.921** (0.891–1.025)                             |
| Race and ethnic minority variables|                                                       |                                                  |
| Black                            | 1.775*** (1.686–1.869)                                 | 1.775*** (1.686–1.869)                            |
| Hispanic                         | 0.956 (0.891–1.025)                                   | 0.956 (0.891–1.025)                              |
| Asian                            | 0.284 (0.232–0.348)                                   | 0.284 (0.232–0.348)                              |
| Native Hawaiian/Pacific Islander | 1.823*** (1.439–2.309)                                 | 1.823*** (1.439–2.309)                            |
| American Indian/Alaskan Native   | 1.374*** (1.199–1.575)                                 | 1.374*** (1.199–1.575)                            |
| Multiple races                   | 1.446*** (1.306–1.602)                                 | 1.446*** (1.306–1.602)                            |
| Other race                       | 1.067 (0.865–1.316)                                   | 1.067 (0.865–1.316)                              |
| $n$                              | 188,252                                               | 188,252                                           |

Note: Endocrine-disrupting chemical (EDC) exposure variables used in multilevel analyses were subsequently standardized to a mean of zero and a standard deviation of one.

**$p < .01$. ***$p < .001$. 

Table 4.
literature on obesity (Warin et al. 2008; Zhang and Wang 2004) as well as the obesogenic research reviewed above (Hatch et al. 2010; Tang-Péronard et al. 2014). Gender differences may be a result of differential susceptibility to metabolic disruption. For example, multiple windows of enhanced susceptibility to endocrine disruptors have been identified for women throughout the life course. These windows include pregnancy, menopause, and old age (Newbold 2010; Schug et al. 2011). Thus, exposure to EDCs likely has different endocrine-disrupting effects on weight for men and women (Heindel et al. 2015). Gender differences in obesity risk associated with EDC exposure are not likely explained by differential pollution exposure between men and women, as environmental inequality research has found little to no evidence of sex-specific risks to airborne pollution exposures (Mohai, Pellow, and Roberts 2009).

Several limitations should be considered when interpreting these research results. First, this study involved a cross-sectional design and did not capture temporal effects. Thus, the effects of cumulative pollution exposures and subsequent latent outcomes cannot be surmised from this study. To ascertain causation versus association, longitudinal, life-span analyses assessing cumulative pollution exposure and obesity should be conducted in the future (Heindel et al. 2015; Janesick and Blumberg 2016; Morello-Frosch et al. 2011).

Second, independent-level variables, including the dependent variable (BMI), relied on self-reported data and were subject to response bias. EDC exposure concentration estimates may also be biased. NATA data that are not directly collected through monitoring systems are self-reported by individual industries, which could lead to biased estimations; some argue that estimations may be more conservative because industries are incentivized to underreport emissions to meet federal regulations (Apelberg, Buckley, and White 2005). The small estimates of the seven individual EDCs also did not allow chemical-specific analyses. In previous studies, statistical analyses have been chemical specific to find associations between obesity risk and certain EDCs to elucidate obesogenic pathways (reviewed in Janesick and Blumberg 2016).

Last, because of the lack of publicly available data, potential MSA-level confounders related to the built environment,

| Table 5. Adjusted Odds Ratios (95 Percent Confidence Intervals) of the Multilevel Logistic Model for Odds of Obesity (Body Mass Index \(> 30 \text{ kg/m}^2\)) for Women. |
|-----------------|-----------------|-----------------|
| Variable        | Non-point-source EDC Pollution Exposure Concentration | Point-source EDC Pollution Exposure Concentration |
| EDC exposure    | 0.999 (0.971–1.028) | 0.998 (0.965–1.032) |
| Demographic variables |                   |                  |
| Age             | 1.109*** (1.103–1.1028) | 1.109*** (1.103–1.114) |
| Age\(^2\)       | 0.999*** (0.998–0.999) | 0.999*** (0.998–0.999) |
| Married         | 0.921*** (0.883–0.942) | 0.921*** (0.883–0.942) |
| Socioeconomic variables |          |                  |
| No high school degree | 2.035*** (1.924–2.152) | 2.035*** (1.924–2.152) |
| High school degree   | 1.605*** (1.543–1.668) | 1.605*** (1.543–1.668) |
| Some college       | 1.511*** (1.454–1.569) | 1.511*** (1.454–1.569) |
| Annual household income ($) |         |                  |
| <15,000          | 1.675*** (1.580–1.775) | 1.675*** (1.580–1.775) |
| 15,001–25,000    | 1.500*** (1.422–1.582) | 1.500*** (1.422–1.582) |
| 25,001–35,000    | 1.446*** (1.365–1.531) | 1.446*** (1.365–1.531) |
| 35,001–50,000    | 1.352 (1.279–1.428) | 1.352 (1.279–1.428) |
| >50,000          | 0.967 (0.918–1.018) | 0.967 (0.918–1.018) |
| Race and ethnic minority variables |                |                  |
| Black            | 2.054*** (1.960–2.151) | 2.054*** (1.960–2.151) |
| Hispanic         | 1.148*** (1.082–1.217) | 1.148*** (1.082–1.217) |
| Asian            | 0.366*** (0.316–0.422) | 0.366*** (0.316–0.422) |
| Native Hawaiian/Pacific Islander | 1.341* (1.046–1.717) | 1.341* (1.046–1.717) |
| American Indian/Alaskan Native | 1.319*** (1.160–1.498) | 1.319*** (1.160–1.498) |
| Multiple races   | 1.400*** (1.273–1.540) | 1.400*** (1.273–1.540) |
| Other race       | 1.146 (0.949–1.381) | 1.146 (0.949–1.381) |

Note: Endocrine-disrupting chemical (EDC) exposure variables used in multilevel analyses were subsequently standardized to a mean of zero and a standard deviation of one.

\(* p < .05, \*** p < .001\).
Despite this study’s limitations, this research adds to prior obesogenic and sociological research in several significant ways. First, few studies have used large populations to examine the direct effects of air pollution on obesity prevalence (Li et al. 2015). And to my knowledge, no study has investigated the specific effects of airborne EDC exposure and obesity with a large sample size. The use of a large, comparative study design is particularly importance because obesogenic scholars have noted that pollution exposure “may be difficult to detect at the individual level due to human genomic variability creating a heterogeneous population requiring a . . . statistical approach (Heindel et al. 2015:4). Commensurate with traditional sociological methods, the results of this nationally representative study are more generalizable than many obesogenic studies, which have relied on small cohort studies.

Second, by separately analyzing non-point-source and point-source emission types, we can potentially identify which environmental hazards affect human health most. In this study, the directional relationship between obesity risk and pollution exposure was not the same for non-point-source and point-source emissions. Point-source emissions were positively associated with obesity risk in certain models, while non-point-source emissions were negatively associated with obesity risk in all models. This signifies that pollution discharge from stationary sources, like industrial facilities, might initially be more worthwhile targets for emission reduction, public health, and environmental justice policies.

In conclusion, in this exploratory study I examined the association between MSA-level EDC emission estimates and individual-level risk for obesity and morbid obesity while accounting for socioeconomic factors, race and ethnicity, and health characteristics of the individual. The findings suggest that non-point-source exposure to EDCs reduces the risk for obesity. Gender differences may drive this finding; although nonsignificant, women were found to have reduced obesity risk with higher EDC exposure concentrations, while this effect was not found in men. The results do

| Variable                          | Non-point-source EDC Pollution Exposure Concentration | Point-source EDC Pollution Exposure Concentration |
|-----------------------------------|-----------------------------------------------------|---------------------------------------------------|
| EDC exposure                      | 0.989 (0.959–1.021)                                 | 1.001 (0.966–1.036)                               |
| Demographic variables             |                                                     |                                                   |
| Age                               | 1.104*** (1.096–1.111)                              | 1.104*** (1.096–1.111)                            |
| Age²                              | 0.999*** (0.998–0.999)                              | 0.999*** (0.998–0.999)                            |
| Married                           | 1.167*** (1.123–1.212)                              | 1.167*** (1.123–1.212)                            |
| Socioeconomic variables           |                                                     |                                                   |
| No high school degree             | 1.522*** (1.418–1.632)                              | 1.521*** (1.418–1.632)                            |
| High school degree                | 1.485*** (1.419–1.553)                              | 1.484*** (1.419–1.553)                            |
| Some college                      | 1.421*** (1.359–1.485)                              | 1.420*** (1.359–1.485)                            |
| Annual household income ($)       |                                                     |                                                   |
| <15,000                           | 1.274*** (1.166–1.391)                              | 1.274*** (1.167–1.391)                            |
| 15,001–25,000                     | 1.213*** (1.125–1.308)                              | 1.213*** (1.125–1.308)                            |
| 25,001–35,000                     | 1.163*** (1.077–1.256)                              | 1.163*** (1.077–1.256)                            |
| 35,001–50,000                     | 1.182*** (1.100–1.270)                              | 1.182*** (1.100–1.270)                            |
| >50,000                           | 1.148*** (1.076–1.224)                              | 1.148*** (1.076–1.224)                            |
| Race and ethnic minority variables|                                                     |                                                   |
| Black                             | 1.378*** (1.292–1.470)                              | 1.376*** (1.292–1.468)                            |
| Hispanic                          | 1.060 (0.985–1.140)                                 | 1.060 (0.985–1.140)                               |
| Asian                             | 0.391*** (0.336–0.453)                              | 0.391*** (0.336–0.453)                            |
| Native Hawaiian/Pacific Islander  | 1.863*** (1.439–2.409)                              | 1.864*** (1.440–2.411)                            |
| American Indian/Alaskan Native    | 1.578*** (1.356–1.835)                              | 1.578*** (1.357–1.836)                            |
| Multiple races                    | 1.354*** (1.210–1.514)                              | 1.354*** (1.210–1.515)                            |
| Other race                        | 0.984 (0.800–1.207)                                 | 0.983 (0.800–1.206)                               |
| n                                 | 75,197                                              | 75,197                                            |

Note: Endocrine-disrupting chemical (EDC) exposure variables used in multilevel analyses were subsequently standardized to a mean of zero and a standard deviation of one.

***p < .001.
not support predictions from the obesogenic hypothesis (Grun and Blumberg 2009). This study implies that some obesogenic pathways may contribute to weight loss instead of weight gain. Because the field of obesogenics is new and research so limited, solid conclusions and patterns have yet to be drawn, and future longitudinal research examining cumulative exposure and obesity prevalence is needed.

Finally, though my findings suggest that EDC exposure is associated with lower risk for obesity, the theoretical and methodological frameworks used in this study could be used by social scientists to further ascertain the social conditions under which weight gain and weight loss are exacerbated by EDC exposure.

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