ENVIRONMENTAL INEQUALITY AND OBESOGENICS: UNDERSTANDING THE RELATIONSHIP BETWEEN UNEQUAL EXPOSURE TO ENDOCRINE DISRUPTING CHEMICALS AND OBESITY PREVALENCE

by

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ABSTRACT

This dissertation investigates the association between exposures to environmental hazards and obesity prevalence. Building on obesogenic and environmental inequality research, this project explores the way in which exposure to a specific class of obesogens, endocrine disruptors, influences obesity risk. This study offers three substantial contributions to the current literature on environmental exposures and obesity by (1) investigating the effects of endocrine disrupting chemical exposure on obesity prevalence using populations-based estimates that are more generalizable then many previous studies, (2) assessing the environmental exposure-obesity association in highly susceptible populations and, (3) identifying social risks associated with increased exposure to endocrine disruptors. The results indicate that the influence of endocrine disruptor exposure on obesity is complex. Exposure type, population of study, and exposure measurements shape obesogenic findings. This study also found mixed results when assessing racial/ethnic and socioeconomic disparities in environmental exposures. Scholars can build off this work to better understand the socio-environmental mechanisms that place certain populations at a greater risk of hazardous exposure and how such exposure is related to health outcomes like obesity.

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Environmental Inequality

Socioeconomic Disparities in the Distribution on Environmental Hazards

The relationship between socioeconomic status and exposure to environmental hazards has been a central focus of environmental justice and inequality research. A strong pattern has emerged from early work on SES and exposure, with the majority of research suggesting that individuals with low SES experience greater exposure rates to environmental risks (Mohai and Bryant 1992). The link between low SES and increased exposure rates has been theoretically explained as such: impoverished individuals have fewer financial resources to mobilize and move away from vulnerable and toxic environments (Bullard 1983). Additionally, poor groups are more likely to be disenfranchised and have limited political power to prevent harmful industries from settling in their neighborhoods. Thus, areas categorized with low SES are more vulnerable on the individual *and* the structural level. In an essence, these low income communities are 'paths of least resistance' that are subjugated to endure more environmental dangers than high SES communities (Morello-Frosh and Lopez 2006).

In the past decades, some scholars have challenged this predominate view of the relationship between SES and environmental exposure. In a follow up to a 1994 critique, Davidson and Anderton (2000) found no evidence for the claim that low SES individuals shoulder the burden on environmental hazards. This echo's their pervious findings of "…almost no support for the general claim of environmental inequality" (Anderton 1994: 243). Bowen (2002) also suggests that the EJ literature has been plagued with methodological incongruences, which make it impossible to claim that SES is associated with environmental risks.

These critiques of the relationship between SES and environmental quality are anomalies compared to recent research on this subject matter. Contemporary studies focusing on environmental inequalities support the relationship between low SES and increased hazardous exposure. Beginning with work from the start of this century, Pastor and colleagues (2001) found that industrial waste facilities were disproportionately located in low-income areas. In their national level study, Evans and Kantrowitz's (2002:323) findings suggest that, "…the poor and especially the non-white poor bear a disproportionate burden of exposure to suboptimal, unhealthy environmental conditions in the United States".

In a more nuanced study, Mohai et al (2009) found that income and education were significant predictors of respondents' proximity to polluting facilities. Income was also a significant predictor in Crowder and Downey's (2010) research on environmental inequality and migration patterns. The authors found that, at the neighborhood level, high household income was a large determinate of moving out of environmental vulnerable communities. In another neighborhood level study, Pais, Crowder, and Downey (2014) found that socioeconomic resources influence trajectories of exposure to pollution because SES predicts whether individuals can move from their neighborhood or not. Finally, in their nationwide study, Zwickl, Ash, and Boyce (2014) found that residents of lower income neighborhoods always experienced higher levels of exposure than residents from upper-income neighborhoods, even when controlling for race.

This collection of new EI research confirms previous findings that socioeconomic status predicts differential exposure to environmental harm. This research supports popular theories which suggest that elements of SES, such as income, wealth and level of education, are important resources that buffer contact with environmental risks. On an individual level, SES resources give people more options and agency when deciding where to ‘live, work and play’ (Schlosberg 2013). Higher SES individuals are able to have more of a say in the physical environments they experience, reducing vulnerability. On the structural level, higher SES areas have more political power and can offer resistance to polluting institutions in their environments (Pais et al. 2014).

EI scholars have acknowledged the power SES has in predicating patters of environmental inequality. Concomitantly, when accounting for the historical process that produce inequality, SES is often highly correlated with race in the United States. Race has traditionally been a strong predictor of environmental inequality in addition to SES. In the next section, a review of racial disparities in the distribution on environmental risk is provided.

Racial Disparities in the Distribution on Environmental Hazards

Racial disparities in the distribution of environmental risks have always been a central tenant of the environmental inequality research. EI research can be traced back to early studies on environmental racism, which is the notion that racial minorities are disproportionally affected by environmental hazards. Foundational pieces suggesting that minorities experience higher exposure rates set the stage for current, more nuanced research on environmental racism (Bullard 1983, 1990; Moahi and Bryant 1992; Pulido 1996).

Expanding environmental racism and inequality research, Downey (2006) found that black neighborhoods in Detroit were disproportionally burdened by facility emission activity. Additionally, his findings suggest that neighborhood racial composition was a strong indicator of proximity to hazardous facilities. In a similar study on neighborhoods, Pais et al. (2014) found that racial disparities in cumulative exposure to environmental hazards persisted after controlling for SES. On a more macro level, Downey (2008) found that, in general, black and Hispanic populations experience higher pollution exposure than other races. Mohai et al. (2009) additionally found that racial disparities were especially pronounced in metropolitan areas of the Midwest and West and in suburban areas of the South. Taken together, these studies highlight the contextual relationship between race and rates of exposure.

Understanding the context and conditions under which environmental racism occurs has been a welcomed evolution in the EI literature. Recent studies are finding that the extent of environmental racism varies for different minority groups. For example, Hooks and Smith (2004) demonstrate that American Indians disproportionally live near nuclear waste sites because waste sites are often located near or on tribal reservations. In a multi-level analysis, Crowder and Downey (2010) find that racial disparities in exposure to industrial hazards vary by race. For example, Asians are the least likely to live in polluting neighborhoods, blacks are most likely to originate in polluting neighborhoods, and Hispanics are more likely to move to polluting neighborhoods. These various degrees of environmental racism suggest that "factors related to race, such as racial targeting or housing discrimination" play a role in differential exposure to risks among minorities (Crowder and Downey 2010:1138).

The structural and historical elements that contribute to environmental racism have been the theoretical underpinnings scholars use to explain the relationship between race and environmental exposure. The 'racial residency' theory suggests that historical patterns of segregation inherently make communities of color more vulnerable to industrial environmental ills (Pais et al. 2014). Similar to SES, it is argued that minority neighborhoods are marginalized areas of 'least resistance' that can be exploited by industries with little repercussion (Downey 2008). Most research, including the aforementioned studies, supports this structural explanation of environmental racism.

Another prevalent theory used to explain environmental racism is the 'racial-income' thesis (Crowder and Downey 2010). This paradigm suggests that patterns of racial environmental inequality can be explained by the differences in income between various races. There are large disparities in income between races and, arguably, it is this disparity that makes minority communities more susceptible to exposure. Research supporting this hypothesis is mixed. Current studies, like Pais et al. (2014), find that racial disparities in exposure rates persist even when controlling for SES. Downey (2008) suggests that the role income inequality plays on environmental burden varies by race. For some minority groups, income may explain racial inequalities, but for other groups, structural factors besides SES may be more influential (Downey 2005, 2010).

On par with this claim, Zwickl et al (2014) find a complex picture when examining within-group and between-group disparities across metro areas in the U.S. For example, they find that Hispanics tend to live in less polluted cities than whites, but within any given city, Hispanics are concentrated in areas that experience higher air pollution exposure.

Finally, some scholars adhere to an 'agnostic' paradigm and suggest that neither income nor race is important. Anderton and colleagues (1994, 2000) adopt an extreme view and suggest that community characteristics such as type of commerce, size and demographic composition are key predictors of environmental inequality. Most scholars, however, offer a more moderate view and espouse that it is not race or SES alone that accounts for inequality, it's the combination of them in addition to other factors. The 'race vs. class' debate predominated early EJ work, but many contemporary scholars have outgrown the debate because when focusing on which is more important, race or SES, it's easy to miss how these variables interact. As recent research illustrates, "in some instances, race shapes [exposure] outcomes in concert with class" (Grant, Trautner, Downey, and Thiebaud 2010: 498).

In summary, scholars are just now beginning to understand the complex processes and interactions that produce environmental inequality. Our newfound knowledge of such processes is spurred by research that recognizes the importance of examining the causal pathways that lead to increased exposure of environmental risks and the health consequences of exposure. In the last decade, there has been a push to understand how differential exposure to environmental hazards helps explain health disparities and outcomes. This new field, known as environmental health inequality, seeks to understand the relationship between environmental quality, environmental exposure and human health. The next section outlines environmental health inequality research and the substantive findings that have emerged from this field.

Environmental Health Inequality

Defining Environmental Health Inequality

Originally situated in the policy arena, early interest in sociodemographic inequalities in environmental exposure and resulting diseases emerged from global health institutions like the World Health Organization and the United Nations (WHO 2010). In efforts to uncover the mechanisms through which social factors influence inequalities in environmental exposure and health disparities, social and health scientists have recently come together under the banner of ‘environment health inequality’ (EHI) research. What makes EHI distinct from related fields like environmental inequality and health disparities research, is that EHI scholars place equal importance on *both* environmental and health indicators. Take racial disparities in asthma rates as an example. Environmental inequality scholars may focus on how housing segregation produces high concentrations of minorities in inner city areas where air pollution exposure is higher; documenting environmental exposure is their primary concern. On the flip side, health disparity researchers tend to focus on how asthma manifests in the body and how different groups access treatment for asthma; documenting health-related outcomes is their primary concern. Bridging these two concentrations together, EHI scholars examine the socioeconomic processes that lead to unequal environmental risk exposure and how that differential exposure explains disparate health outcomes. In other words, EHI researchers recognize that “it is not just the difference in exposure that matters, but the fact that these differences contribute to health inequalities” (Kruize et al. 2014:5809).

Environmental Health Inequality: Conceptual Frameworks

Blending social science and health research together to measure environmental health inequalities has been conceptually and theoretically successful, but empirically challenging. Many scholars have put forth conceptual models that integrate sociodemographic, environmental and health components at multiple levels. In this section, I will discuss influential frameworks used in EHI research, and I refer you to Kruize and colleagues (2014) and Linder and Sexton (2011) for an exhaustive review of additional conceptual frameworks that have emerged from this field.

Evans and Kantrowitz (2002) were perhaps the first scholars to outline how socioeconomic status, environmental quality and health outcomes could be linked. The authors argue that SES resources play a key role in determining an individual’s exposure to natural and built environmental risks like air and water pollution, ambient noise and housing quality. They further outline how differential exposure to poor quality environments can negatively affect health and wellbeing. In their model, environmental quality mediates the socioeconomic and health gradient. In 2010, Evans and Kim expanded their original model by examining how exposure to multiple environmental risks could account in part for the SES health gradient. Their change in focus from one environmental risk (e.g. water pollution) to multiple environmental risks reflects the need to account for multiple environmental mechanisms that may affect health.

Cumulative risk assessment models have been adapted by EHI researchers to conceptually outline "the combined harmful effects from exposure to a mixture of environmental stressors" (Sexton, Stephen, and Linder 2011:S81). Cumulative risk assessments (CRA) allow scholars to account for chemical stressors in the environment as well as social psychological stressors when assessing health outcomes (Linder and Sexton 2011). Because CRA encompasses social, psychological and environmental phenomena, it is a popular framework for EHI scholars. Gee and Payne-Sturges (2004) use a stress-exposure-disease framework to model how race, the environment and health relate. The authors argue that psychosocial stress from institutional discrimination, namely segregation, can help explain the differences we see in health outcomes between whites and minorities. For example, because minorities tend to live in areas with higher rates of exposure and lower SES, community and neighborhood stressors may cause personal stress that makes individuals more susceptible to illness. This model therefore accounts for multiple stressors (segregation, poverty, increased environmental exposure, individual stress) that potentially mediate the relationship between exposure and health inequality.

In a similar, more nuanced framework, Morello-Frosch and Shenassa (2006) incorporate the biophysical elements of health by adding allostatic load and psychosocial stressors into a cumulative risk assessment model. Their theoretical framework posits that chemical and non-chemical factors contribute to individual stress and allostatic load; allostatic load refers to the 'wear and tear' of the body that occurs from living with chronic, accumulated stress (Morello-Frosch, Zuk, Jerrett, Shamasunder, and Kyle 2011). Allostatic load, in turn, increases individual vulnerability to disease, thereby contributing to subsequent health disparities. This exposure-stress-effect model elaborates on 'riskscape' theories to show how sociopolitical discrimination can lead to increased exposure and how a body's defense system weakens with contact to environmental contaminants (Morello-Frosch 2002; Morello-Frosch and Lopez 2006). Collectively, this model accounts for structural, environmental, community and individual stressors that accumulate overtime to produce environmental and health inequalities.

The aforementioned conceptual models have theoretically advanced the field of environmental health inequality. To date, however, there is no scientific consensus on which approach best explains the pathways linking unequal distributions of environmental burdens to racial and socioeconomic health disparities (Linder and Sexton 2010).

Empirical Findings from Environmental Health Inequality Research

Due to the methodological issues discussed above, the number of studies analyzing how environmental inequality mediates socioeconomic and racial disparities in health is relatively small. The following review summarizes studies that explicitly examine how environmental exposure risks vary by race or income and, in turn, how those risks are linked to specific health outcomes and disparities.

Jerret, Burnett, Brook, Kanaroglou, Giovis, Finkelstein, and Hutchison (2004) were some of the first scholars to examine how SES modifies the relationship between air pollution exposure and mortality. The authors found that city neighborhoods with low SES were associated with higher levels of air pollution and increased mortality rates. More importantly, they found that low educational attainment and high manufacturing employment significantly modified the mortality effects of air pollution exposure in

Hamilton, Canada. In their case study review, Schulz and Northridge (2004) also find evidence that lower levels of education are associated with increased health and mortality risks from particulate matter. Zeka, Zanobetti, and Schwartz (2006) found an inverse relationship between level of education and mortality risks associated with exposure to particulate air matter in Boston.

Despite these related findings, Laurent, Bard, Filleul, and Segala's (2007) multiscalar review on how SES modifies short-term and long-term effects of air pollution exposure on mortality risk yielded mixed results. At larger units of analysis (e.g. cities, the authors found no SES modification effect of exposure on mortality. However, at finer geographic units (e.g. neighborhoods), SES did help explain exposure to air pollution and subsequent mortality outcomes. Such results lead the authors to the conclude that, "evidence does not yet justify a definitive conclusion that socioeconomic characteristics modify the effects of air pollution on mortality" (Laurent et al. 2007: 665).

Moving beyond mortality indicators, Apelberg, Buckley, and White (2005) examined how estimated cancer risks varied by exposure to air pollution and socioeconomic and racial characteristics in Maine. They found that air pollution exposure and subsequent cancer risks were higher in census tracts with low SES and high minority concentrations. Racially, blacks and Hispanics had the highest potential risk of pollution related cancers but, income remained the largest predicator of risk. In a similar study conducted by Pastor, Morello-Frosch, and Sadd (2005), the authors found a pattern of unequal exposure by race and income to cancer causing air toxins in California. Cancer risks were higher for racially and economically disadvantaged groups because they live in "riskscapes" where they are disproportionately exposed to cancer causing environmental toxins. This pattern between SES, race, exposure and cancer risks has been documented throughout the U.S. in Huston (Linder, Marko, and Sexton 2008), Tampa (Chakraborty (2012) and cities across Texas (Prochaska, Nolen, Kelly, Sexton, Linder, and Sullivan 2014).

Unequal exposure to air pollution concentrations has also been linked to low birth weights. Brink, Benson, Marshall, and Talbott (2014) conducted a study of Allegheny County, PA to determine if socioeconomic disparities in pollution exposure could partly explain differences in birth weights and preterm births. The author's found that pollution concentrations were in fact higher in lower income census tracts. Moreover, within the lowest income and highest exposed tracts, the odds of low birth weights and preterm births increased by 14 percent and 16 percent, respectively, when compared to the highest income tract. These findings suggest that poor birth outcomes may partially be attributed to higher pollution levels in low-income neighborhoods. This study further supports Clougherty, Shmool, and Kubzansky's (2014) review findings that socioeconomic position increases susceptibility to environmental pollution.

Susceptibility was also a factor in another study looking at how racial and socioeconomic disadvantage modifies the relationship between blood lead and blood pressure (Hicken, Gee, Mornoff, Connell, Snow, and Hu 2012). The authors found that social stressors related to race and low SES enhance vulnerability to hypertension related to lead exposure. Hicken and colleagues conclude that social factors increase vulnerability to health effects of environmental hazards.

One such social factor, which has largely been ignored in environmental health and environmental inequality research, is gender and sex. Gender and sex differentials in environmental quality has historically be discussed as an interesting afterthought, but contemporary scholars are giving gender and sex primacy in their analyses of environmental health issues. Clougherty (2010) outlines the importance of a gendered framework in understanding how gender shapes the types of environments people spend their time in. Women for example, generally perform more cooking, which can increase exposure to indoor fossil fuel pollution (Bell and Ebisu 2012), and use potentially toxic personal care and cleaning products more frequently than men (Meding 2000). Men often engage in work that is classified by higher amounts of exposure to fossil fuel (e.g. mechanics), air particulate matter (e.g. outside road construction workers), and heavy metals (e.g. mining operators; Messing et al. 2003). Gender norms and expectations shape the types of exposures individuals experience at home, in occupational settings, and recreationally and each of these diverse environments pose unique health risks associated with toxic exposures; health risks that can manifest differently for men and women. A gendered analysis is therefore essentially to understanding inequalities in environmental exposures and how those exposures effect human health.

The aforementioned studies conducted by Clougherty (2010), Hicken et al. (2012) and Brink et al. (2014) represent a desired trend in expanding the health and environmental outcomes EHI scholars utilize, particularly gender. In recent years, research on one particular health outcome, obesity, has surged, as scholars believe there is link between harmful environmental exposures and obesity prevalence. This budding field is known as obesogenics.

The Obesogen 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 2007). In a landmark paper, Baillie-Hamilton (2002) argued that exposure to chemical toxins is correlated to rising obesity rates by showing that the global obesity epidemic coincides with significant increases of industrial chemicals in the environment over the last 40 years. Expanding on this work, Grun and Blumberg (2007) proposed the "environmental obesogens" hypothesis, which espouses that environmental pollutants can disrupt and interfere with the body's metabolic, energy balancing system and fat storage. Specifically, exposure to obesogens "promotes adiposity by altering programming of fat cell development [and] increasing energy storage in fat tissue" (Janesick and Blumberg 2016:1). 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 adipogensis, 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, adipogensis 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 and mis-regulate the critical pathways involved in adipogensis. In their review, Grun and Blumberg (2007) highlight research that demonstrates how chemicals, such as bisphenol-A (BPA), inhibit nuclear hormone receptor signaling pathways during adipogensis; which can cause in *increase* in fat cell production and, subsequently, obesity. In another review, Janesick and Blumberg (2016:8) conclude "that increased adipogenesis during early development permanently establishes an elevated fat cell number in adulthood". If this pathway exists, whereby obesogenic exposure early in the life course permanently increases the fat cell number within an individual, it's 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 suggest 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 the number of fat cells is largely independent of body mass index (BMI). This supports prior research which suggests that obesity mechanisms are complex and that by in large, obesity is a product of both increased adipose cell number *and* increased cell size (Salanes, Cushman, and Weismann 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 a higher number 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).

Obesogens are particularly dangerous on a public health level because of their ubiquity and research has demonstrated that the obesogenic environment consists of a broad range of environmental factors. For example, dietary foods, such as monosodium glutamate and fructose, are known obesogens and certain pharmaceuticals, like synthetic estrogens estradiol and diethylstilbestrol, have been found to effect metabolism and body weight processes adversely (Newbold 2007). Built environment obesogens have also been identified and include lead, formerly used in house paint (Janesick and Blumberg 2011), and fine particulate matter generated by car exhaust (World Health Organization 2013).

The obesogenic environment also includes non-chemical elements. The majority of obesity based researched has focused on delineating the social and physical factors that can influence an individual's body weight. Scholars have found strong associations between social conditions, like marital status (Sobal, Rauschenbach, and Frongillo 2003), low socioeconomic status (Flegal et al. 2012; Mokdad et al. 2003), race and ethnicity (Ogden et al. 20140, sex (Wang and Beydoun 2007), age (Baskin et al. 2005), and mental health status (Sajjadi and Nakhodai 2016), and obesity risk. Beyond these individual-level risk factors, physical built environments have also been shown to affect body weight. Neighborhood characteristics related to walkability (Brown et al. 2013), the presence of green space and parks (Bancroft et al. 2015: Wei et al. 2016), degree of urbanization (Wang, Wen, and Xu 2013), perceived crime and safety (Forster and Files-Corti 2008; Kooshari et al. 2015), and levels of segregation (Corral et al. 2015) have been linked to obesity outcomes. Taken together, the obesity literature paints a broad, multi-dimensional picture of how obesity is caused by numerous factors that operate on the individual-level (e.g. lifestyle), the meso-level (e.g. neighborhood effects) and the macro-level (socioeconomic status; Arcaya et al. 2016). Because many of these contextual elements have been extensively studied within the obesity literature, this project examines an obesogenic mechanism that is not well documented: chemical obesogens. This study fills gaps in obesity and obesogenic research by examining chemical obesogens that have been linked to the disruption of the endocrine system and metabolic processes.

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 and Janesick and Blumberg 2016). As obesogens, EDCs are thought to increase the risk of obesity in individuals by the aforementioned pathways, by increasing the number of fat cells and/or the storage of fat in existing cells (Holtcamp 2012). Additionally, EDCs have been shown to modify metabolic rate (Heindel 2011), 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 toxicology findings establishing a causal link between chemical exposure and metabolic disruption have involved endocrine disruptors (Navas-Acien et al. 2008). Endocrine disrupting chemicals are ubiquitous in both natural and built environments and common EDCs include: industrial chemicals like BPA and polychlorinated biphenyl ethers (PCBs); herbicides such as atrazine; pervasive environmental pollutants including particulate matter; and naturally occurring heavy metals such as mercury and lead (Ahearn 2012; Thaddeus 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 last few decades, exposure to EDCs has become increasingly widespread (Diamanti-Kandarakis et al. 2009).

Concern about exposure to endocrine disrupting chemicals is also becoming more widespread as evidence connecting EDCs to health issues related to fertility, cancer, metabolism, and obesity mounts. Within the last 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 biphenyl-A (BPA) in plastic water bottles. As a plasticizing 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 like baby bottles, water bottles, plastic cutlery, and plastic toys (CDC 2012). As such, we urgently need more research examining how these pollutants effect health risks and outcomes.

The Sociology of Obesogenes

Although considerable progress has been made in understanding the role toxic chemical exposure plays in obesity, obesogenic research has two glaring problems. First, obesogenic research has largely been conducted 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 (SES) and 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. Throughout these chapters, I seek to approach obesogenics from a sociological perspective, embedding findings within environmental inequality and health disparity frameworks.

Study Objectives and Organization of the Dissertation

The overarching aim of this research is to understand how social conditions produce greater risk of hazardous exposure among certain populations and how this exposure is related to obesity prevalence. Specifically, I have three main objectives. *The first objective is to assess the influence socioeconomic and racial/ethnic factors have on exposure to endocrine disruptors*. Poor and minority populations often face increased exposure to various forms of pollution (Downey and Hawkins 2008) and it is increasingly important to uncover exact pollution sources and chemical-specific pollution. If research can find associations between pollution source type (e.g. automobile exhaust, factory smoke stack) and specific chemicals (e.g. BPA), public policy can be more direct and efficient in reducing environmental hazards that threaten public health. Chapter 2 uses chemical-specific emissions data for various pollution source types to examine socioeconomic and racial disparities in exposure to endocrine disrupting chemicals. Using data from the Environmental Protection Agency's 2005 National Air Toxics Assessment (NATA) and the 2010 U.S. Census, this cross-sectional county-level study examines if measures of social class and racial/ethnic composition predict EDC exposure.

The second objective is to examine the associations between obesity risk and exposure to endocrine disruptors. Chapter 3 of this dissertation employs a cross-sectional design to assess airborne endocrine disrupting chemical exposure and obesity prevalence across metropolitan statistical areas in the United States. Linking emissions data from NATA and health data from The Behavioral Risk Factor Surveillance System (BRFSS), I was able to conduct a population-based analysis for the year 2005. The large, nationally representative nature of this data are important in establishing generalizable trends in environmental exposure-obesity associations.

The third objective is to assesses obesogenic associations in highly susceptible populations. The exposure-obesity relationship seems to be complex, which may help explain why evidence regarding this association varies. Identifying windows of exposure susceptibility throughout the life course may yield more robust correlations and bolster our ability to make stronger conclusions about the role obesogens play in the etiology of obesity (Schug et al. 2011). In Chapter 4, I analyze a population known to have heightened susceptibility to exposure: pregnant women. Using data from the Utah Population Database, I compare gestational weigh gain outcomes among women with different occupational exposure probabilities to endocrine disruptors. In this retrospective study, exposure probability measures were assigned based on a job-exposure matrix (Brouwers et al. 2009), which allowed me to also examine racial and ethnic differences in occupational exposures in subsequent analyses.

Finally, Chapter 5 summarizes key research findings and addresses the limitations and significance of this research. Potential directions for future research on obesogenics and environmental inequality are also discussed.

CHAPTER 2

ECONOMIC DEPRIVATION, ETHNIC COMPOSITION, IMMIGRANT ISOLATION AND ENVIORNMENTAL INEQUALITY: ASSESSING COUNTY-LEVEL EXPOSURE TO AIRBORNE ENDOCRINE DISRUPTORS IN THE UNITED STATES

Introduction

Theories of environmental inequality and environmental justice suggest that some populations experience environmental hazards and risks more than others. The U.S. Environmental Protection Agency (EPA) refers to environmental justice as the "fair treatment and meaningful involvement of all people regardless of race, color, national origin, or income with respect to the development, implementation, and enforcement of environmental laws, regulations, and policies" (EPA 2010). This administrative declaration stems from decades of research documenting the disparities in environmental quality between groups with different socioeconomic and racial/ethnic status. Early environmental justice (EJ) studies focused on proximity to potentially harmful locations like waste sites and industrial polluters (e.g. incinerators) (Bullard 1990; U.S. General Accounting Office 1983). In addition to contemporary research on proximity (Bullard et al. 2008; Chakraborty et al. 2011; Mohai and Saha 2007; Pastor et al. 2004), many other forms of environmental injustices have been studied.

Air pollution exposure is a commonly analyzed indictor of environmental inequality and strong associations have been drawn between increased exposure and low socioeconomic status (SES) and racial/ethnic composition. For example, Bell and Ebisu (2012) found higher exposure rates of air particulate matter (PM 2.5) among non-white Hispanics, blacks, and individuals with low SES when compared to whites and those with higher SES. The findings of Jones and colleagues (2014) suggest that living in a majority white neighborhood was associated with lower air pollution exposure, while living in majority non-white Hispanic neighborhood was correlated to increased pollution exposure. In a longitudinal study, Pais et al. (2014) documented racial disparities in cumulative exposure to airborne toxins, even when controlling for socioeconomic status. Finally, Ard (2015) found that although exposure to industrial air toxins has decreased throughout the last decade, the disparity in exposure between whites and minority groups has not decreased. She concludes that, “the differential exposures between these groups have remained relatively consistent across time” (387).

Studies such as these illustrate that minority status and socioeconomic positon effect environmental exposure risk (Zwickl, Ash, and Boyce 2014). This new body of EJ research is moving away from the ‘race-versus-class’ debate, which dominated early environmental justice theory (Bullard 2005; Downey 1998; Massey and Denton 1993), to focus on uncovering the conditions in which race shapes exposure outcomes in concert with class (Grant, Trautner, and Downey 2010). Historical factors and residential choice have been incorporated in analyses to determine specific social underpinnings of environmental inequality. For example, it has been theorized that historical discrimination processes constrain the choices minority and low-SES individuals have when moving to or from areas with poor environmental quality (Mohai, Pellow, and Roberts 2009). To test this theory, scholars have assessed how neighborhood-level racial composition (Downey and Hawkins 2008), migration status (Crowder and Downey 2010), segregation levels (Ard 2015; Crowder and Krysan 2016), immigrant isolation (Bravo et al.; Lievanos 2015), and family structure (Downey, Crowder, and Kemp 2016) effect the risk of environmental exposure. Taken together, results suggest that social and cultural conditions can put some groups in more jeopardy of experiencing environment hazards.

Given the mountain of evidence documenting environmental injustices, scholars advocate for the integration of environmental inequality and its associated health impacts into sociological studies (Brulle and Pellow 2006). Research in this vein has found associations between increased pollution exposure and diabetes (Alnoso-Magdalena and Nadal 2011; Zoeller et al. 2012), autism (Talbott et al. 2015), and obesity (Holtcamp 2012; Hyman 2010; Janesick and Blumber 2016). A new field, known as obesogenics, has emerged studying the relationship between exposure to environmental toxins and obesity prevalence. Obesogenic research has singled out a subclass of toxins, endocrine disruptors, that have been associated with obesity in children and it is thought this correlation exists in adults as well (Agay-Shay et al. 2015; Vafeidali et al. 2015; Valvi et al. 2014). Identifying direct linkages between health outcomes, chemical-specific pollution, and increased social susceptibility to pollution exposure can help us better understand both health disparities and environmental inequality.

Building on this body of literature, this research examines the relationship between socioeconomic status, race and ethnicity and exposure to a particularly harmful type of environmental hazard, airborne endocrine disrupting pollution. Specifically, this project assesses the relationship between air pollution exposure and (1) economic deprivation, (2) immigrant concentration and, (3) ethnic concentration across counties in the United States. This population-level study uses pollution-source estimates for two types of pollution, non-point (e.g. mobile emissions from automobiles) and point (e.g. location specific emissions), which helps disentangle differential exposure effects by pollution type. In addition to examining various pollution forms, this research adds to the environmental justice literature by extending the few analyses that have incorporated sociocultural factors like immigrant isolation. Furthermore, by analyzing chemical-specific pollution emissions, these findings can inform environmental health and obesogenics research. I specifically hypothesize that:

H_1: more economically depraved counties will have higher annual emission concentrations to airborne endocrine disrupting chemicals (EDCS) for both point and non-point emission sources.

H_2: counties with higher concentrations of minority populations will have higher annual emission concentrations to airborne endocrine disrupting chemicals (EDCS) for both point and non-point emission sources.

Methods

Data

National Air Toxics Assessment

Air pollution emissions data were obtained from the United States Environmental Protection Agency's (EPA) 2005 "National Air Toxics Assessment" (NATA) online database (https://www.epa.gov/national-air-toxics-assessment/2005-nata-assessment-results#state, accessed December 2015). NATA emissions data were compiled from a variety of sources including: state and local air pollutant inventories, the EPA's Toxic Release Inventory (TRI) database, and emissions estimates from the EPA's Office of Transpiration and Air Quality. This compiled National Emissions Inventory (NEI) is 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 by the EPA 2011). In 2005, NATA estimated 177 of 187 air toxins listed under the 1990 Clean Air Act Amendment. Furthermore, NATA data is 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 smoke stack or sewage treatment plant. Non-point emission sources are mobile sources that include automobiles, wildfire smoke, and sediment kick-up from mining and construction areas (EPA 2012).

United States Census Bureau, 2010

County-level population characteristics were gathered from the 2010 United States Census (https://factfinder.census.gov/faces/nav/jsf/pages/index.xhtml, accessed November 2015). The decennial census gathers demographic information on every household across the U.S., including The District of Columbia (Census Bureau 2010). Although Census and NATA data were collected from different years, data can be joined without risking too much bias within the sample because population changes occur at a slow pace; meaning that population estimates from 2010 are likely very similar to those from 2005 (Wang, Wen, and Xue 2013).

Sample

National Air Toxics Assessment

Because prior obesogenic research has found strong associations between endocrine disrupting chemicals and obesity, estimated ambient concentration of air toxins was limited to seven know EDCs (Arner et al. 2010; Grun and Blumberg 2006; Janesick and Blumberg 2011; Vandenberg et al. 2012). These seven compounds include: five insecticides/fungicide (chlordane, hexachlorobenzene, hexachlorocyclohexane**,** methoxychlor and toxaphene), one polychlorinated organic compound (polychlorinated biphenyls (PCBs)), and one phthalate (bis(2-ethylhexyl)phthalate (DEHP)). Characteristics of each toxin are summarized in Table 3.1. County level annual ambient concentration estimates ($\mu g/m^3$) for the seven EDCs was collected 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, all seven concentration estimates were added together to produce more robust exposure variables.

United States Census Bureau, 2010

Census data was collected for 2,056. Counties with a population less than one-hundred people were excluded from analysis (n=43). Due to missing data for the dependent emissions exposure variable, counties in Alaska (n=19) and Wyoming (n=23) were omitted. This yields a sample of 1,971 county-level observations. Additionally, for ease of interpretation and to address non-normal distributions, census variables were standardized.

Measures

Dependent Variable

Airborne endocrine disrupting chemical pollution exposure was assessed by using annual ambient emission concentration estimates ($\mu g/m^3$) for point, non-point, and total (point plus non-point) emission sources. These total estimates are a summation of concentration estimates for the seven EDCs discussed above and are measured as continuous variables. Exposure measures were standardized to account for nonlinearity and heteroscedasticity (Long and Freese 2006). Analyzing non-point, point source and total pollution estimates separately can be helpful in assessing cumulative impacts of air pollution exposure (Morello-Frosh, Pastor, and Sadd 2001; Morello-Frosh et al. 2011) and potentially identify which environmental hazards pose the most health risks (Linder et al. 2008).

Key Independent Variables

The three key independent variables were economic deprivation, ethnic composition, and immigrant concentration. An index for *economic deprivation* was constructed using factor principal component analysis (Cronbach's α=0.72). The index was created from the following four variables: percent of the population 25 years or older without a high school degree, percent of the population 16 years or older unemployed, percent of female-headed households, and percent of the population living below federal poverty guidelines (U.S. Census Bureau 2010). Factor loadings for the four items ranged from 0.45 to 0.88; higher values of this variable indicated greater levels of economic deprivation. *Immigrant isolation* was assessed using two variables: percent of the population foreign born and percent of households linguistically isolated that speak foreign languages associated with their racial/ethnic group (e.g. Spanish, American Indian dialects). These two variables were also indexed using factor principle component analysis (Cronbach's α=0.68, factor loadings: 0.41-0.79). Component variables similar to the two employed in this study have been used before to simplify and operationalize complex concepts (Lievanos 2015; Trinh and Wen 2015). *Ethnic composition* was measured as the percentage of the population non-white.

Control Variables

Three covariates were included in this study and each was informed by previous research. The first is *population density*, measured as population per square kilometer. This measure has been positively associated with concentrated air pollution exposure and low-SES (Downey and Hawkins, 2008) and minority concentration and immigrant isolation (Lievanos 2015). *Percent of the population identifying as female* is included to control for disparities in poverty rates between women and men (Jerret et al. 2004). The final control variable measured *average travel time to work* in minutes per week for individuals who work away from home. This variable accounts for the positive association between worker-commuter rates and mobile emissions sources associated with vehicles (Lievanos 2015).

Statistical Analyses

Factor principal component analysis was conducted to create the economic deprivation scale and a series of OLS logistic regression analyses were used to test the hypotheses. Models 1 through 3 tested the main effect of economic deprivation on estimates of each pollution source type net of control variables. Models 4 through 6 added immigrant isolation and ethnic composition to Models 1 through 3. Analyses were conducted using Stata Software, version 13 (StataCrop. 2013. *Stata Statistical Software: release 13. College Station, TX: StataCorp L*P).

Results

Table 2.2 shows descriptive statistics for exposure concentrations and population characters at the county-level. The average estimated ambient concentrations of airborne endocrine disputing chemicals were 0.05, 0.29, and 0.35 micrograms per cubic meter of air for non-point, point and total source pollution types, respectively. Although the range of concentrations is quite large (e.g. from 0 to 32.52 for point sources), mean concentrations are consistent with previous literature analyzing endocrine disruptors in airborne particles (Salgueiro-González et al. 2015). Non-point pollution sources emitted more EDCs in 2005 when compared to point source and total estimates. In demographic regards, the average percentage of high school drop outs and female-headed households was 7 percent and 1 percent, respectively. The average percentage of county populations unemployed (2%) and living below the poverty line (6%) were much lower compared to the national average of unemployment (8%) and adults living in poverty (13%) in 2010 (Bureau of Labor Statistics). Counties differed in their degree of immigrant isolation, but the mean percentage of foreign born and linguistically isolated households was 5 percent and 11 percent correspondingly.   16 percent was the average non-white population size per county, and the range was 0.8 percent to 97.1 percent. Population density ranged from 100 to 69,467 people and about 50 percent of that population was female. The average travel time commuters spent traveling to work was approximately 23 minutes a week.

Table 2.3 presents the logistic regression results for all three exposure sources. In Models 1 through 3, economic deprivation was significantly associated with higher levels of point source and total estimated concentrations of airborne endocrine disruptors when controlling for covariates. In Models 4-6, immigrant isolation and ethnic composition were added to extend previous models and both were positive, significant predictors of non-point source emission concentrations. Immigrant isolation was associated with a 0.156 unit increase in non-point emissions and ethnic composition was correlated with a 0.085 unit increase in non-point emissions. Additionally, the estimated effect of economic deprivation became non-significant for point-source and total emissions estimates, but is significantly and negatively associated with non-point source emissions. This suggests that minority and immigrant concentration may mediate the extent to which economic deprivation predicts emissions estimates.

Average commuter time to work was a non-significant predictor except in Model 4.   Average time to work was a significant, positive predictor (0.034, $p<0.05$) of non-point source emission concentrations. This result is consistent with previous studies and reflects the effect mobile sources, mainly automobiles, have on non-stationary forms of emittances, which are increasingly becoming a concern in urban areas (Bell and Ebisu

2012). This finding may also signify that non-point source concentrations are driving total exposure concentrations. Finally, despite being non-significant in most models, population density was negatively correlated with exposure estimates in the fully saturated model. This finding does not support prior research which has found a positive association between population size and pollution exposure (Pastor et al. 2005; Zwickl, Ash, and Boyce 2014). Because this is a chemical-specific exposure analysis, while most others have examined general air pollution, this null result might suggest that there is no difference in risk of exposure to EDCs between urban and rural settings.

Discussion and Conclusions

The results of this cross-sectional, population-based analysis indicate that the associations between exposure to estimated levels of airborne endocrine disputing chemicals, economic deprivation, and racial/ethnic concentration are complex. On one hand, the results of this study bolster previous research on environmental inequality as counties with higher levels of immigrant isolation and ethnic composition were found to have higher levels of non-point source emission concentrations. This result suggest that immigrant and minority communities may experience higher amounts of air pollution stemming from non mobile sources like automobiles. Related studies have found that racial and ethnic minority communities and foreign-born persons are more likely to live within 150 meters of major highways than whites and, as such, may have increased exposure to traffic-related air pollution and associated health risks (Collins et al. 2011; Woghieren-Akinnifesi 2013).

On the other hand, the negative and largely non-significant findings regarding the effect economic deprivation has on emissions concentrations are not consistent with traditional environmental inequality scholarship. However, similar null results have been found when examining the SES-race-exposure relationship (Anderton et al. 1994; Bryant and Mohai 1992; Ringquist 2005). Inconsistent findings can partially be explained by the complexity of these relationships. Researchers have found that environmental inequality outcomes vary widely across communities (Downey 2007) because community-level characteristic, such as economic deprivation, have interacting effects on environmental risks (Grant et al. 2010). An additional challenge in this area, which may have influenced the results of this study, includes the operationalization and choice of indicators. For example, true socioeconomic status relates to the historical conditions in which economic deprivation is produced. Creating a quantitative index that captures these processes is a challenge and may simplify the phenomenon to the point where effects are non-detectable (Bell et al. 2002).

This study is unique within the field of environmental inequality because it examined chemical-specific air pollution exposure. Correlations between overall air pollution exposure, SES, and race/ethnicity have been studied widely (Ard 2015; Bravo et al. 2016; Pais, Crowder, and Downey 2014). An aim of this research was to integrate environmental inequality and potential health impacts into sociological research by specifically examining a sub-class of chemicals linked to adverse health outcomes like obesity. Endocrine disruptors have been associated with various health issues and their potential to cause serious health risks is so alarming that the European Union is currently deciding on wheatear to ban the use of EDCs in commonly used products (e.g. personal hygiene products) (World Health Organization 2013). Although non-significant predictor effects were found, our analyses pave the way for future research on the unequal distribution of EDCs within diverse communities; which is urgently needed to inform environmental health policy.

Identifying specific pollution sources related to environmental disparities and health risks can also influence public policy. The design of this study highlights the importance of analyzing various forms and manifestations of pollution exposure. Few studies have disentangled the separate effects of mobile and stationary air pollution sources (Schlosberg 2013). In subsequent research, exposure source types should be assessed with the goal of providing evidence for more efficient, targeted environmental regulations.

The results of this study must be considered within the context of two limitations. First, the analyses relied on cross-sectional data from different years. As such, no causal inferences can be drawn between the observed associations. The cross-sectional design also failed to measure cumulative risks associated with endocrine disruptor exposure overtime. Second, environmental exposure measures were estimated and not objectively collected. We are therefore making educated assumptions about the amount and degree of exposures to EDCs, and this can produce biased results. However, exposure estimates used in this study are likely more conservative than not, because emittance data was largely self-reported by facilities, facilities that likely have more incentive to under report than over report their pollution emissions (Bullard 2005). Longitudinal studies employing objective measures of pollution exposure, such as biomonitoring, and multilevel modeling are needed to capture the combined effects of chemical exposures and sociocultural stressors on health in future research.

This study contributes to the multidimensional research on environmental inequality, health, and obesogenics. By analyzing how air pollution exposure varies across populations with different levels of economic deprivation, immigrant isolation, and ethnic composition, this study adds to the growing number of environmental inequality studies examining sociocultural processes that lead to discrimination. This research helps address gaps in traditional environmental justice work by seeking to uncover how cultural power differentials that lead to phenomena like immigrant isolation, increase the risk of environmental exposure. Scholars have urged environmental health inequality researchers to integrate “critical analyses of power as it plays out in (mal)distribution of harms and opportunities related to the environment with special attention to race and class” (Sze and London 2009: 1348). This study partially answers this call and hopefully it will encourage other scholars to pursue similar empirical investigations.

Table 2.1 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 (PCBs)	Electronics, plasticizer
Bis(2-ethylhexyl)phthalate(DEHP)	Plasticizer

Table 2.2 Descriptive statistics for census tract level data.

	N	Percent	Mean	SD	Minimum	Maximum
County-level Data (N= 1,971)						
Dependent Variables						
Total Non-Point EDC Pollution Exposure Concentration	1,971	100	0.05	0.31	0	9.50
Total Point EDC Pollution Exposure Concentration	1,971	100	0.29	1.64	0	32.52
Total EDC Pollution Exposure Concentration	1,971	100	0.35	1.68	0	32.59
Key Independent Variables						
Economic Deprivation Factor Variables						
Percent of population with no high school degree	1,971	100	17.53	7.42	1.34	53.56
Percent of population unemployed	1,971	100	7.08	2.93	0	30.77
Percent of population below poverty level	1,971	100	16.16	6.34	3.11	62.00
Percent female headed household	1,971	100	4.52	1.49	0.83	11.76
Immigrant Composition Factor Variables						
Percent of population foreign born	1,971	100	4.56	5.66	0	49.43
Percent of population linguistically isolated	1,971	100	9.26	11.59	0	96.00
Percent of population non-white	1,971	100	17.85	16.44	0.81	97.10
Control Variables						
Population density (per square mile)	1,971	100	319	2,094	100	69,467
Percent of population female	1,971	100	50.27	2.02	27.94	55.21
Average Time to Work (minutes)	1,971	100	9.38	2	0	23.15

Note:
Variables used in analyses were standardized to a mean of zero and standard deviation of one.

Table 2.3 OLS regression coefficients and standardized errors for airborne endocrine Disrupting chemical emissions, economic deprivation, and minority concentration at the county level.

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
	Non-Point Source EDC Pollution Exposure Concentration	**Point-Source EDC Pollution Exposure Concentration**	**Total EDC Pollution Exposure Concentration**	**Non-Point Source EDC Pollution Exposure Concentration**	**Point-Source EDC Pollution Exposure Concentration**	**Total EDC Pollution Exposure Concentration**
Key Independent Variable						
Economic Deprivation	0.004	0.034*	0.035*	-0.056*	-0.004	-0.014
	(0.010)	(0.016)	(0.016)	(0.027)	(0.025)	(0.025)
Immigrant Isolation				0.156**	0.019	0.040
				(0.058)	(0.025)	(0.027)
Ethnic Composition				0.085**	0.060	0.075
				(0.031)	(0.041)	(0.040)
Control Variables						
Population density (per square km)	0.037**	0.009	0.008	-0.023	-0.010	-0.015
	(0.012)	(0.008)	(0.009)	(0.026)	(0.005)	(0.008)
Percent of population female	0.012	0.017	0.019	0.021**	0.014	0.018
	(0.006)	(0.015)	(0.015)	(0.007)	(0.017)	(0.017)
Average Time to Work	0.013	-0.001	0.001	0.034*	0.0004	0.006
	(0.159)	(0.010)	(0.010)	(0.016)	(0.010)	(0.011)
Pseudo R Squared	0.002	0.016	0.018	0.034	0.004	0.008

Notes: ***p<.001 **p<.01 *p<.05

Robust standard errors in parentheses, N=1,971

Variables used in multilevel analyses were standardized to a mean of zero and standard deviation of one.

CHAPTER 3

AIRBORNE ENDOCRINE DISRUPTION CHEMICAL EXPOSURE AND OBESITY PREVALENCE ACROSS U.S. METROPOLITAN STATISTICAL AREAS

Introduction

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 35%) and approximately 17 percent of children in the U.S. are obese (Center for Disease Control 2015). More importantly, recent research suggests that the high level of obesity prevalence among adults and children has remained unchanged since 2003 and affect virtually all ages, races, sexes and socioeconomic groups (Newbold 2010; Ogden, Carroll, Kit, and Flegal 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, cardio vascular diseases, and hypertension (Flegal, Carroll, Kit, and Ogden 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 U.S. has increased because 1) caloric intake, especially of saturated fats, has increased and 2) physical activity has decreased (Trasande 2009). Contemporary research has broadened our understanding of obesity and we now know that obesity is most likely caused by complex interactions between behavioral, genetic and environmental factors (Hyman 2010). Although research and policy has focused on these interests, especially how to incorporate healthy foods in our diets and more exercise in our lifestyles, the exact etiology of obesity is still unknown. Using a nascent theoretical perspective, known as obesogenics, this study examines how environmental factors, specifically air pollution, may contribute to adult obesity prevalence in the United States.

The Obesogen 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 2007). In a landmark paper, Baillie-Hamilton (2002) argued that exposure to chemical toxins is correlated to rising obesity rates by showing that the global obesity epidemic coincides with significant increases of industrial chemicals in the environment over the last 40 years. Expanding on this work, Grun and Blumberg (2007) proposed the "environmental obesogens" hypothesis, which espouses that environmental pollutants can disrupt and interfere with the body's metabolic, energy balancing system and fat storage. Specifically, exposure to obesogens "promotes adiposity by altering programming of fat cell development [and] increasing energy storage in fat tissue" (Janesick and Blumberg 2016:1). 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 adipogensis, 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, adipogensis 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 and mis-regulate the critical pathways involved in adipogensis. In their review, Grun and Blumberg (2007) highlight research that demonstrates how chemicals, such as bisphenol-A (BPA), inhibit nuclear hormone receptor signaling pathways during adipogensis; which can cause in *increase* in fat cell production and, subsequently, obesity. In another review, Janesick and Blumberg (2016:8) conclude "that increased adipogenesis during early development permanently establishes an elevated fat cell number in adulthood". If this pathway exists, whereby obesogenic exposure early in the life course permanently increases the fat cell number within an individual, it's 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 suggest 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 the number of fat cells is largely independent of body mass index (BMI). This supports prior research which suggests that obesity mechanisms are complex and that by and large, obesity is a product of both increased adipose cell number *and* increased cell size (Salanes, Cushman, and Weismann 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 a higher number 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 of obesity in individuals by the aforementioned pathways; by increasing the number of fat cells and/or the storage of fat in existing cells (Holtcamp 2012). Additionally, EDCs have been shown to modify metabolic rate (Heindel 2011), 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 toxicology findings establishing a causal link between chemical exposure and metabolic disruption have involved endocrine disruptors (Navas-Acien et al. 2008). Endocrine disrupting chemicals are ubiquitous in both natural and built environments and common EDCs include: industrial chemicals like BPA and polychlorinated biphenyl ethers (PCBs); herbicides such as atrazine; pervasive environmental pollutants including particulate matter; and naturally occurring heavy metals such as mercury and lead (Ahearn 2012; Thaddeus 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 last few decades, exposure to EDCs has become increasingly widespread (Diamanti-Kandarakis et al. 2009).

Concern about exposure to endocrine disrupting chemicals is also becoming more widespread as evidence connecting EDCs to health issues related to fertility, cancer, metabolism, and obesity mounts. Within the last 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 biphenyl-A (BPA) in plastic water bottles. As a plasticizing 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 like baby bottles, water bottles, plastic cutlery, and plastic toys (CDC 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 leech into the contents of the plastic product (Vandenberg et al. 2007) or be absorbed through the skin (WHO 2013). BPA leeching is so ubiquitous that in their study of 2,517 participant urine samples, the Center for Disease Control (CDC) found detectable levels of BPA in nearly every participant, which "indicates widespread exposure to BPA in the U.S. population" (2012: 186). In response to findings such as these, consumers demanded that plastic water bottles be made BPA-free and in the last decade, many producers begun making BPA-free baby bottles, water bottles and canned food products.

Airborne Endocrine Disrupting Chemicals

As noted in the above example of BPA leeching, there are different routes through which toxic chemicals, like 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). Exposures to EDCs via dermal absorption and ingestion has been the focus of most epidemiolocal research to date. For example, several studies have detected endocrine disrupting chemicals, including EDC substances banned in the European Union, in everyday cosmetic products that contact our skin like 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 (Perez-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 micrometers in diameter or less, have the ability to cause the most damage because they can migrate deep into the lungs and potentially into the blood stream (Salgueiro-Gonzalex et al. 2015). Once in the blood stream, 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 certain class of EDCs that include polyvinyl chloride (PVC), are among the most common outdoor and indoor airborne endocrine disrupting chemicals. 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 concentration of certain phthalates and obesity prevalence; specifically, men had generally higher odds of obesity compared to women with similar EDC exposure levels. Zhang and colleagues (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 endocrine disrupting chemicals and obesity prevalence.

The Sociology of Obesogenes

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 largely been conducted 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 socieoeconomic status (SES), 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 1998,2006; Mohai and Saha 2007; Ringquist 2005; Zwickl, Ash, and Boyce 2014), environmental racism (reviewed in 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 (1999) social determinates of health and Link and Phelan's (1995) fundamental causes of disease theories could help elucidate why certain groups (e.g. males compared to females or Whites comparted to non-Whites) experience differential obesity outcomes with the same degree of obesogenic exposure.

Employing a sociological lens, the present study addresses gaps in obesogenic research by employing multilevel analyses to examine the association between airborne endocrine disrupting chemical exposure and adult obesity prevalence across U.S. metropolitan areas. This large scale comparative study is, to the author's knowledge, the first of its kind. Based on findings from previous obesogenic research, it is specifically hypothesized that:

H_1: increased exposure to annual concentrations of airborne EDCs is associated with greater probability of being obese.

H_2: increased exposure to annual concentrations of airborne EDCs is associated with greater probability of being morbidly obese.

H_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 (BRFSS)

Individual-level demographic and health data for the present study were collected from the 2005 Behavioral Risk Factor Surveillance System (https://www.cdc.gov/brfss/annual_data/ annual_2005.htm, accessed December 2015). The BRFSS is a longitudinal survey project randomly administer via phone to households across the U.S. by the Centers for Disease Control and Prevention (CDC). The BRFSS collects individual level data for adults 18 years or older in all 50 states. Survey questions are designed to ascertain preventative 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 (CDC 2005). To protect participant privacy, publicly assessable 2005 BRFSS data is aggregated to the level of metropolitan statistical area (MSA) and denoted as the BRFSS SMART dataset. MSAs are geographic areas mapped every ten years by the U.S. Office of Management and Budget in preparation for the decennial census. MSAs regions consist of two or more adjacent counties that share an urban core, have a population greater than 50,000 each, or a high degree of economic and social integration (United States Census Bureau 2016). For example, the 2005 Salt Lake-Utah MSA was comprised of both Salt Lake and Toole county.

National Air Toxics Assessment

Air pollution emissions data was obtained from the United States Environmental Protection Agency's (EPA) 2005 "National Air Toxics Assessment" (NATA) online database (https://www.epa.gov/national-air-toxics-assessment/2005-nata-assessment-results#state, accessed December 2015). The NATA emissions data is compiled from a variety of sources including: state and local air pollutant inventories, the EPA's Toxic Release Inventory (TRI) database, and emissions estimates from the EPA's Office of Transpiration and Air Quality. This compiled National Emissions Inventory (NEI) 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 by the EPA 2011). In 2005, NATA estimated 177 of 187 air toxins listed under the 1990 Clean Air Act Amendment. Lastly, NATA data is 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 smoke stack or sewage treatment plant. Non-point emission sources are mobile sources that include automobiles, wildfire smoke, and sediment kick-up from mining and construction areas (EPA 2012). County level ambient air toxin concentration estimates were aggregated to the level of metropolitan statistical area to match data obtained from the BRFSS.

Sample

Behavioral Risk Factor Surveillance System (BRFSS)

202,904 records were initially retrieved from the 2005 BRFSS SMART dataset. Although the 2005 BRFSS SMART dataset collected 216,379 observations, at the time data was downloaded for this project, only 202,904 records were available for public download. Due to lack of exposure data from NATA (see following subheading) observations from Wyoming and Alaska were excluded from the final sample. Additionally, women pregnant during the survey period were exclude 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 (n =3,312), extremely obese with BMI > 60 (n = 9,457), or had missing BMI data (n =418) were excluded from the sample

(Brown et al. 2013). List-wise deletion on relevant covariates was conducted and 517, 749 and 419 observations were dropped from the sample due to missing data for level of education, marital status, and BMI, respectively. Finally, individuals from Alaska and Wyoming (n=222) were excluded from the sample due to missing pollution exposure data (detailed below). an analytical sample of 188,252 individuals, approximately 93 percent of the original BRFSS sample, was generated.

National Air Toxics Assessment

Because prior obesogenic research has found strong associations between endocrine disrupting chemicals and obesity, estimated ambient concertation of air toxins was limited to seven know EDCs (Arner et al. 2010; Grun and Blumberg 2006; Janesick and Blumberg 2011; Vandenberg et al. 2012). These seven compounds include: 5 insecticides/fungicide (chlordane, hexachlorobenzene, hexachlorocyclohexane**,** methoxychlor, and toxaphene), 1 polychlorinated organic compound (polychlorinated biphenyls (PCBs)), and 1 phthalate (bis(2-ethylhexyl)phthalate(DEHP)). Characteristic of each toxin summarized in Table 3.1. County level annual ambient concentration estimates ($\mu g/m^3$) for the 7 EDCs was 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, all 7 concentration estimates were added together to produce more robust exposure variables. Point and non-point source ambient air toxic estimations for 153 metropolitan statistical areas were used in this analysis.

Measures

Dependent Variables

Body mass index (BMI) was computed using respondents' self-reported height and weight (BMI= mass (kg)/(height (m))^2. Obesity was measured by a categorical variable indicating obesity status as either obese (BMI$\geq$ 30) or morbidly obese (BMI$\geq$ 35), consistent with CDC guidelines (2016) and previous studies (Fan, Wen, and Kowaleski-Jones 2016; Lie et al. 2015; Ogden et al. 2014).

Metropolitan Statistical Area EDC Exposure Variables

Airborne endocrine disrupting chemical pollution exposure was assessed by using annual ambient emission concentration estimates (μg/m^3) for point, non-point, and total (point plus non-point) emission sources. These total estimates are a summation of concentration estimates for the seven EDCs discussed above and are measured as continuous variables. Exposure measures were standardized to account for nonlinearity and heteroscedasticity (Long and Freese 2006). Analyzing non-point and point source pollution estimates separately can be helpful in assessing cumulative impacts of air pollution exposure (Morello-Frosh, Pastor, and Sadd 2001; Morello-Frosh 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

Based on past research, individual demographic covariates used in the analysis include age and age^2 (Flegal et al. 2012), sex (Grun and Blumberg 2009; Weng and

Beydoun 2007), and marital status (Sobal, Rauschenbach, and Frongillo 2003). Socioeconomic (SES) status was measured with two indicators, annual household income and level of education. Binary coded household income categories include (in U.S. dollars): 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 their 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 is a categorical variable comparing Whites (reference group), Hispanics, Blacks, Asians, Native Hawaiian/Pacific Islander, American Indian/Alaskan Native, Multiracial and individuals that identify as an unlisted race.

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 (Wang, Wen, and Xu 2013; Wen and Maloney 2011; Xu, Wen, and Wang 2014). The hierarchal 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 Statistical Software, version 13 (StataCrop. 2013. *Stata Statistical Software: release 13. College Station, TX: StataCorp L*P; Rabe-Hesketh and Skrondal 2008).

Results

Table 3.2 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 Hawaiian/Pacific Islander, American Indian/Alaskan Native, multiracial, and those that 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 have attended college. At the MSA-level, the non-point source estimated ambient concertation of EDCs ranges from -0.67 to 6.22 (μg/m^3) and point source estimated ambient concertation range from -0.23 to 7.21 (μg/m^3).

Table 3.3 presents odds ratios from the multilevel models for EDC concentrations on individual risk of obesity for non-point source and point source pollution concentration estimates. Both non-point and point source airborne pollution concentrations are negatively associated with obesity (OR 0.999 and 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 "age-squared" is negative and significant, suggesting that the age-obesity trend reverses after reaching a certain age (Xu, Wen, and Wang 2014). Each levels of education and income category is significant and positively associated with obesity. In regards to race, being black (OR 1.81, $p<.001$), Hispanic (OR 1.12, $p<.001$), Native Hawaiian or Pacific Islander (OR 1.56, $P<.001$), and 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 of obesity when compared to Whites. Marital status is statistically non-significant, but being female is a significant predictor of lower obesity odds (OR 0.957, p<.001).

Table 3.4 shows odds ratios for morbid obesity and EDC concentration estimates for both pollution source types. Although statistically non-significant, non-point source is negatively associated with morbid obesity while point source concentrations estimates are positively associated with morbid obesity. Individual-level results from Table 3.3 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<0.001) is associated with lower odds of morbid obesity when compared to single individuals. Additionally, the significantly positive association between being Hispanic and obesity is rendered insignificant when assessing morbid obesity.

Table 3.5 and 3.6 report gender specific results for obesity. When models were estimated separately by gender, findings show that non-point source ECD concentration estimates were not significantly associated with obesity for women or men. Although non-significant, 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 making $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 a higher obesity for women and men, although the odds are higher for women compared to men. Identifying as Hispanic was a positive predictor for obesity risk among women (OR 1.14, p<.001), but was a non-significant 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*, non-significant associations for key pollution exposure predictors were found (results now shown) and those models are available upon request from the author.

Discussion and Conclusion

The purpose of this research was to ascertain the effect MSA-level endocrine disrupting chemical emission estimates have on individual-level risk of 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 effect obesity risk differently. In these analyses, airborne endocrine disrupting chemical emissions was not associated with obesity, regardless of pollution source type. This finding rejects hypothesis 1, which predicts that higher exposure levels should be associated with a greater risk of obesity based on 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 2 EDCs in utero, hexachlorobenzene (HCB) and dichlorodiphenyldichloroethane (DDE), and increased BMI of children at age four in a longitudinal cohort study conducted in Greece. Tang-Peronard et al. (2014) also found an association between elevated BMIs in female children 7 years old and prenatal exposure to polychlorinated biphenyls (PCBS) and DDE; associations for male children were non-significant. Wang and colleagues (2012) found that urine biphenyl-A (BPA) concentrations were significantly associated with increasing BMI values 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 (MEHP), was associated with lower BMI in adolescent girls and women aged 20-59. The authors hypothesize that EDC exposure may reduce hormone levels in the body, which “could help explain the inverse relationship between MEHP and BMI” (Hatch et al. 2010: pg 6). In other words, exposure to endocrine disrupting chemicals may alter hormone 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 non-significant. This finding suggests that if correlations between EDC exposure and excessive weight do exists, 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 and point source EDC emissions were not significant predictors of obesity risk. Although non-significant, 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 general obesity literature (reviewed in Flegal et al. 2012; Tang-Peronard et al. 2008) as well as the obesogenic research reviewed above (Hatch et al. 2010; Tang-Peronard 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 their 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 males and females (Heindel et al. 2015). Gender differences in obesity risk associated with EDC exposure is 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 employed 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, lifespan analyses assessing cumulative pollution exposure and obesity should be conducted in the future (Heindel et al. 2015; Janesick and Blumberg 2016; Morello-Frosh et al. 2011). Second, independent-level variables, including the dependent variable (BMI), relied on self-reported data and was subject to response bias. EDC exposure concentration estimates may also be bias. NATA data that is not directly collected through monitoring systems are self-reported by individual industries, which could lead to biased estimations; some argue estimations may be more conservative because industries are incentivize to underreport emissions to meet federal regulations (Apelberg, Buckley, and White 2005). The small estimates of the seven individual EDCs also did not allow for 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 Jansick and Blumberg 2016). Lastly, due the lack of publicly available data, potential MSA-level confounders related to the built environment, such as street connectivity and the ratio of fast food to full service restaurants, was not accounted for (Xu, Wen, and Wang 2014).

Despite this study's limitations, this research adds to prior obesogenic research in several significant ways. First, few studies have used a large study population to examine the direct effects of air pollution on obesity prevalence (Li et al. 2015). And to the author's 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: pg. 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 and point source emission types, we can potentially identify which environmental hazards impact human health most. In this study, non-point source emission concentration of EDCs was found to significantly effect obesity risk while point source estimates were non-significant; signifying that emissions from mobile sources, like cars and mining sites, might be more worthwhile targets for emission reduction and public health policies.

In conclusion, this exploratory study examined the association between MSA-level endocrine disrupting chemical emission estimates and individual-level risk of obesity and morbid obesity while accounting for socioeconomic factors, race and ethnicity, and health characteristics of the individual. Findings suggest that non-point source exposure to EDCs reduces the risk of obesity. Gender differences may drive this finding; although non-significant, women were found to have reduced obesity risk with higher EDC exposure concentrations, while this effect was not found in men. The results do 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 of obesity, more sociologically driven research is needed to ascertain the social conditions under which weight gain/weight loss is exacerbated by endocrine disrupting chemical exposure.

Table 3.1

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 (PCBs)	Electronics, plasticizer
Bis(2-ethylhexyl)phthalate(DEHP)	Plasticizer

Table 3.2

Descriptive statistics for Metropolitan Statistical Areas and individual variables used in two-level analyses.

	N	Percent	Mean	SD	Minimum	Maximum
Level 2 Metropolitan Statistical Area (n= 153)						
Non-Point Source Estimated Airborne EDC Concentration	188,252	100.00	0.0003	0.0004	-0.676	6.22
Point Source Estimated Airborne EDC Concentration	188,252	100.00	0.001	0.008	-0.233	7.21
Level 1 Individual level (n=188,252)						
Obese (BMI≥30)	46,227	24.56	0.24	0.43	0	1
Morbidly Obese (BMI≥35)	16,247	8.63	0.08	0.28	0	1
Body Mass Index	188,252	100.00	27.19	5.49	18.5	60
Median Annual Household Income, US Dollars						
≤ 15,000	16,600	8.82	0.08	0.28	0	1
15,001-25,000	26,597	14.13	0.14	0.34	0	1
25,001-35,000	20,641	10.96	0.10	0.31	0	1
35,001-50,000	27,004	14.34	0.14	0.35	0	1
≥ 50,001	74,213	39.42	0.39	0.48	0	1
Income Unknown	23,197	12.32	0.12	0.32	0	1
Education Level						
No High school degree	16,252	8.63	0.08	0.28	0	1
High school degree	52,385	27.83	0.27	0.44	0	1
Some college	49,406	26.24	0.26	0.43	0	1
College degree	70,209	37.30	0.37	0.48	0	1
Married	107,561	57.14	0.57	0.49	0	1
Female	113,055	60.06	0.60	0.48	0	1
Age	188,252	100.00	50.76	17.2	18	99
White	144,854	76.95	0.79	0.42	0	1
Black	16,646	8.84	0.08	0.28	0	1
Hispanic	12,755	6.78	0.06	0.25	0	1
Asian	4,776	2.54	0.02	0.15	0	1
Native Hawaiian/Pacific Islander	598	0.32	0.003	0.56	0	1
American Indian/Alaskan Native	1,969	1.05	0.01	0.10	0	1
Multiple Races	3,886	2.06	0.20	0.14	0	1
Other Race	2,768	1.62	0.01	0.07	0	1

Notes:
EDC exposure variables used in analyses were subsequently standardized to a mean of zero and standard deviation of one.
Body mass index was calculated as weight (kg)/height(m)2

Table 3.3

Adjusted odds ratios (95% confidence interval) of the multilevel logistic model for odds of obesity (BMI≥30).

	Non-Point Source Estimated Airborne EDC Concentration	**Point Source Estimated Airborne EDC Concentration**
MSA-Level Variables		
EDC Exposure	0.999	0.998
	(0.972-1.026)	(0.967-1.031)
Individual-Level Variables		
Age	1.106***	1.106***
	(1.101-1.110)	(1.101-1.110)
Age2	0.999***	0.999***
	(0.998-0.999)	(0.998-999)
Married	0.987	0.985
	(0.936-1.011)	(0.963-1.011)
Female	0.957***	0.956***
	(0.936-0.979)	(0.936-979)
No high school degree	1.779***	1.779***
	(1.703-1.858)	(1.703-1.858)
High school degree	1.534***	1.534***
	(1.490-1.580)	(1.490-1.580)
Some College	1.457***	1.457***
	(1.415-1.499)	(1.415-1.499)
Annual Household Income, US Dollars		
≤ 15,000	1.567***	1.567***
	(1.490-1.580)	(1.493-1.644)
15,001-25,000	1.398***	1.398***
	(1.339-1.460)	(1.339-1.460)
25,001-35,000	1.327***	1.327***
	(1.267-1.389)	(1.267-1.389)
35,001-50,000	1.278***	1.278***
	(1.223-1.335)	(1.223-1.335)
≥50,001	1.066**	1.066**
	(1.024-1.109)	(1.024-1.109)
Black	1.813***	1.812***
	(1.746-1.882)	(1.746-1.882)
Hispanic	1.124***	1.124***
	(1.074-1.177)	(1.074-1.177)
Asian	0.381***	0.382***
	(0.344-0.423)	(0.344-0.423)
Native Hawaiian/Pacific Islander	1.569***	1.569***
	(1.313-1.874)	(1.313-1.874)
American Indian/Alaskan Native	1.418***	1.417***
	(1.286-1.563)	(1.286-1.563)
Multiple Races	1.379***	1.379***
	(1.282-1.483)	(1.283-1.483)
Other Race	1.061	1.061
	(0.924-1.218)	(0.924-1.218)
N	188,252	188,252

Notes: ***p<.001 **p<.01 *p<.05

95% confidence intervals in parentheses

EDC exposure variables used in multilevel analyses were subsequently standardized to a mean of zero and standard deviation of one.

Table 3.4

Adjusted odds ratios (95% confidence interval) of the multilevel logistic model for odds of morbid obesity (BMI≥35).

	Non-Point Source EDC Pollution Exposure Concentration	**Point-Source EDC Pollution Exposure Concentration**
EDC Exposure	0.999	1.001
	(0.967-1.033)	(0.963-1.040)
Demographic Variables		
Age	1.131***	1.131***
	(1.123-1.138)	(1.123-1.138)
Age2	0.998***	0.998***
	(0.998-0.998)	(0.998-0.998)
Married	0.918***	0.918***
	(0.884-0.952)	(0.884-0.952)
Female	1.288***	1.281***
	(1.237-1.327)	(1.237-1.326)
Socioeconomic Variables		
No high school degree	1.852***	1.852***
	(1.737-1.975)	(1.737-1.975)
High school degree	1.496***	1.496***
	(1.429-1.566)	(1.429-1.566)
Some College	1.488***	1.488***
	(1.423-1.557)	(1.423-1.557)
Annual Household Income, US Dollars		
≤ 15,000	1.817***	1.817***
	(1.693-1.950)	(1.693-1.950)
15,001-25,000	1.479***	1.479***
	(1.385-1.581)	(1.351-1.581)
25,001-35,000	1.339***	1.339***
	(1.248-1.437)	(1.248-1.437)
35,001-50,000	1.211***	1.211***
	(1.131-1.296)	(1.131-1.296)
≥50,001	0.956**	0.921**
	(0.865-0.982)	(0.891-1.025)
Race and Ethnic Minority Variables		
Black	1.775***	1.775***
	(1.686-1.869)	(1.686-1.869)
Hispanic	0.956	0.956
	(0.891-1.025)	(0.891-1.025)
Asian	0.284***	0.284***
	(0.232-0.348)	(0.232-0.348)
Native Hawaiian/Pacific Islander	1.823***	1.823***
	(1.439-2.309)	(1.440-2.309)
American Indian/Alaskan Native	1.374***	1.374***
	(1.199-1.575)	(1.200-1.575)
Multiple Races	1.446***	1.446***
	(1.306-1.602)	(1.306-1.602)
Other Race	1.067	1.067
	(0.865-1.316)	(0.865-1.316)
N	188,252	188,252

Notes: ***p<.001 **p<.01 *p<.05
95% confidence intervals in parentheses
EDC exposure variables used in multilevel analyses were subsequently standardized to a mean of zero and standard deviation of one.

Table 3.5

Adjusted odds ratios (95% confidence interval) of the multilevel logistic model for odds of obesity (BMI≥30) for females.

	Non-Point Source EDC Pollution Exposure Concentration	**Point-Source EDC Pollution Exposure Concentration**
EDC Exposure	0.999	0.998
	(0.971-1.028)	(0.965-1.032)
Demographic Variables		
Age	1.109***	1.109***
	(1.103-1.028)	(1.103-1.114)
Age2	0.999***	0.999***
	(0.998-0.999)	(0.998-0.999)
Married	0.912***	0.912***
	(0.883-0.942)	(0.883-0.942)
Socioeconomic Variables		
No high school degree	2.035***	2.035***
	(1.924-2.152)	(1.924-2.152)
High school degree	1.605***	1.605***
	(1.543-1.668)	(1.543-1.668)
Some College	1.511***	1.511***
	(1.454-1.569)	(1.454-1.569)
Annual Household Income, US Dollars		
≤ 15,000	1.675***	1.675***
	(1.580-1.775)	(1.580-1.775)
15,001-25,000	1.500***	1.500***
	(1.422-1.582)	(1.422-1.582)
25,001-35,000	1.446***	1.446***
	(1.365-1.531)	(1.365-1.531)
35,001-50,000	1.352	1.352
	(1.279-1.428)	(1.279-1.428)
≥50,001	0.967	0.967
	(0.918-1.018)	(0.981-1.018)
Race and Ethnic Minority Variables		
Black	2.054***	2.054***
	(1.960-2.151)	(1.960-2.151)
Hispanic	1.148***	1.148***
	(1.082-1.217)	(1.082-1.217)
Asian	0.366***	0.366***
	(0.316-0.422)	(0.316-0.422)
Native Hawaiian/Pacific Islander	1.341*	1.341*
	(1.046-1.717)	(1.046-1.717)
American Indian/Alaskan Native	1.319***	1.319***
	(1.160-1.498)	(1.160-1.498)
Multiple Races	1.400***	1.400***
	(1.273-1.540)	(1.273-1.540)
Other Race	1.146	1.146
	(0.949-1.381)	(0.949-1.381)
N	113,055	113,055

Notes: ***p<.001 **p<.01 *p<.05

95% confidence intervals in parentheses

EDC exposure variables used in multilevel analyses were subsequently standardized to a mean

Table 3.6

Adjusted odds ratios (95% confidence interval) of the multilevel logistic model for odds of obesity (BMI≥30) for males.

	Non-Point Source EDC Pollution Exposure Concentration	**Point-Source EDC Pollution Exposure Concentration**
EDC Exposure	0.989	1.001
	(0.959-1.021)	(0.966-1.036)
Demographic Variables		
Age	1.104***	1.104***
	(1.096-1.111)	(1.096-1.111)
Age2	0.999***	0.999***
	(0.998-0.999)	(0.998-0.999)
Married	1.167***	1.167***
	(1.123-1.212)	(1.123-1.212)
Socioeconomic Variables		
No high school degree	1.522***	1.521***
	(1.418-1.632)	(1.418-1.632)
High school degree	1.485***	1.484***
	(1.419-1.553)	(1.419-1.553)
Some College	1.421***	1.420***
	(1.359-1.485)	(1.359-1.485)
Annual Household Income, US Dollars		
≤ 15,000	1.274***	1.274***
	(1.166-1.391)	(1.167-1.391)
15,001-25,000	1.213***	1.213***
	(1.125-1.308)	(1.125-1.308)
25,001-35,000	1.163***	1.163***
	(1.077-1.256)	(1.077-1.256)
35,001-50,000	1.182***	1.182***
	(1.100-1.270)	(1.100-1.270)
≥50,001	1.148***	1.148***
	(1.076-1.224)	(1.076-1.224)
Race and Ethnic Minority Variables		
Black	1.378***	1.376***
	(1.292-1.470)	(1.290-1.468)
Hispanic	1.060	1.061
	(0.985-1.140)	(0.986-1.141)
Asian	0.391***	0.391***
	(0.336-0.453)	(0.336-0.453)
Native Hawaiian/Pacific Islander	1.863***	1.864***
	(1.439-2.409)	(1.440-2.411)
American Indian/Alaskan Native	1.578***	1.578***
	(1.356-1.835)	(1.357-1.836)
Multiple Races	1.354***	1.354***
	(1.210-1.514)	(1.210-1.515)
Other Race	0.984	0.983
	(0.800-1.207)	(0.800-1.206)
N	75,197	75,197

Notes: ***p<.001 **p<.01 *p<.05

95% confidence intervals in parentheses

EDC exposure variables used in multilevel analyses were subsequently standardized to a mean

CHAPTER 4

GESTIONAL WEIGHT GAIN AND OCCUPATIONAL EXPOSURE TO ENDOCRINE DISRUPTING CHEMICALS AMONG WOMEN IN UTAH FROM 2004-2008

Introduction

Obesity prevalence in the United States is at epidemic levels and remains a significant public health threat. Over 115 million adults and children in the U.S. are considered overweight and many are at risk for obesity related conditions including, type-2 diabetes and heart disease (Ogden et al. 2015). To date, most health campaigns aim to reduce the risk of excessive body weight by focusing on promoting healthy-lifestyle habits. This individual level approach is informed by the energy-balance theory, which suggest that body weight is simply a product of energy intake (e.g. eating, consuming calories) and energy output (e.g. physical activity, burning calories; Hill et al. 2003). Many scholars think this theory is too reductionist and find it ignores important social, demographic, and economic conditions that contribute to obesity. One particular school of thought, known as obesogenics, suggests that exposure to environmental hazards and toxins can increase obesity risk and prevalence. Researchers in this camp point to obesity trends and the fact that as a population, we are 20 to 30 percent heavier than we were two decades ago; concluding that "whatever is happening is happening to everyone, suggesting an environmental trigger" (Baillie-Hamilton 2002; Lustig 2006:448). Of course, environmental triggers refer to a broad range of factors including social conditions related to the built environment and diet, to anthropogenic conditions related to pollution exposure to non-chemical and chemical toxins.

Chemical obesogenic research is driven by the theory that chemical "obesogens" present in the environment may alter metabolic processes within the body and predispose some people to gain weight (Blumberg 2006). Over the last ten years, scholars from endocrinology and toxicology have identified chemical toxins that once in the body, disrupt metabolic processes that control fat storage (Schug et al. 2011) and metabolism (Heindel et al. 2010). Interruptions such as these can make it harder to maintain and lose weight and, more importantly, set vulnerable populations (e.g. children and adolescents) on trajectories of excessive weight gain throughout the life course (Zoeller et al. 2012). Chemicals with the ability to affect the body's energy balancing system are called endocrine disruptors (EDCs) and include common pesticides, pharmaceuticals, and industrial compounds like plasticizers (Newbold 2009; Janesick and Blumberg 2011). Studies examining the direct linkages between EDC exposure and obesity have yielded mixed results. In Chinese populations, researchers have found positive associations between exposure to specific endocrine disruptors, known as phthalates, and increased body mass index (BMI) and waist circumference among children (Wang et al. 2012; Zhang et al. 2014). Phalates have also found to be positively and significantly correlated with abdominal obesity in men (Stahlhut et al. 2007), male children and adolescents (Buser et al. 2014). For women, exposure to EDCs is positively correlated with BMI for certain types of phalates, those often found in shampoo and perfume, while negatively correlated for other chemical subclasses (Hatch et al. 2008). In addition to gendered variations, obesogenic research has uncovered age-specific effects of exposure on obesity outcomes (Hatch et al. 2010). Although a pattern is hard to establish with so few studies, adolescents and older adults may experience an inverse association between exposure and weight gain compared to children and adults (Heindel et al. 2015; Tang-Peronard et al. 2008). A few studies have found no correlation between exposure and obesity. In their cohort study examining urine samples of children in India, Xu et al. (2015) found non-significant associations between obesity and 10 of the11 endocrine disruptors analyzed. Buckley and colleagues (2016) also found no evidence of association between phalate exposure and childhood obesity.

Although research exploring direct links between body weight and EDC exposure in adults and children is limited in number, two general conclusions can be drawn from the obesogenic literature. First, associations between exposure and obesity seem to be chemical, gender, and age specific. Meaning, specific chemicals may influence the weight of women and men differently and these effects likely change throughout the life course. The exposure-obesity relationship seems to be complex, which may help explain why evidence regarding this association varies. Identifying windows of exposure susceptibility throughout the life course may yield more robust correlations and bolster our ability to make stronger conclusions about the role obesogens play in the etiology of obesity (Schug et al. 2011). The present study examines one such window of exposure susceptibility, pregnancy.

Previous obesogenic work has focused on studying the relationship between EDCs exposure during pregnancy and obesity outcomes of the child. To date, research strongly suggests that increased exposure to endocrine disruptors in utero produces higher obesity risks for children later in life. For example, Agay-Shay and colleagues (2015) found significant and positive associations between the concentration of common EDCs, including bisphenol A (BPA), in pregnant women and BMI among their children at age seven. Similar studies have demonstrated links between prenatal exposure and increased infancy weight (Valvi et al. 2014) and higher BMI at age four (Vafeidali et al. 2015). Unlike prior research, this study analyzes the association between EDC exposure and the mother's weight gained during pregnancy (gestational weight gain (GWG)). Because the endocrine system, which regulates hormones and metabolism, undergoes more productivity and stress during pregnancy, women's bodies are thought to be highly susceptible to the adverse effects of endocrine disruptors while pregnant (Schug et al. 2011; World Health Organization 2010). As such, obesogenic effects might be magnified in pregnant women and more easily discernable.

The second general finding stemming from chemical obesogenic research is that endocrine disrupting chemicals are ubiquitous in both the natural and built environment (Zoeller et al. 2012). EDCs have been found in air particulate matter (Tiel 2016), drinking water (Valvi et al. 2011), plastic toys, (Stahlhut et al. 2007), and even in vinyl records and designer handbags (Kannan et al. 2010). It stands without question that EDCs have become an integral part of the environments where we live, work, and play. It is suggested that the most common location for EDC and obesogenic exposure is the workplace (Snijder et al. 2011). A rich body of literature has linked EDC occupational exposure to adverse health outcomes, including lymphoma (Costas et al. 2015), diabetes (Arner et al. 2010) and hypospadias (Van Tongeren 2002). As far as the author is aware, this study is the first of its kind to assess the association between occupational exposure to endocrine disruptors and gestational weight gain.

The fundamental question driving obesogenic research is, do environmental toxins play a role in the etiology of obesity? This query aligns well with sociological methodology and theory but unfortunately, social science has largely been absent from obesogenic research. The present study seeks to address this gap by drawing on theories from environmental inequality to examine the effects race and ethnicity have on associations between EDC exposure and gestational weight gain. A strong body of evidence suggests that racial and ethnic minorities are disproportionately exposed to environmental hazards and harms (Bowen 2002). In general, racially marginalized and ethnically isolated groups experience higher risks of: living near waste sites (Atlas 2002; Bullard 1990; Hooks and Smith 2004) and polluted industries (Crowder and Downey 2010; Downey 2005; Downey and Hawkins 2008; Grand et al. 2010; Mohai et al. 2009), air pollution exposure (Ard 2016; Bravo et al. 2016; Downey 2005; Jerrett et al. 2004; Pastor et al. 2005; Zwickle, Ash, and Boyce 2014), and poor drinking water quality (Szasz and Meuser 1997). Especially salient to this study, racial/ethnic disparities in exposure to occupational hazards have been well documented. Findings suggest that certain minority groups are at risk for higher occupational exposures because cultural, economic, and social forces funnel them into more hazardous jobs (Murray 2003). Greater exposure to environmental toxins may make racial/ethnic populations more susceptible to health risks associated with various forms of pollution exposure.

Environmental health research has documented various pathways through which social conditions effect both environmental quality and health. For example, low socioeconomic status (SES) has been correlated with higher levels of air pollution exposure and associated asthma rates (Jerret et al. 2004; Morrello-Frosh 2001), cancer prevalence (Apelberg et al. 2005; Chakraborty 2012; Lievanos 2015; Linder Mrko, and Sexto 2008;), and overall mortality risk (Laurent et al. 2007; Zeka et al. 2006). SES influences individuals' health by constraining the choices they have when deciding what neighborhood to live in, where to work, and how to access health care, to name a few (Evans and Kantrowitz 2002). Thus, low income individuals are more likely to live and work in hazardous and polluted environments than those with higher SES (Kruize et al 2014). Additionally, due to the racialized history of the United States that goes beyond the scope of this literature review, socioeconomic status and race/ethnicity in the U.S. are inexorably linked (Brulle and Pellow 2006; Cushing et al. 2015; Downey 1998). Many racial minority populations are characterized by low socioeconomic status and as such, have fewer resources and power to combat environmental hazards and associated health risks (Downey 2005; Prochaska et al. 2014; Ringquist 2005).

Examining the relationship between race, occupational exposure to EDCs, and gestational weight gain will help us better understand the mechanisms that lead to unequal chemical obesogenic exposure and obesity prevalence. Because research has been so focused on establishing strong correlations between obesogenic exposure and weight gain, the social conditions which produce inequality in exposure have largely been ignored. This study aims to: 1) expand chemical obesogenic literature by assessing the relationship between occupational exposure to chemically based endocrine disruptors and weigh gain and 2) examine racial disparities in occupational exposure among mothers.

Methods

Data

Utah Population Database

Data for this study were obtained from the Utah Population Database (UPDB). The UPDB contains high-quality socio-demographic, medical, family, and vital records for the Utah population over a span of 100 years. The UPDB is an extremely rich data source that contains information on over 8 million individuals and much of that data can be linked to multi-generational pedigrees. Although the data is Utah specific and perhaps less generalizable, the temporal and data-linked nature of the UPDB allowed for a large sample size to be drawn for analyses. The UPDB is based on linked vital records into multigenerational pedigrees, medical records from the University of Utah Hospitals and Clinics (UUHC), statewide hospitalization records provided by the Utah Department of Health (UDOH), and height, weight, and residence information provided by the Department of Motor Vehicles (DMV). Medical, hospitalization, and DMV records are available from 1995-present. This study uses data specifically drawn from state-wide birth certificates and has been approved by the University of Utah's Resource for Genetic and Epidemiologic Research and its Institutional Review Board. Additionally, to guarantee confidentiality, UPDB staff did all data linkage and returned a deidentified data set to the author for analysis

Job-Exposure Matrix (JEM) Database

Occupational exposure to endocrine disrupting chemicals was determined by a job-exposure matrix (JEM) and based on the self-reported industry and occupation data collected from birth certificate data from UPDB. In 2002, Van Tongeren et al. published a database to assess the likelihood of exposure to EDCs based on occupation and industry type. This original JEM database was constructed by three occupational hygienists who separately assigned exposure scores to each occupation. Job titles were specifically linked to three endocrine disruptor exposure probability scores: unlikely, possible or probable. 348 job titles were linked to exposure and seven broad categories of EDCs were identified: pesticides, phthalates, polychlorinated compounds, alkyl phenolic compounds, heavy metals, bi-phenolic compounds, and a miscellaneous substrate group (methodology further detailed in Van Tongeren et al. 2002). Building off Van Tongeren's (2002) JEM and using similar methodology to expand the database, scholars have developed era-specific (1997-1999; 2000-2002) and solvent specific job-exposure databases to assess the probability of occupational exposures to EDCs; methods outlined in detail elsewhere (Brouwer et al. 2009; Costas et al. 2015; Desrosiers et al. 2015; Snijder et al. 2011; Snijder et al. 2012). The Appendix lists occupations with possible or probable occupational exposures.

The most up-to-date EDC job-exposure matrix (Brouwer 2009) used in this study codes industry and occupation according to the 2000 Standard Occupations Classification (SOC) system. Using coding schemes from The National Institute for Occupational Safety and Health's (NIOSH) Industry and Occupation Computerized Coding System (NIOCCS), occupational data from UPDB was coded using the 2000 SOC guidelines. UPDB data was joined with the job-exposure matrix, producing a dataset with health, demographic, and occupational exposure measures.

Sample

The UPDB data used for this study includes women who gave birth to their first child between 2004 and 2008 in Salt Lake County, Utah. Of the 30,731 women originally captured in the UPDB dataset, 534 were missing pre-pregnancy weight or post-pregnancy weight, from which the dependent variable gestational weight gain is obtained, and were eliminated from the sample. Women who gave birth to twins (n= 526) and triplets (n=19) were also excluded because multiple births are found to affect gestational weight gain (Bodnar et al. 2014). Characteristics of the father collected from birth certificate data were largely complete; however, 3,429 birth certificates did not have father's information listed and missing data was coded as unknown for each covariate. List wise deletion on relevant covariates for mothers and fathers restricted the sample to its final size. The final analytic sample is comprised of 29,652 observations, which is approximately 93 percent of the original sample size.

Measures

Dependent Variables

Gestational Weight Gain (GWG) was calculated as delivery weight minus pre-pregnancy weight. The Institute of Medicine (IOM, 2009) has put forth GWG recommendations based on pre-pregnancy body mass index (BMI) ranges for underweight, normal weight, overweight, and obese women. Based on pre-pregnancy BMI categories, weight gained during pregnancy is classified as inadequate, appropriate, or excessive (IOM 2009). Consistent with prior research, pre-pregnancy BMI was determined from self-reported height and weight and used to create one gestational weight gain variable with three categories: “inadequate”, “appropriate”, or “excessive” (Headen et al. 2015). Inadequate weight gain refers to below-guideline GWG, appropriate is within-guideline GWG, and excessive is above-guideline GWG. Women from the appropriate GWG group are used as the referent group in the analyses.

Key Independent Variable

Exposure Probability was operationalized as occupational exposure to endocrine disruptors and was measured using 4 dummy variables based on employment status (worked versus unemployed) and probability of exposure (unlikely, probable, and possible). The dummy variables are labeled as: Works: Unlikely (0), Works: Possible (1), Works: Probable (2), and No Occupation (9). Missing occupational data was gleaned from self-reported industry, but if both industry and occupation were left blank, the observation was coded as “No Occupation” (mothers n= 985, father n= 248).
Among women in the sample, the “No Occupation” category also includes stay-at-home mothers (n=4,579) and students (n=2,677). For men, “No Occupation” was also comprised of homemakers (n= 546) and students (n=1,436). For this study, an individual was considered exposed if their job was classified as exposed to EDC solvents (i.e., exposure probability 1 or 2); they were considered unexposed if their job was classified as unexposed to EDC solvents (i.e., exposure probability 0). Unlikely exposure probability was used as the reference category in analyses.

Covariates

Mother's Health Characteristics include variables that have known associations with gestational weight gain. Smoking tobacco during pregnancy was measured continuously as the number of cigarettes smoked (average per day) throughout the entire pregnancy (Haugen et al. 2014). The effects of smoking on GWG can differ depending on the stage of pregnancy, but due to collinearity among trimester specific smoking measures, the total average was used. Alcohol consumption (Gaillard et al. 2013) during pregnancy is a continuous variable assessing the average number of drinks per day consumed during pregnancy. Trimester specific measures of alcohol consumption were too highly correlated to model. Risk factors for pre-existing and gestational diabetes were accounted for using dummy coded variables (Chakkalakal et al. 2015). Finally, mother's (and father's) age at time of birth was calculated by subtracting the parents' year of birth from the year of the baby's birth.

Race and Ethnicity are binary dummy coded variables comparing Whites, Hispanics, Blacks, Asians, American Indian/Alaskan Natives, Native Hawaiian/Pacific Islander, multiple races, other races not listed on vital records, and unknown (including not reported; coded as 1= identified with racial group, 0= did not identify with racial group). Although the counts for some of these categories (e.g. black) were small and could be combined with each other for more meaningful comparisons, analyzing each racial category separately is conceptually important. White is the group used for reference in the logistic models.

Level of Education measures the highest year of schooling completed by mothers and fathers. The self-reported number of years of education, which were collected from

UPDB birth certificate data, were collapsed into 3 dummy variables: no high school degree, high school degree or equivalent, attended college. The latter category includes respondents who both attended, but did not graduate, from college and college graduates. There were 283 missing education records for mothers and 2,330 for fathers and a multiple imputation regression imputation model was used to compute missing values for level of education for mothers and fathers (Schafer and Schenker 2000). Specifically, values of missing data were estimated by fitting a regression model for each missing observation based on known values using the following equation:

$$Y_J = \beta_0 + \beta_1 Y_1 + \beta_2 Y_2 + \ldots \beta_{(J-1)} Y_{(J-1)}$$

During multiple imputation, subsequent regression models for each missing variable, Y_J, are constructed with non-missing observations (β_0, β_1 …. $\beta_{(J-1)}$) and replaced by the predictive distribution of the missing data ($\beta_{*0} + \beta_{*1}y_1 + \beta_{*2}y_2 + \beta_{*(J-1)y(j-1)} + z_i\sigma_{*j}$) as detailed by Rubin (1987).

Analyses

Sample distributions and bivariate associations were assessed statistically to determine normality and linear associations. Multinomial logistic regression was employed to test the main effect occupational exposure to endocrine disruptors has on BMI-adjusted gestational weight gain when accounting for potential cofounders and mediators. Multinomial models are advantageous in comparing different outcomes (e.g. gestational weight gain) in the same model. The first model examines the main effect of exposure on BMI adjusted gestational weight gain when controlling for mothers’ demographic and health characteristics only. Model 2 and Model 3 examine how race/ethnicity and levels of education influence the main effects. Fathers' data are added as controls in subsequent models, presented in Table 4.3.

The second study aim is to ascertain if mothers from racial and ethnic minority groups experience a higher probability of occupational exposure to endocrine disruptors when compared to Whites. Logistic regression modeling was used to examine this question. All analyses were conducted using Stata Software, version 13 (StataCrop. 2013. *Stata Statistical Software: release 13. College Station, TX: StataCorp L*P).

Results

Descriptive statistics are presented in Table 4.1. Regarding the dependent variable, gestational weight gain among women in the sample ranged from -32 to 130 pounds, with the average mother gaining 34 pounds. When adjusting gestational weight gain by pre-pregnancy BMI, approximately 13 percent of the sample did not gain adequate amount of weight (n=3,839), 35 percent gained appropriate weight (n=10,314), and 52 percent gained excessive weight during pregnancy. This distribution mirrors previous findings on gestational weight gain levels (Hickey 2000; Kapadia et al. 2015).

In regards to demographic data, the average age of mothers was 25 years old and approximately 28 for fathers. Whites comprise most of the sample as 75 percent of mothers and 73 percent of fathers were white. Approximate 18 percent of mothers and 17 percent of fathers identified as Hispanic. The remaining racial/ethnic categories each made-up less than 3 percent of the sample. The most common category of education for mothers (n=16,039) and fathers (n= 15,048) was some college. Approximately 31 percent of parents had a high school degree and 14 percent of mothers and 11 percent of fathers had less than a high school degree. Finally, parents were more likely to be employed than unemployed, although 27 percent of women were classified as non-working. Sixty-three percent of women who worked were employed in occupations with “unlikely” exposure to EDCs. Almost 9 percent had job titles associated with “possible” exposure and less than 1 percent worked in occupations with “probable” exposure. Men were more likely to work in occupations with “probable” (3%) and “possible (31%) exposure than women. Most men, however, held an occupation where exposure to endocrine disruptors was unlikely (58%).

Table 4.2 presents multinomial regression models that account for mothers’ covariates only. In Model 1, possible and probably exposure probabilities were not significantly associated with any BMI-adjusted gestational weight gain category. The relative risk ratio of gaining above-guideline GWG compared to those gaining within-guideline GWG was statistically higher for women with no listed occupation (RRR= 1.262, $p<0.001$). Maternal age was significant and negatively associated with inadequate and excessive GWG in Model 1 and Model 2, suggesting a mediating effect. These patterns largely held up when racial/ethnic variables were introduced in Model 2. No occupation remained the only significant exposure predictor and it was positively associated with excessive GWG (RRR=1.176, $p<0.01$). Consistent with the literature, black and Hispanic mothers had higher risks of gaining above-guideline weight during pregnancy, while Native Hawaiian/Pacific Islanders, American Indians/Alaskan Natives, and Asian mothers were more likely to gain below the guidelines relative to women in other GWG categories (Headen et al. 2015). Asian and Hispanic mothers were also at a lower risk for gaining excessive weight relative to women from other racial groups. These significant racial disparities persisted when controlling for level of education. I found the relative risks of gaining an inadequate amount of weight during pregnancy was higher for women who attended college and non-significant for women who had less that a high school degree; the latter of which also had a higher, significant risk of gaining excessive weight (RRR= 1.251, $p<0.001$) compared to other women in the sample.

Controlling for paternal variables yielded very similar results (Table 4.3) and improved model fit. Occupational exposure probabilities were non-significant in the fully saturated model. The mediating effect of maternal age became non-significant in the final model, while smoking continued to have a strong mediating effect for excessive GWG. In regards to race, results were similar to the previous model with a few notable differences. First, the significant effects found for Native Hawaiian/Pacific Islander mothers became insignificant when accounting for paternal variables. However, the risk of inadequate GWG increased if the father's race was Native Hawaiian or Pacific Islander. These changes could be a result of small sample size or indicative of complex socio-cultural determinants. Significant trends in educational effects on GWG differ slightly between parents; having a partner who attended college is associated with lower odds of gaining an inadequate amount of weight during pregnancy. To summarize findings thus far, no significant association was found between occupational exposure probability and gestational weight gain. Racial disparities in GWG exist among women in the sample: black and Hispanic mothers have higher risk for below-guideline GWG and; Asian and Native Hawaiian/Pacific Islander mothers have lower risk of above-guideline or excessive GWG relative to other racial groups and controlling for all covariates.

To assess if racial differences in occupational exposure exist among mothers, logistic regression modeling was conducted and odds ratios are displayed in Table 4.4. Hispanic mothers had significant, positive correlations with working in an occupation with possible or probable exposure to endocrine disruptors compared to other racial/ethnic groups. The significance of the possible exposure variable became insignificant when accounting for levels of education, but the association between probable exposure (OR= 1.213, $p<0.001$) remained unchanged. Being Asian or Native Hawaiian/Pacific Islander were also significant racial predictors of exposure. Specifically, Asian mothers were more likely (OR= 1.798, $p<0.001$) to have a job where exposure to endocrine disruptors was possible when compared to non-Asian women and Native Hawaiian and Pacific Islanders were found to have decreased odds of working in occupations with possible exposure (OR= 0.520, $p<0.001$), which is not what we'd expect to see given occupational disparities literature (percentages calculated from standardize coefficients not shown, but available upon request). Again, these findings may reflect the very small sample size of this population within the sample. These findings mirror what other occupation health disparities researchers have found; Hispanic and Asian women disproportionally work in the domestic, agricultural, and beauty sectors of the labor market (Murray 2003). These occupations are associated with higher probabilities of exposure to endocrine disruptors found in cleaning supplies, pesticide/herbicides, and beauty products.

Discussion and Conclusion

The results of this retrospective study of pregnant women in Utah suggest three important messages. First, the probability of occupational exposure to endocrine disruptors did not significantly affect gestational weight gain. This finding does not support the obesogenic hypothesis which suggests that exposure to environmental toxins, endocrine disruptors specifically, increases obesity risk (Grun and Blumberg 2009). This finding is consistent with some obesogenic research which has found little to no effect of EDC exposure on childhood obesity risk (Buckley et al. 2016; Xue et al. 2015). Although research examining occupational exposure during pregnancy using similar methodologies has found associations between exposure and adverse health outcomes of the infant, such as restricted fetal growth, this is potentially the first study to focus on health outcomes of the mother (Desrosiers et al. 2015). This fact should be taken into account when interpreting these findings, as more research is needed in order to draw conclusions about the relationship between obesogenic exposure and gestational weight gain.

Second, certain racial and ethnic identities were predictors of gestational weight gain. Similar to previous research, black and Hispanic mothers were at a higher risk for gaining less weight than recommended based on their pre-pregnancy BMI (Rasmussen et al. 2009). Scholars suggest this trend can partially be explained by the connection between minority status and socioeconomic position. Black and Hispanic women are disproportionally poor and may have less access to prenatal care (Rosal et al. 2016) and nutritional interventions (Rasmussen et al. 2009); both risk factors associated with GWG. Additionally, health risks found frequently in Blacks and Hispanic populations, namely gestational diabetes, have been shown to restrict gestational weigh gain (Walker, Hoke, and Brown 2009). Asian and Hispanic women were found to have lower risks of above-guideline GWG relative to other racial/ethnic groups. Again, these finding are consistent with previous research. In detailed studies examining associations by country of origin, Asian (Cheng et al. 2015) and Hispanic (Sangi-Haghpeykar 2014) sub-groups are still less likely to gain excessive weight during pregnancy compared to Whites. One result to note is the positive association found between smoking and excessive GWG. A mountain of literature has identified smoking as a correlate of being under weight, largely due to appetite suppression induced by tobacco (Jessen et al. 2005). New research is examining this association among pregnant women, and thus far, findings suggest that mothers who cease smoking once pregnant, tend to gain more weight and faster compared to mothers who don't smoke; perhaps because their appetite is no longer suppressed (Hulman et al. 2016).

Third, when examining racial disparities to occupational exposure probabilities, Asian and Hispanic women were more likely to work in sectors with higher probability of endocrine disrupting chemical exposure. This finding provides support for environmental inequality theories, which suggest that racial/ethnic minorities experience more exposure to environmental hazards and risks (Brulle and Pellow 2006). Within the labor market specifically, Asian and Hispanic populations may disproportionally experience exposure risks due to the nature of work they self-select or are funneled into (e.g. agriculture, cosmetology, domestic cleaning). Although this analysis strengthens the occupational disparities literature by further documenting racially unequal exposure probabilities in the workforce, more research is needed to fully understand these findings.

There are several limitations of this study. First, the exposure estimate has some weaknesses. Relying on self-reported occupational data runs the risk of exposure misclassification. Exposure measures also do not account for additional exposure parameters, such as frequency, dosage, and route of exposure. Moreover, using probabilities of exposure did not allow for chemical-specific analyses. Based on results from previous studies that use job-exposure matrixes, these limitations likely produced more conservative estimations (Brouwers et al. 2009).

Another limitation lies in the dependent variable. The key outcome was BMI-adjusted gestational weight gain. Pre-pregnancy weight was self-reported, which could bias the results. The type of work (e.g. part-time, full-time) and how long women worked into their pregnancy was also not accounted for. As such, cumulative effects from occupational and non-occupational exposures could not be measured; which is extremely important when assessing direct associations between hazardous environmental exposures and health outcomes (Payne-Sturges Gee 2006). Ideally, objective and reliable measure of BMI and gestational weight gain would be used and exposure estimates would account for frequency, dosage and routes of exposure overtime (Sexton and Linder 2011).

Despite these limitations, this research is significant in two regards. First, drawing on sociological methods, this is a population-based study and is likely the first of its kind to look at obesogenic associations using a large sample size. Women in the sample also represented a wide range of occupations. Therefore, results from this study are potentially more generalizable than pervious studies with smaller sample sizes and occupation specific analyses (Janesick and Blumberg 2016). This is important because the diverse results found amongst previous obesogenic research may be due to differences in occupational exposure profiles (Desrosiers et al. 2015).

Second, this work contributes to multiple academic fields by documenting associations between: 1) endocrine disrupting chemical exposure and gestational weight gain; 2) racial disparities in gestational weight gain, and 3) occupational exposure differentials by race. Scholars in obesogenics, environmental inequality, and occupational health disparities can build off this work to better understand the socio-environmental mechanisms that place certain populations at a greater risk of hazardous exposure and how such exposure is related to health outcomes like obesity. Specifically, future research should focus on uncovering links between pollution exposure and obesity risk at the population level using longitudinal, prospective data and comprehensive exposure measures (Morello-Frosh et al. 2011). I also advocate that racial differences in exposure levels within the work force should be given more priority within the environmental inequality literature.

In conclusion, this study found non-significant associations between gestational weight gain and occupational exposure to endocrine disruptors among women in Utah who gave birth to their first child between 2004 and 2008. This finding does not support the obesogenic hypothesis, which theorizes that obesity etiology may be related to exposures of environmental toxins. This study also assessed racial disparities in gestational weigh gain and occupational exposure. Race was found to have a mediating effect on both GWG and environmental exposures. Scholars in obesogenics, environmental inequality, and occupational health disparities can build off this work to better understand the socio-environmental mechanisms that place certain populations at a greater risk of hazardous exposure and how such exposure is related to health outcomes like obesity.

Table 4.1. Descriptive Statistics for Mothers and Fathers Used in analyses

	N	Percent	Mean	SD	Minimum	Maximum
Characteristics of Mothers (n= 29,652)						
Occupational Exposure Probability to EDCs						
Works: Unlikely EDC Exposure	18,791	63.63	0.633	0.481	0	1
Works: Possible EDC Exposure	2,484	8.38	0.083	0.277	0	1
Works: Probable EDC Exposure	136	0.46	0.004	0.067	0	1
No Occupation	8,241	27.79	0.277	0.447	0	1
Age	29,652	100	25.21	5.13	13	51
Race and Ethnicity						
White	22,497	75.87	0.759	0.186	0	1
Black	346	1.17	0.011	0.107	0	1
Hispanic	5,372	18.12	0.181	0.385	0	1
Asian	520	1.75	0.017	0.131	0	1
Native Hawaiian/Pacific Islander	552	1.86	0.018	0.135	0	1
American Indian/Alaskan Native	244	0.82	0.008	0.090	0	1
Other Race	36	0.12	0.001	0.034	0	1
Unknown	85	0.29	0.002	0.053	0	1
Education Level						
No High school degree	4,312	14.54	0.145	0.352	0	1
High school degree	9,301	31.34	0.313	0.467	0	1
Attended college	16,039	54.09	0.540	0.498	0	1
			0.998	1.317		
Health Characteristics						
Pre-pregnancy BMI	29,652	100	24.42	5.21	18	59
Underweight (BMI<18.5)	768	2.59	0.025	0.158	0	1
Normal (BMI 18.5≤BMI>25)	18,950	63.91	0.630	0.482	0	1
Overweight (25≤BMI>30)	6,042	20.38	0.203	0.402	0	1
Obese (BMI≥30)	3,892	13.13	0.130	0.331	0	1
Gestational weight gain (GWG)	29,652	100	34.63	13.49	-32	130
BMI adjusted inadequate GWG	3,839	12.95	0.13	0.34	-32	27
BMI adjusted approriate GWG	10,314	34.78	0.35	0.48	11	40
BMI adjusted excessive GWG	15,499	52.27	0.52	0.50	21	130
Characteristics of Fathers (n= 26,223)						
Occupational Exposure Probability to EDCs						
Works: Unlikely	15,130	57.56	0.151	0.499	0	1
Works: Possible	8,101	30.89	0.308	0.462	0	1
Works: Probable	761	2.90	0.029	0.167	0	1
No Occupation	2,231	8.50	0.085	0.278	0	1
Age	26,223	100	27.97	5.73	14	81
Race and Ethnicity						
White	19,464	73.76	0.736	0.243	0	1
Black	416	1.59	0.015	0.124	0	1
Hispanic	4,337	16.54	0.165	0.371	0	1
Asian	479	1.83	0.018	0.133	0	1
Native Hawaiian/Pacific Islander	775	2.96	0.029	0.169	0	1
American Indian/Alaskan Native	179	0.68	0.006	0.082	0	1
Other Race	37	0.14	0.001	0.037	0	1
Unknown	536	2.04	0.020	0.141	0	1
Education Level						
No High school degree	3,014	11.49	0.114	0.318	0	1
High school degree	8,161	31.12	0.311	0.462	0	1
Attended college	15,048	57.38	0.573	0.494	0	1

Note:
BMI, Body mass index: was calculated as weight (kg)/height(m)2

Table 4.2. Relative risk ratios of multinomial models of BMI-adjusted gestational weight gain and sociodemographic for women in Utah, 2004-2008

	Pre-pregnancy BMI Adjusted Gestational Weight Gain Categories (IMO)					
	Model 1		Model 2		Model 3	
	Inadequate	Excessive	Inadequate	Excessive	Inadequate	Excessive
Occupational Exposure Probability to EDCs						
Worked: Possible EDC Exposure	1.044	1.116	1.063	1.085	0.994	1.011
	(0.952-1.145)	(0.973-1.280)	(0.968-1.166)	(0.946-1.244)	(0.904-1.092)	(0.880-1.162)
Worked: Probable EDC Exposure	0.878	1.092	0.889	1.024	0.840	0.935
	(0.608-1.269)	(0.643-1.854)	(0.615-1.285)	(0.602-1.742)	(0.580-1.217)	(0.549-1.593)
No Occupation	1.022	1.262***	1.046	1.176**	1.005	1.082
	(0.962-1.085)	(1.156-1.378)	(0.982-1.115)	(1.072-1.290)	(0.941-1.073)	(0.983-1.192)
Age	0.984***	0.987***	0.985***	0.989**	0.996	1.002
	(0.979-0.989)	(0.979-995)	(0.980-0.990)	(0.981-0.997)	(0.990-1.001)	(0.993-1.010)
Race and Ethnicity						
Black			0.980	1.709***	0.945	1.633**
			(0.768-1.251)	(1.260-2.317)	(0.739-1.208)	(1.204-2.216)
Hispanic			0.924*	1.247***	0.875***	1.129*
			(0.861-0.992)	(1.130-1.376)	(0.812-0.941)	(1.019-1.249)
Asian			0.582***	1.092	0.594***	1.118
			(0.479-0.706)	(0.850-1.405)	(0.489-0.722)	(0.870-1.438)
Native Hawaiian/Pacific Islander			1.718***	1.166	1.635***	1.134
			(1.406-2.101)	(0.852-1.594)	(1.338-1.998)	(0.829-1.550)
American Indian/Alaskan Native			1.529**	1.018	1.446**	0.955
			(1.140-2.051)	(0.639-1.621)	(1.077-1.942)	(0.599-1.522)
Other Race			2.057	1.070	2.081	1.102
			(0.928-4.559)	(0.283-4.039)	(0.944-4.585)	(0.290-4.177)
Unknown			1.294	1.184	1.317	1.245
			(0.792-2.112)	(0.605-2.314)	(0.808-2.147)	(0.641-2.417)
Education Level						
No High school degree					0.960	1.251***
					(0.878-1.049)	(1.108-1.412)
Attended college					0.737***	0.792***
					(0.693-0.784)	(0.721-0.870)

Notes:
N= 29,652, 95% confidence intervals in parentheses
The reference groups for gestational weight gain, EDC occupational exposure, race/ethnicity, and education level are: appropriate (recommended) GWG; worked: unlikely exposure; white and; high school degree, respectively.
*p<0.05, ** p<0.01, *** p<0.001

Table 4.3. Relative risk ratios of multinomial models of BMI-adjusted gestational weight gain and sociodemographic for mothers and fathers in Utah, 2004-2008

	Pre-pregnancy BMI Adjusted Gestational Weight Gain Categories (IMO)			
	Model 1		**Model 2**	
	Inadequate	**Excessive**	**Inadequate**	**Excessive**
Mother's Occupational Exposure Probability to EDCs				
Works: Possible EDC Exposure	1.062	1.082	0.992	1.009
	(0.968-1.166)	(0.943-1.241)	(0.903-1.090)	(0.887-1.159)
Works: Probable EDC Exposure	0.892	1.022	0.838	0.936
	(0.616-1.292)	(0.601-1.739)	(0.578-1.219)	(0.549-1.595)
No Occupation	1.042	1.174**	1.004	1.097
	(0.977-1.111)	(1.070-1.289)	(0.578-1.219)	(0.982-1.192)
Mother's Age	0.986***	0.989***	0.997	1.001
	(0.981-0.991)	(0.981-997)	(0.991-1.002)	(0.993-1.011)
Mother's Race and Ethnicity				
Black	0.952	1.620**	0.934	1.559**
	(0.738-1.232)	(0.117-2.231)	(0.723-1.212)	(1.132-2.150)
Hispanic	0.893**	1.212***	0.866***	1.115
	(0.823-0.968)	(1.083-1.356)	(0.796-0.940)	(0.993-1.250)
Asian	0.545***	1.298	0.558***	1.305
	(0.405-0.731)	(0.900-1.871)	(0.414-0.750)	(0.906-1.877)
Native Hawaiian/Pacific Islander	1.177	1.061	1.144	1.038
	(0.920-1.512)	(0.820-1.862)	(0.896-1.468)	(0.719-1.503)
American Indian/Alaskan Native	1.457**	0.992	1.400*	0.943
	(1.081-1.960)	(0.616-1.594)	(1.039-1.885)	(0.586-1.515)
Other Race	1.779	1.023	1.832	1.057
	(0.799-3.980)	(0.271-3.859)	(0.827-4.059)	(0.279-4.012)
Unknown	1.305	0.135	1.339	1.057
	(0.793-2.146)	(0.575-2.240)	(0.818-2.203)	(0.611-2.350)
Father's Race and Ethnicity				
Black	1.108	1.177	1.074	1.162
	(0.879-1.393)	(0.859-1.611)	(0.850-1.352)	(0.848-1.592)
Hispanic	1.087	1.058	1.012	0.996
	(0.994-1.180)	(0.935-1.194)	(0.920-1.104)	(0.873-1.134)
Asian	1.105	0.779	1.107	0.796
	(0.811-1.506)	(0.516-1.178)	(0.811-1.513)	(0.528-1.200)
Native Hawaiian/Pacific Islander	2.079***	1.236	1.945***	1.224
	(1.579-2.726)	(0.820-1.862)	(1.478-2.549)	(0.813-1.840)
American Indian/Alaskan Native	1.248	1.090	1.187	1.073
	(0.891-1.748)	(0.648-1.832)	(0.847-1.665)	(0.637-1.806)
Other Race	0.551	0.620	0.566	0.656
	(0.278-1.089)	(0.209-1.833)	(0.283-1.127)	(0.220-1.951)
Unknown	0.853	1.405	0.845	1.398
	(0.502-1.461)	(0.726-2.729)	(0.496-1.458)	(0.722-2.719)
Mother's Education Level				
No High school degree			0.973	1.233***
			(0.893-1.071)	(1.091-1.399)
Attended college			0.778**	0.801***
			(0.730-0.833)	(0.933-1.249)
Father's Education Level				
No High school degree			0.980	1.081
			(0.880-1.084)	(0.933-1.249)
Attended college			0.877***	0.991
			(0.822-0.938)	(0.898-1.095)

Notes:
N= 29,652, 95% confidence intervals in parentheses
The reference groups for gestational weight gain, EDC occupational exposure, race/ethnicity, and education level are: appropriate (recommended) GWG; worked: unlikely exposure; white, and high school degree, respectively.
*p<0.05, ** p<0.01, *** p<0.001

Table 4.4. Odds ratios of logistic regression models for occupational exposure to endocrine disrupting chemicals for mother's in Utah by race, 2004-2008

	Probability of Occupational Exposure to EDCs			
	Model 1 Possible	Model 2 Probable	Model 3 Possible	Model 4 Probable
Mother's Race & Ethnicity				
Black	1.061	1.135	0.895	1.000
	(0.728-1.548)	(0.945-2.125)	(0.612-1.309)	(0.894-1.998)
Hispanic	1.207***	2.920***	0.989	1.213***
	(1.089-1.338)	(1.995-4.047)	(0.885-1.10)	(1.525-3.378)
Asian	1.512**	1.404	1.798***	1.512
	(1.152-1.983)	(0.274-4.573)	(1.359-2.355)	(0.292-4.881)
Native Hawaiian/Pacific Islander	0.652*	1.463	0.520***	1.389
	(0.450-0.945)	(1.051-6.444)	(0.358-0.756)	(0.958-5.934)
American Indian/Native Alaskan	1.216	1.008	0.955	0.861
	(0.796-1.857)	(0.140-7.262)	(0.623-1.464)	(0.122-6.370)
Other	1.028	1.000	1.006	1.000
	(0.315-3.358)	(0.892-1.035)	(0.305-3.317)	(0.846-2.012)
Unknown	0.773	1.056	0.754	0.984
	(0.318-1.689)	(1.001-2.568)	(0.326-1.744)	(0.921-1.587)
Mother's Level of Education				
No High School Degree			0.709***	1.346
			(0.626-0.804)	(0.868-2.157)
Attended College			0.389***	0.695
			(0.354-0.427)	(0.495-1.112)

Notes:
N= 29,652, 95% confidence intervals in parentheses
The reference groups for race/ethnicity and education level are White and high school degree, respectively.

*p<0.05, ** p<0.01, *** p<0.001

CHAPTER 5

CONCLUSION

This dissertation investigated socioeconomic and racial/ethnic differentials in chemical obesogenic exposure and associations between environmental exposure and obesity risk. Employing theoretical frameworks from environmental inequality and obesogenic research, this study provided a more nuanced understanding of mechanisms involved in environmental disparity and obesity etiology. Findings suggest that environmental inequality does not manifest equally across all pollution types and populations. These results support contemporary environmental inequality literature illustrating that socioeconomic status and racial/ethnic position doesn't necessarily dictate the amount of hazardous environmental exposure certain groups experience (Anderton et al. 1994; Bryant and Mohai 1992; Ringquist 2005). This isn't to say, however, that differentials in exposure do not vary between groups with different social class and racial/ethnic status. Rather, current research, like this study, is focused on uncovering how specific types of pollution and pollution sources vary among marginalized groups in society.

Further discovering patterns of environmental inequality can help inform our understanding of environmental health issues, particularly obesity. If toxic airborne environmental exposure does play a role in the development of obesity, as the obesogenic hypothesis espouses, we need to find direct linkages between obesogenic chemicals and obesity prevalence. A sparse amount of literature has connected a certain class of toxins, endocrine disruptors (EDCs), to increased obesity risk in some populations (Heindel et al. 2015; Zoeller et al. 2012). Other research, however, has found little evidence of the association between EDCs and excessive weight. The results presented in this study mirror previous research and yielded mixed results. Inconsistent results likely reflect the complexity of the relationship between obesity risk and EDC exposure. Perhaps not all populations are equally susceptible to obesogenic mechanisms. Or most likely, methodological restraints have not allowed us to fully capture environmental exposure and the development of obesity overtime, which would permit us to assess cumulative impacts of exposure on body weight.

The results of this study were limited by two important factors. First, the cross-sectional nature of this study does not account for the effects cumulative risks of environmental exposure play in potentially creating obesity. There is empirical evidence that interactive effects from exposure to a combination of environmental stressors can contribute to adverse health effects (Sexton and Linder 2011). Cross-sectional design methods are not appropriate when trying to assess the environmental exposures overtime.

Second, although this study relied on pollution-source specific measures and occupational exposure metrics, the results are bound by the environmental exposure measures available. Emissions data, especially in the U.S., is partially reliant on self-reported data from industry sources. Additionally, when assessing occupational exposures, much of the data is also reliant on self-reported job titles and duties. The nature of available data is likely bias, as true exposure amounts are not accounted for.

This makes it very difficult for researchers to draw strong associations or potentially glean causality between exposure and health when using such data. Ideally, population-based studies using biomarkers that monitor direct exposure sources would be used to capture environmental health inequalities.

In spite of these limitations, this study offers three substantial contributions to the current literature on environmental exposures and obesity by (1) investigating the effects of endocrine disrupting chemical exposure on obesity prevalence using populations-based estimates that are more generalizable then many previous studies, (2) assessing the environmental exposure-obesity association in highly susceptible populations and, (3) identifying social risks associated with increased exposure to endocrine disruptors. Scholars in obesogenics, environmental inequality, and environmental health inequality can build off this work to better understand the socio-environmental mechanisms that place certain populations at a greater risk of hazardous exposure and how such exposure is related to health outcomes like obesity.

Future Directions for Research

Although recent literature on obesogens represents considerable progress in understanding the role toxic chemical exposure plays in obesity, three substantial improvements can be made in future work. First, obesogenic research has largely been conducted 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 largely been absent in exploring how social processes, such as socioeconomic status and race and ethnicity, mediates the effect chemical exposure has on human health (Vafeiadi et al. 2015). Non-chemical obesogens, such as proximity to parks, neighborhood walkability and access to public transportation, have been studied more extensively by social scientists (see Ding and Gebel (2012) and Feng, Glass, Curriero, Stewart, and Schwartz (2010) for comprehensive reviews). As a result, obesogens related to the built environment are better understood by social scholars than chemical obesogens.

Second, multilevel analyses have been noticeably absent in obesogenic and environmental health research. Contemporary environmental exposure studies tend to focus on proximity to polluting areas (e.g. industries and waste sites) or broad level exposure at the county or state level (Mohai and Saha 2007; Ringquist 2005). A few studies have scaled down their analysis to look at hazardous exposure in specific metropolitan areas such as Detroit (Downey 1998) and Atlanta (Zwickl, Ash, and Boyce 2014), but again, this in only capturing one geographic level. Analyzing pollution exposure at multiple levels would allow researchers to identify under what conditions localized, neighborhood pollution exposure is more important than general, broad-scale pollution exposure and vice versa.

Third, studies exploring chemical obesogens have been restricted to cross-sectional methods due to data restraints (Kruize, Droomers, van Kamp, and Ruijsbroek 2014). To my knowledge, not one study has longitudinally examined the association between chemical exposure and obesity rates overtime. Assessing the temporal effects of chemical exposure on weight is crucial in understanding how low-dose, chronic exposure to environmental toxins affects the prevalence of obesity (Hyman 2010).

Investigating the longitudinal association between toxic environmental exposures, socio-demographic changes and obesity is needed to improve our understanding of the complex etiology of obesity.

Finally, in regards to future investigations of environmental justice, research needs to recognize and account for the fact that environmental inequality outcomes vary widely across communities (Downey 2007). Designing studies that examine the shared, interacting effects of community-level characteristics are needed to fully disentangle all the social conditions- not just race/ethnicity and class- that effect the unequal distribution of environmental hazardous (Grant el al. 2010). Few studies, including Chapter 2 of this dissertation, have explored how sociohistorical factors, particularly segregation, work in tandem with other social characteristics to produce environmental risks (Lievanos 2015; Zwickl 2014). However, more research in this vein is needed to establish clear mechanisms of environmental inequality.

Conclusion

The focus of this dissertation was to bridge environmental justice, environmental health inequality, and sociological research on obesogens to investigate the association between environmental exposures and obesity prevalence. This study sought to expand our knowledge of obesogenics by exploring the way in which exposure to a specific class of obesogens, endocrine disruptors, influences obesity risk. This dissertation offers three substantial contributions to the current literature on environmental exposures and obesity by (1) investigating the effects of endocrine disrupting chemical exposure on obesity prevalence using populations-based estimates that are more generalizable then many previous studies, (2) assessing the environmental exposure-obesity association in highly susceptible populations and, (3) identifying social risks associated with increased exposure to endocrine disruptors. This study highlights the importance of incorporating sociohistorical frameworks in the study of environmental inequality. It also attempts to link environmental inequality to health disparities by examining the relationship between environmental exposure and associated health risks. It is my hope that this study encourages other scholars to pursue further research investigating the complex relationship between social characteristics, susceptibility of exposure and associated health outcomes.

APPENDIX

List of occupational titles assigned to a possible or probable exposure to EDCs Costas et al. (2015)

Agricultural and fishing trades n.e.c.
Air transport operatives
Ambulance staff (excluding paramedics)
Animal care occupations n.e.c.
Artists
Assemblers (electrical products)
Assemblers (vehicles and metal goods)
Auto electricians
Bar staff
Beauticians and related occupations
Bookbinders and print finishers
Bus and coach drivers
Car park attendants
Carpenters and joiners
Chemists
Cleaners, domestics
Conservation and environmental protection officers
Countryside and park rangers
Dental practitioners
Driving instructors
Electrical/electronics engineers n.e.c.
Electrical/electronics technicians
Electricians, electrical fitters
Farm managers
Fishing and agriculture related occupations n.e.c.
Floorers and wall tilers
Fork-lift truck drivers
Furniture makers, other craft woodworkers
Gardeners and groundsmen/groundswomen
Glass and ceramics process operatives
Goldsmiths, silversmiths, precious stone workers
Hairdressers, barbers
Horticultural trades
Industrial cleaning process occupations
Laboratory technicians
Launderers, dry cleaners, pressers
Leather and related trades
Medical and dental technicians
Metal production and maintenance fitters
Mobile machine drivers and operatives n.e.c.
Motor mechanics, auto engineers
Molders, core makers, die casters
Musical instrument makers and tuners
Natural environment and conservation managers
Officers in armed forces
Painters and decorators
Paper and wood machine operatives
Paramedics
Pest control officers
Photographers and audio-visual equipment operators
Pipe fitters
Plastics process operatives
Plumbers, heating and ventilating engineers
Police officers (sergeant and below)
Postal workers, mail sorters, messengers
Precision instrument makers and repairers
Printing machine minders and assistants
Road construction operatives
Roofers, roof tilers and slaters
Routine laboratory testers
Rubber process operatives
School crossing patrol attendants
Screen printers
Sheet metal workers
Taxi, cab drivers and chauffeurs
Textiles, garments and related trades n.e.c.
Transport operatives n.e.c.
Tire, exhaust and windscreen fitters
Upholsterers
Van drivers
Veterinarians
Waiters, waitresses

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