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Correlations Between Childhood Obesity and Obesogenic Environmental Variables Within Durham County, North Carolina

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MPH, Walden University, 2006

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BA, Pepperdine University, 1981

Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

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Abstract

Geographic information systems were used to map obesogenic conditions by zip code tabulation areas in Durham County, North Carolina and evaluated associations between those conditions and the understudied area of early childhood obesity. Of the 16,478 two - four year old children who received supplemental nutrition services for women, infants and children in North Carolina in 2010 also had BMI $\geq 95^{th}$ percentile for obesity; 433 (19.7%) of those children resided in Durham County, North Carolina and were used as the criterion variable in this study (N=433). The study's research questions examined conditions of the neighborhood that impeded the weight status of young children. The theoretical framework included the environmental stress, socioecological, multiple exposures –multiple effects, and attachment theories. The 26 variables used in this secondary quantitative study included demographics on socioeconomic and education levels, home occupancy and vacancy rates, age of homes, neighborhood accessibility to amenities and neighborhood incivilities and reported crimes. Histograms and scatterplots showed a low R^2 value too weak for linear regression analysis. The *t* test configured as Grouping Variables with the cut-point of 18.7% and an Alpha of .05 produced statistical significance on 5 of 26 variables. A mean rate of 19.3% yielded statistical significance on 10 of 26 variables. The Levene's Test for Equality of Variances for statistical significance on t-test at the Alpha = 0.05 level met 24 of 26 variables. The results of this study and the use of the innovative geographic information systems could inform policy decisions, environmental interventions and environmental design on obesogenic correlates between the understudied area of early childhood obesity and the built environment.

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Dedication

This dissertation is dedicated to my Lord and Savior Jesus Christ for the many gracious gifts given me. To my husband, Thomas, thank you for your undying love and support and to my sons Devin, Brant, Kyle and Corey thank you for the incredible young men you have become and for keeping me grounded and calling me your "SHE-RO." To know that I have positively influenced your lives is a job well done. I cannot forget "Typical Chaos" (my Dobie) who burned the midnight oil with me many a nights. I challenge each of my sons to soar beyond their wildest dreams!

Luke 12:48…where much is given, much is required

****My joy for the past 8 years, Typical Chaos passed away on June 4, 2014…***

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It is a pleasure to thank my chair Dr. Raymond Thron whose encouragement and support guided me through this iterative scholarly process.

I would also like to thank Dr. Chester Jones who was my favorite professor during my Masters in Public Health courses and who graciously sat as a member on my dissertation committee.

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Chapter 1: Introduction to the Study

The epidemical nature of childhood obesity has caused considerable challenge to the public health community (Risica 2004). Its disease impact in children has crossed genders, all ages, and all ethnicities (Dekelbaum & Williams, 2001; Ogden, Flagon, Carroll, & Johnson, 2002; Risica 2004). Nearly every state in the nation has responded to the public health call to address childhood obesity and mitigate its contributory disease impact (Centers for Disease Control and Prevention [CDC], 2005; U.S. Department of Health, 2000).

The Institute of Medicine [IOM] (2004) Committee on Prevention of Obesity in Children and Youth describes this public health issue as indiscriminately affecting children in the United States in every socioeconomic strata and ethnic group. Obesity also increases the risks for early comorbid diseases in children (IOM, 2004). Its multiple etiologies have complicated the issue since any one contributing factor has not ascribed total causation (American Academy of Pediatrics [AAP], 2003; Institute of Medicine [IOM], 2005; Lavizzo –Mourey, 2007). The Health Resources and Services Administration (HRSA, 2010) stated that childhood obesity is one of the most urgent public health issues of the times. Accordingly, HRSA (2010) awarded $5 million to the National Initiative for Children's Healthcare Quality [NICHQ] to build The Prevention Center for Healthy Weight to provide treatment interventions at the primary care level.

Surveillance surveys from the National Health and Nutrition Examination Survey [NHANE] from 2005 - 2008, issued by the Center for Disease Control and the National Center of Health Statistics (2004), emphasized the importance of examining the body mass index (BMI) levels of all children since the obesity rates for children have disproportionately risen over the

past 2 decades. Likewise, epidemiological studies have shown that young children exhibit signs of adiposity relative to excess weight (AAP, 2003, p. 424). The 2009-2010 NHNES stated that 12.5 million children and adolescents across the nation had BMI levels greater than the 95th percentile and were considered obese. BMI is a common gauge for obesity in children, calculated by using the standard formula, BMI = kg/m^2, or weight in kilograms divided by the squared height in meters (CDC, 2013).

Data from the Pediatric Nutrition Surveillance System (PedNSS, 2009) indicated that 1.23 million low-income children between the ages of 2- and 4-years old were overweight and another 541,000 were obese. In addition, 1 out of 7 children ages 2–4 from urban families were considered obese (PedNSS, 2009). The PedNSS, (2009) surveillance of toddler-aged, low-income children posited that BMI for age at or above the 95th percentile was an important risk factor for latency-age, adolescent, and adult obesity. The same PedNSS (2009) surveillance showed disproportionate rates of morbid obesity in this age group: African American (19.5%); Hispanic American (23.7%); Caucasian (11.8%); and American Indian and Alaskan Native children (20.7%). In addition, the highest percentage for obesity by gender in the PedNSS (2009) surveillance was African American girls (22.2%) and Hispanic-American males (27.3%).

Researchers have argued that environmental conditions are obesity-producing or obesogenic (Stafford & Marmot, 2003; Van Hook & Altman, 2012). Stafford and Marmot suggested in their study on neighborhood scarcity that deprivation indices contributed to obesogenic environments that placed urban children at risk. Singh, Siahpush, and Kogan (2010) similarly asserted in their study that children living in disparate environments had a 20–60% likelihood of being obese. Research studies also hypothesized a link between the zip codes where

children lived and elevated BMI levels (Åberg, Fritzell, Lundberg, Diderichsen, & Burström, 2003; Frank, Andresen, & Schmid, 2004). Frank, Andersen, and Schmid (2004) suggested that physical location, residential density, and land use impeded the ability of residents to walk verses drive and affected BMI levels by 6% across both gender and ethnicity. Sallis and Glanz (2006) examined barriers found in the environment and suggested that sprawl, density, and safety issues prevented children from participating in opportunistic outdoor activity that, as a result, increased their indoor sedentary activities. Sallis and Glanz further pointed out that those neighborhoods with favorable conditions had more children who participated in outdoor activities.

Wang (2001) found in a national and international logistical study on socioeconomic status (SES) and childhood obesity that urban children between the ages of 6 and 18 in the Unites States had higher obesity rates than like-aged children in Russia and China. Both the Russian and Chinese children were more obese the higher their SES (Wang (2001). Conversely, Kumanyika and Grier (2006) postulated that obesity was more prevalent in low-income minority children particularly due to effects from environmental disparities that included unsafe outdoor conditions that led to increased participation in indoor sedentary activities such as television watching (Campbell & Hesketh, 2007; Kumanyika & Grier, 2006; Sallis & Glanz, 2006, Wang, 2001). Those children typically had more discrepant energy balances with broad energy differences between intake and expenditure (IOM, 2005; Lumen Appugliese, Cabral, 2008).

Blackwell and Macinko (2008) suggested that unfavorable neighborhood designs not only affected children's activity levels and energy expenditures, but also increased the potential for comorbid diseases and subcutaneous fat. Children with subcutaneous fat tended to show signs of adipocytes leaking fatty acids in their systems that caused tissue damage and inflammation

(Deckelbaum &Williams, 2001; Parsons, Power, Logan, & Summerbell, 1999). Adiposity accumulation was described as a major risk factor in pediatric obesity since it contributes more specifically to adult-like comorbid heart disease, blood clots, and diabetes and correlates with the onset of adult obesity (Dietz, 2004; Freedman et al. 2004; Lazarus, Baur, Webb, Blyth, 1996; Serdula et al. 2000; & Steinberger, Moran, Hong, Jacobs, Sinaiko, 2001). Other adult-like diseases that obese children exhibited included nonalcoholic hepatic steatosis or fatty liver, hypertension, glucose intolerance and insulin resistance, sleep apnea, muscular/joint disorders, dyslipidemia, endocrine abnormalities, and elevated metabolic syndrome (Owens et al. 1998; Steinberger, Moran, Hong, Jacobs, Sinaiko, 2001).

The purpose of this research was to examine childhood obesity and the geographic spatial conditions of the environment that interfere with urban children's outdoor activity. Conditions of the built environment are considered obesogenic if they posed barriers in children's natural environment for physical activity.

Problem Statement

The Child and Adolescent Health Measurement Initiative (2012) named North Carolina the 23rd most obese state in the United States for children. Thirty-one percent of the children in North Carolina were overweight or obese; and North Carolina was also named the fifth highest state for overweight and obese adolescents between the ages of 10 and 17 (Robert Wood Johnson Foundation, 2010). The 2012 Children's Defense Fund State data on children showed that 559,875 (24.9%) of the children in North Carolina between the ages of 0 and 17 were poor, and another 11.5% (258,770) were living in harsh conditions of poverty. Mitchell (2012) stated in the North Carolina Budget and Tax Center's brief that North Carolina's child poverty rates had

increased more than 25% since 2007. WIC services in Durham County, North Carolina estimated that 1 in 5 (27%) of 2- to 4-year-old urban children who received supplemental nutrition services were obese with BMI levels for gender and age greater or equal to the 95th percentile (The Partnership for Health Durham, 2007).

Hence, this research examined obesogenic conditions in urban neighborhoods in Durham County, North Carolina considered likely to influence early childhood obesity in the study sample. I used SPSS software to compare the quantitative *t* tests of secondary analysis and means between two unrelated groups on the same continuous, grouping variable: the predictor variable (BMI) and 26 independent environment domain variables. The Levene's Test for Equality of Variances was used to determine if the two conditions had about the same or different amounts of variability between scores as a test of the null hypothesis. The BMI of 2- to 4-year-old children who were considered obese by the federally-funded WIC program was used as the response variable. The geographic location of the study included neighborhoods by zip code in Durham County, North Carolina. The study additionally used data collected from geographic information systems (GIS) used in Durham that independently assessed conditions at the neighborhood level by zip codes across the county as well as census bureau data at the census tract level.

This study adds to the knowledge base of childhood obesity as demonstrated by its focus on the early recognition of childhood obesity at 2 years old in addition to the added efficacy, intervention and prevention strategies that should occur at the youngest age to enhance energy balance during this critical period of childhood (Harrington et al. 2010; Salsberry & Reagan, 2005). A number of earlier studies on childhood obesity focused on the context of school-aged children ages 5 years old and up and included a number of issues such as overeating, SES,

parent's educational level, parental employment, and physical activity (Bronfenbrenner, 1989; Kumanyika & Grier 2006; Luke, Dugas, & Kramer 2007; Pate, O'Neill, & Lobelo, 2008; Proshansky, 1987; Sallis & Glanz 2006; Schmid 2003). In contrast, this study focused on obesity at the earliest ages, 2- through 4-years old, and examined the ecologies where children first acquired food and activity habits. Parents' perceptions of the neighborhood, moreover, served as proxies for whether very young children played outdoors; the relation between this perception and children's outdoor play was evidenced by the children's subsequent sedentary behaviors and BMI levels (Ewing, 2005; Farley, Baker, Watkins; Finkelstein, Hill, Whitaker, 2008; Floyd, Taylor, Whitt-Glover, 2009; Hanson & Chen , 2007; Johnson, Webber, 2007; Moore, Diez Roux, Evenson, McGinn, Brines, 2008)

In retrospective medical chart reviews (*N*) = 184 of pediatric diagnostic codes for excessive weight and obesity in children between the ages of 2 and 20 years, Harrington et al. (2010) found that 111 (58.1%) of the study's children were considered overweight before the age of 2 years old. Harrington et al. indicated that the progression of weight gain that led to obesity in children happened during the first 24 months of life when the child also began to exhibit medical complications and comorbidities. Harrington et al. stated that this critical period of development should be protective to avoid the "tipping point" for obesity (p. 638-643).

Comparable studies on childhood obesity focused on treatment of school-aged children over the age of 4 years as opposed to early age obesity (ages 2-4). Although many of those studies addressed the onset of multiple comorbidities and medical complications that obese children developed, few addressed factors associated with toddler obesity and prevention (Burton, Lethbridge; Caprio, 2006; Droller, 2003; Graham, 2005; Nesbitt et al. 2004; Osberg,

Phipps, 2006). Many of those studies tended to address childhood obesity at the point when medical complications had already begun (Deckelbaum & Williams, 2001; Dietz, 2004; Galvez, Frieden, Landrigan 2003; Hansen 2007; Schmid 2004; Whitaker & Orzol, 2006). Consequently, preventative measures implemented in the first 2 to 4 years of life could abate future obesity through preemptive actions and risk-reduction (Galvez, Frieden, & Landrigan 2003; Whitaker & Orzol, 2006). Also, early childhood obesity managed at the earliest ages could reduce body fat or adiposity and thwart a lifetime of obesity (CDC, 2004; Gomez et al. 2004; Taylor, Falorni, Jones, Goulding, 2003; Whitaker & Orzol, 2006; Wilson, Lirtland, Ainsworth, & Addy, 2004).

Purpose of the Study, Research Questions, and Hypothesis

The purpose of this research study was to determine if relationships exist between BMI and environmental variables in Durham County, North Carolina such that when the variables are grouped by neighborhood zip codes they correlate to childhood obesity. Neighborhood-level conditions specific to urban neighborhoods are among the variables that have an impact on the healthy weight of children (James, 2008).

National attention on childhood obesity has significantly increased over the past 2 decades, and childhood obesity has arguably become a dominant public healthcare issues in recent times (Anderson & Butcher, 2006; Kumanyika & Grier, 2006; Perdue, Stone, Gostin, 2003; Popkin, Duffey, Gordon-Larsen, 2005; Saelens, Sallis, Black, Chen 2003; SGA, 2006). The use of secondary data and GIS software adds to the relevance of social change. This type of spatial analysis supports potential correlations between early childhood obesity and environmental variables that would further the arguement that the obesogenic environment

impedes natural opportunities for outdoor play, which in turn could preclude early childhood obesity.

The following research questions and hypothesis directed this research study:

1. Are the obesogenic conditions of urban neighborhoods associated with early childhood obesity as measured by BMI≥ 95%?
    - H_{01}: Children living in obesogenic environments have no discernible difference in BMI levels.
    - H_{a2}: Children living in obesogenic environments have BMI levels ≥ 95%.
2. Are obesogenic neighborhood characteristic spatially more prevalent in neighborhoods where obese children live?
    - $H_{02:}$ Adverse obesogenic characteristic are not more spatially concentrated in urban neighborhoods where obese children live.
    - H_{a2}. Adverse obesogenic characteristic are more spatially concentrated in urban neighborhoods where obese children live.
3. What are the most prevalent obesogenic neighborhood conditions that are significant explanatory variables that categorically contribute to BMI levels ≥ 95^{th} percentile?
    - H_{03}: Urban children's exposures to neighborhood conditions have no predictive value or increase in BMI levels
    - H_{a3}: Neighborhood conditions have explanatory values that influence the BMI levels in urban children.

Nature of the Study

I hypothesized that the continuous exposure to obesogenic conditions at the neighborhood level affected the BMI levels in urban children and further hindered their ability to expend energy through opportunistic outdoor activities. In this research, I grouped children by neighborhood zip code tabulation areas (ZCTA) and hypothesized that their BMI was influenced by obesogenic conditions of their neighborhoods.

I used the socioecological and environmental framework as the basis to determine any recognizable relationship between the environment and the BMI of urban children. I argued that as neighborhood barriers rise, then the opportunities for outdoor physical energy expenditure diminish, thus making the environment obesogenic (Saelens, Sallis, Black, & Chen, 2003; Van Lenthe & Mackenbach, 2002).

BMI Percentile for Age and Gender

The measure of obesity in children is calculated by using the standard formula, BMI = kg/m^2 or weight in kilograms divided by squared height in meters. This calculation is the most widely used measure by pediatricians for calculating childhood obesity (CDC, 2004). When measuring BMI, metric values are plotted on pediatric developmental charts that are specific to children's gender and age and are referred to as BMI for age (AAP 2003; CDC, 2004; Galvez, Frieden, Landrigan, 2003; HRSA, 2010; Schneider, et al. 2010).

The computation for BMI for age proportionate to or less than the 85^{th} percentile is considered ideal for healthy weight (Schneider & Brill, 2005). Conversely, children with BMI for age that is equal to or greater than 95^{th} percentile are considered obese (CDC, 2004). The standardized percentile ranges used in this study were as follows: healthy weight is from the 5^{th}

to less than the 85th percentile; overweight is greater than the 85th percentile or less than the 95th percentile; obesity is equal to or less than the 95th percentile (CDC, 2000).

A number of studies determined that BMI levels tended to be higher in children under age 5 who lived in urban neighborhoods where variance in neighborhood conditions compromised opportunities for children to expend energy through outdoor activities (Carstairs & Morris, 1989; CDC, 2004). The role between neighborhood conditions and BMI is a health disparity of the environment (Lopez, 2004; Miech et al. 2006; Wang & Dietz, 2002). I, too, believed that continuous exposures to obesogenic conditions at the neighborhood level causes an imbalance in the energy output of children whose subsequent BMI levels are in the range of obesity (Daniels, Khoury, & Morrison, 1997; Paquette & Ryan, 2001).

Table 1

BMI-for-Age Weight Status Categories and the Corresponding Percentiles

Weight Status Category	**Percentile Range**
Underweight	Less than the 5th percentile
Healthy weight	5th percentile to less than the 85th percentile
At risk of overweight	85th to less than the 95th percentile
Overweight	Equal to or greater than the 95th percentile

Note: From Center for Disease Control and Prevention. (n.d.). *Body mass index: About body mass index for children and teens*. Retrieved May 7, 2013, from http://www.cdc.gov/healthyweight/assessing/bmi/childrens_bmi/about_childrens_bmi.html. Used with Permission

Theoretical Framework

Theoretical constructs used in this study were from the ecological and environmental models and provided the basis for gathering data on the correlation between environmental conditions and the BMI levels in urban children aged 2–4 years (Bronfenbrenner, 1989; Proshansky, 1987). Bronfenbrenner's (1989) socioecological theory and the environmental psychology theory on environmental stress as described by Proshansky (1987) assume that neighborhoods have inflexible constraints and physical limitations that, for the purposes of this research, prohibited the healthy expenditure of energy in urban children through outdoor physical activities in their resident neighborhoods. Those theories posit that the physical environment, though modifiable, could have an adverse impact on health and behavior (Bronfenbrenner, 1989; Proshansky, 1987).

The Socio ecological Theory

The socioecological model implies that the environment can correct itself and therefore can change and rebalance (Gregson et al. 2001). Consequently, the ongoing interactions between the environment and behaviors positively or negatively influence health outcomes (Bronfenbrenner, 1989; Gregson, 2000; Stokols, 1996; Gregson, 2000). The premise of the socioecological theory, when applied to early childhood obesity, supposes that salient obesogenic conditions at the neighborhood level influence both the activity level and eating habits of certain subsets of children by their consequential interference with opportunities for outdoor energy expenditure (Brownell, 2002; Cullen, Rittenberry, Olvera, Baranowski, 2000; Swinburn, Egger, Raza, 1999).

Bronfenbrenner (1989) offered the compelling argument that the child's normative development begins in the center core of a multilevel set of ecologies where their biological makeup is impacted by outer-level ecologies. The family is the primary ecology where children develop their initial connections on how to interact with the various environments that they were exposed to (Bronfenbrenner, 1989). Additionally, Bronfenbrenner (1989) proposed that children are dependent on their parents and families at the micro level so that their development is prescriptive and influenced by those initial relationships that further influence how they interact with other ecologies such as their neighborhood level. It is at the micro level where children's interactions are initially formed with their neighborhoods. Those interactions are largely determined by parents who act as policymakers by proxy and thus choose and influence their children's diets, actions, activity levels, and values and shape how their children navigate their primary environment (Bronfenbrenner, 1989; Swinburn, Egger, & Raza, 1999).

Bronfenbrenner (1989) further postulated that the dimension of time spent in any one layer of the ecologies is critical since children develop differently, either positively or negatively, depending on the quality of their relationships, influences, experiences, and physiological experiences within that ecology. The quality of time spent particularly in the micro- and meso-systems is most critical since the former includes the child's primary caretakers and the latter includes day care centers and schools. Bronfenbrenner called the concept of *time* spent in any ecology a chronosystem.

Paquette and Ryan (2001) suggested that the family/parents teach either compatible or incompatible interactions within the ecologies based on quality conditions of the primary environment. This type of chasm between children and their environments has ebb and flow

qualities determined by the changing conditions of the ecologies, and for the purposes of this research, ecologies could rebalance and realign to more favorable forms and provide optimal conditions for the child's energy-balance and activity level (Paquette & Ryan, 2001).

The socioecological model distinguishes between multiecological systems that impact resident children, and those ecologies may or may not have had obesogenic tendencies at the mircosystem, mesosystem, exosystem, and marcrosystem levels. Ecologies that are considered balanced for the child's optimal development are presumed to have fewer obesogenic tendencies and to be healthier (Bronfenbrenner, 1986). Figure 1 illustrates the dynamism of the ecological systems that, by definition, impact children's development and duly influences their energy balance by virtue of the child's interactions within and across the ecological systems (mircosystem, mesosystem, exosystem, and marcrosystem; Bronfenbrenner, 1986).

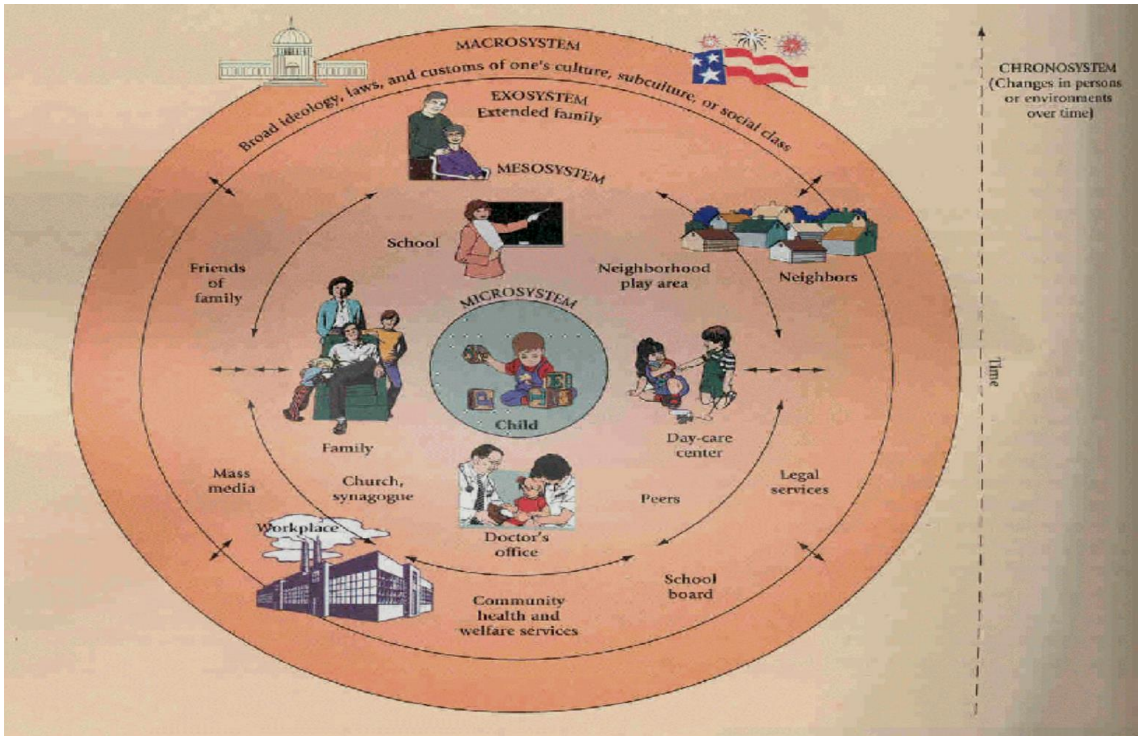

Figure 1. Bronfenbrenner's ecological model. From Bronfenbrenner, U. (1989). Ecologica systems theory. In R. Vasta (Ed.), *Annals of child development* (Vol. 6, pp. 187–251). Greenwich, CT: JAI. Used with permission.

The Environmental Stress Theory

The environmental stress theory surmises that environmental stress either hinders or enhances a child's health based on the child–environment interactions (Baker, 1968; Proshansky, 1987). Baker (1968) also presumed that the eco-behavioral conditions of the environment are contextual while Gibson (1979) concluded that the environment allows affordances based on information-based opportunities for residents to take action towards a desired end. Here, affordances when applied to urban children either hinder or enhance their activity levels (Gibson, 1979; Proshansky, 1987).

How disruptive environmental stressors and affordances are to the basic orientation and development of children in their natural environments contributes to adverse health outcomes at the population level (Gibson, 1979; Proshansky, 1987). Hallman (1984) posited that neighborhoods produce risks that keep children from meeting their desired activity levels since quality factors and the physical makeup of neighborhoods have natural and inherent obstructions and encumbrances that impede outdoor activity such as lack of open space, lack of sidewalks, and lack of outdoor play areas. Additionally, Proshansky (1987) noted that *place dependence* or the relationship between the physical environment and the individual is essential when considering children's behavioral tendencies in relation to their natural environment. Hence, when favorable, the environment meets the children's daily physical activity needs and goals within their own neighborhoods but when unfavorable, adds additional stress (Proshansky, 1987).

McEwen and Stellar (1993) coined the term allostatic load (AL) to describe how stressors from the environment cause neurochemical reactions in the body stemming from physiological overload and deterioration. Under recurring stress or allostatsis, the body begins to wear and bodily systems become deregulated (McEwen, 1998; McEwen & Stellar, 1993). Cumulative allostatic load to the physiological and biological systems of children results in dysregulation and accelerated weight gain (Gundersen, Mahatmya, Garasky, & Lohman, 2011).

The Multiple Exposures Multiple Effects Theory

The multiple exposures multiple effects (MEME) theory coined by the World Health Organization describes a conceptual framework for children's health factors and purports that multiple exposures to compelling environmental conditions results in contributory disease and

health impact (Briggs, 1999; WHO, 2003). Here, the environment and its contextual specificity cause forced interactions between the environment and its residents and, due to those multiple exposures, eroded health. This complex relationship between different environmental exposures and children's health, for the purposes of this research, implied that children's health outcomes were affected by the contextual conditions of the environment (Briggs 2003; WHO, 2003). The MEME model also presumes that socioeconomic factors and environmental vulnerabilities unjustly impact resident populations (WHO, 2004). Unjust environmental conditions under the MEME model that adversely affect children's health were described as physical inequities, biological inequities, poor air quality, poor food and water quality, home degradation, poor quality of schools and community, and the poor spatial distribution and quality of other life amenities (WHO, 2004).

The Attachment Theory

Vaughn, Egeland, Sroufe, & Waters (1979) argued in their research on the attachment theory that children experience proportionately the same stressors in the environment that their primary caretakers experience and that those stressors impede the primary maternal–child attachment. Hence, the higher the environmental stressors, the higher the stress levels of the neighborhood children's parents (Vaughn, Egeland, Sroufe, & Waters, 1979). The more deprived the environment is of natural supports and resources, the more stressed are its members (parents or primary caretakers). Cassidy, Parke, Butkovsky, & Braungart (1992) explored the emotional responses in families and the children's tendencies to take on those emotional responses as evidenced by the dynamic interactions between parent and child. Here, the emotional climate and stress in the family determine the child's coping habits. The child's interactions with the

environment per the attachment to the primary caretaker differ based on the child's culture, race, class, and gender (Cassidy et al. 1992).

Individualistic Theory

For the purposes of this research, the individualistic theory surmises that genetic and metabolic predispositions in children influence favorable or unfavorable weight outcomes (Hoppin & Taveras, 2004). Individual children with high-risk genetic profiles are more susceptible to excessive weight (Burrage & McCandless, 2007). Children with certain genetic makeup or genetic encoding have the propensity to become obese due to greater heritability factors that are non-modifiable (Burrage & McCandless, 2007; Hoppin & Taveras, 2004). The gene mechanisms that influence specific weight regulations suggest particularized set points determine the child's weight (Hoppin & Taveras, 2004). The extent and duration of breastfeeding and the physiologic factors in breast milk, conversely, is thought to mediate the onset of childhood obesity by providing protective factors against early childhood obesity and excess weight (Schneider & Brill, 2005).

Definition of Terms

Childhood or p*ediatric* o*besity:* The adiposity (fat) in tissue in excess of lean body mass (The University of Maryland Media Center Medical Encyclopedia, 2012).

Energy b*alance:* The total energy intake equals the total energy expenditure (The Nutrition Diet Medical Dictionary, 2008).

Energy e*xpenditure:* The balance between energy intake and energy output (Encarta Webster's Dictionary, 2004).

Geographic information systems (GIS): Quantitative and spatial reference to measures of the built environment using location as a key variable to create interactive maps (Thornton, Pearce, & Kavanagh, 2011).

Health behavior: The actions that a person takes to prevent maintain or regain optimal health.

Index of multiple deprivations (IMD) Environmental indices linked to economic disadvantage (Carstairs & Morris, 1991; IMD, 2000).

Multifaceted interventions: Interventions that complement but do not compete with one another.

Neighborhood-level conditions: Neighborhoods that are impoverished and that subsequently impede the activity behaviors in children.

Obesogenic: Conditions in the environment that encourage weight gain in people by evidence of low calorie expenditure (Swinburn, Egger, & Raza, 1999).

Physical activity: The required physical skills or endurance above the basal level needed to improve overall health (U.S. Department of Health & Human Services, 2008).

Poverty: Children who are urban by virtue of their family's income that meets the U.S. poverty limit (The U.S. Census Bureau, 2010).

Sedentary activity: Activities that do not increase the expenditure of energy above the basal level (Pate, O'Neill, & Lobelo, 2008).

ZIP code tabulation area: Aggregation of census blocks adjusted to define the land area to approximate delivery areas and to tabulate census data (US Census Bureau Decennial Management Division Glossary, 2010)

Assumptions, Limitations, and Delimitations

I surmised that the relationships between the BMI levels ≥ 95 percentile of urban 2- to 4-year-old children and obesogenic variables in neighborhoods in Durham County, North Carolina were remarkable and that the data from this study would support the county's 2011 CEHI's Community Assessment Project. I also assumed that the Durham County, North Carolina 2011 CEHI's Community Assessment Project on the built environment could be accessed for purposes of this research and could be layered geographically by ZCTA with the 2011 NC-NPASS BMI levels coded by the Durham County, North Carolina WIC Program for children whose neighborhoods were categorized by ZCTA. Data from both the CEHI's Community Assessment Project and the NC-NPASS WIC data were assumed to be from reliable databases and available for this secondary data analysis.

Additionally, I assumed the NC-NPASS data used in this research to be inclusive of only 2- to 4-year-old children seen in the Durham County Public Health WIC center, but I also considered the data limiting since it did not offer comparison BMI data from like-aged peers not receiving services through the WIC center. Another recognized limitation of this research was that the data from secondary sources might not have agreed with the intended purpose and measures needed for this particular research. In addition, a potential barrier to this study's replication was costs. To complete this type of GIS study in inner cities as primary or initial research study can be cost-prohibitive with projected cost in the hundreds of thousands of dollars. Hence, cost of an initial GIS study could limit future GIS studies and thus impede the use of this type of technology in health prevention and interventions. Another assumption to this research study was that chances for outdoor activities for the study population could continue to

remain limited since this age group will remain dependent on a parent acting in proxy for the activity to happen.

BMI was used as the best measure childhood obesity in this study even though there are studies that used other measure such as the air displacement plethysmography (ADP; Buchholz, Majchrzak, Chen, Shankar, & Buchowski, 2004). The ADP analyzes the pressure-volume association between body volume and body mass to determine adiposity. This tool is not widely used in children and not practical in the clinical pediatric office since it requires that the child be seated inside a fiberglass test chamber (Buchholz et al. 2004; Dempster & Aitkens, 2005). Other alternative measures of obesity not considered for this study included the use of bioelectrical impedance analysis (BIA) Ricciardi and Talbot (2007) argued that BIA is a more comprehensive measure of the high risk morbidities in children than BMI since this test measures body fat in addition to water weight, hydration, and tissue ischemia.

The measurement of waist and hip circumference was not considered for this study but is a frequently used measure for fat distribution in children and is purported to capture visceral adipose tissue that is indicative of comorbid health risks and metabolic disorders and the skin fold thickness measure fat measure (Freedman & Sherry, 2009; Himes, 2009; Bagust & Walley, 2000). Although alternative measures of obesity/adiposity in early childhood obesity is important for research and for ruling out specific risk factors such as cardiovascular and diabetic diseases, the use of BMI for age and gender percentiles is supported in the literature as being the most practical in routine pediatric practices (CDC, 2013). BMI is sufficiently practicable and offers appreciable information in the early identification and monitoring of overweight and obesity in early childhood (CDC, 2013).

Another limitation of this study was the sample size (*N*=33) and whether the criterion variable could yield a properly-specified regression model given the explanatory variable's GIS data. In addition, I anticipated that GIS spatial testing with census tract data has limitations due to large amounts of data within the census tracts and a small sample size, potentially causing poor testability. Another expected limitation of the study was the expense of using GIS spatial analysis. The use of this fairly new technology in public health studies has steep costs associated with it for both consultation and software. Parenthetically, I have limited training in GIS spatial analysis and needed to acquire the basic concepts, methods, mapping of polygon data, and key terms related to using GIS software for purposes of this study.

The literature on childhood obesity showed a correlation between body fatness/adiposity and BMI in children below the age of 5 years old but was limited in studies using alternative measures of adiposity in early childhood obesity (Bagust & Walley, 2000). Although BMI is considered a sufficient measure of obesity in children, it does not measure excessive accumulation of adiposity tissue (Freedman & Sherry, 2009; Himes, 2009). Researchers have argued that the percentage of body fat in children studied in both clinical and research settings shows unremarkable scientific use of such measures (Bagust and Walley, 2000; Freedman and Sherry, 2009; Himes, 2009). The evidence in the literature on early childhood obesity also was less extensive than those studies on latency-aged and adolescent obesity (Freedman & Sherry, 2009). Participants in this study were delimited to 2- to 4-year-old children who participated in the WIC program and resided in a neighborhood in Durham County, North Carolina. The participants were delimited to those whose family met the poverty threshold at or below 185% of

the U.S. Poverty Income Guidelines. Delimitation also required that participants had BMI-for-age weight $\geq 95^{th}$ percentile.

Significance of the Study

This research explored the relationships between BMI levels and neighborhood-level conditions that, when applied to early childhood obesity, add a compelling thrust to the breadth and depth of the research on childhood obesity. The use of emerging GIS technologies to geographically map obeseogenic relationships between BMI and built environment variables offered important thoughts on early prevention and interventaion strategies at the neighborhood level (Frank, 2005; Glanz, Sallis, Saelens, Frank, 2005; Nuckols, Ward, Jarup, 2004; Shaw, 2012). Applying GIS technologies to the study of childhood obesity could influence policy changes that address neighborhood inequities and their impact on population health. This research might also enhance partnerships across stakeholders to address early childhood obesity in urban children at a more fundamental neighborhood level.

I focused on children ages 2 to 4 in this research study based on the theory that very young obese children become obese adolescents and consequently obese adults. This early onset of obesity is critical when considering public health and community-based prevention and intervention strategies at its lowest population level (Miller, Rosenbloom, & Silverstein, 2004; Moran,1999; Nesbitt et al. 2004). This research offers a snapshot of spatial and geographic population obesity in urban areas to inform decisions and program development tailored to population and cultural sensitivities (Freedman et al. 2004; Kerbs, 2005). More to this point, this study's findings might drive vigorous discussion and future momentum on the effects of the environment on the early health of urban children at the neighborhood level (Anderson &

Butcher, 2006; Flegal, Tabak, & Ogden, 2006). Scholarly support for this argument included the Pediatric Nutrition Surveillance Report (2009) that stated that children from birth to 4 years old have a 14.7% prevalence rate for obesity which was an increase from 13.2% in 2000 and the National Survey of Children's Health (NSCH, 2003) that postulated that approximately 22% of urban children ages 5 and under were obese and were most affected by salient neighborhood-level conditions such as socioeconomic status, degradation, and access to community resources.

Implications for Social Change

The social change aspects of this research are increased attention on a comprehensive approach and shared partnership across the health sector to use innovative GIS technologies to address the complex health challenges of the built environment in urban neighborhoods. This research holds up the hypothesized link between neighborhood conditions and obesity outcomes in children supported in the literature and used GIS technology to identify any correlation with those obesogenic conditions. Other social change aspects of this research include arguments on policy and environmental design as barriers to health; critical discussions in those areas would inform and improve influences on early childhood obesity at the micro and macro levels (Bronfenbrenner, 1986). I supposed that GIS tools used in the study of early childhood obesity could influence critical cross-disciplinary discussions and debate and drive social change.

I expect this study to initiate conversations between public and private entities on policy, personnel, and political attitudes and in media promotions by focusing on this younger population of obese children. Findings from this research might also fuel global considerations on early childhood obesity in urban children. Other social changes might include the informed redesign and use of land and space in urban neighborhoods such as the use of open space,

building of playgrounds and community parks, and making recreation center available to neighborhood residents. In addition, this research encourages examination of neighborhood degradation as obesogenic, recommending tearing down condemned and dilapidated buildings and houses, removing litter and debris, and promoting neighborhood cleanliness. Hence, the practical social change implications inherent in the study on early age obesity, when used as a benchmark for social change, has far reach that could inform policy changes, environmental design, and health promotion activities.

Chapter 2: Literature Review

In order to draw relationships between the conditions of urban neighborhoods and BMI levels in the research population, I focused this literature review on the probable, inclusive, and interrelated built environment variables arguably linked with the BMI levels of urban children. I examined variables considered corollary for early childhood obesity under the assumption that early age exposures to obesogenic conditions at the neighborhood level not only interfere with natural and opportunistic outdoor activity in children but influence their BMI levels associated with obesity.

Literature Search

This literature review was comprised of a comprehensive search of scholarly manuscripts and refereed journals in both electronic and print form. I used Google Scholar, Medline, PubMed, SAGE, ProQuest, and EBSCO databases and collected and reviewed many peer-reviewed journal articles and dissertations. A number of publications used in this study identified plausible relationships between the contextual conditions in urban neighborhoods and BMI levels in urban children and tended to associate those with factors of socioeconomic, demographic, and health disparities. In addition, numerous publications emphasized adult-like comorbidities that were often found in obese children, and many others surmised that joint efforts are necessary between public, private, state, and federal partners to reverse the upward trend of childhood obesity. A number of articles concluded that neighborhoods had obesogenic conditions that interfered with children's ability to expend energy through outdoor activity, and those scholarly resources were mainly screened for statistical associations between neighborhood obesogenic conditions and children's activity and BMI levels.

Search terms and phrases used for this study included *BMI, obesity, adiposity, obesogenic, childhood obesity, body weight, nutrition, built environment, ethnic differences in obesity, metabolic syndrome, and obesity risk factors, environmental health disparities, and geographic information systems.* Accordingly, I reviewed over 1000 abstracts, journal articles, and citations that were relevant to early childhood obesity, BMI, and the impact of neighborhood disparities on health and I included over 370 of those as references that added to the depth and breadth of this research study.

Conceptual Framework

The conceptual framework for this research was based on multiple theories that associated the well-being of children with their interactions and exposures to social, ecological, and environmental conditions at the neighborhood level that were considered obesogenic (Bronfenbrenner, 1989; Paquette & Ryan, 2001; Proshansky, 1987).

Carstairs and Morris's (1999) research on determinants of deprivation indices in relation to spatial epidemiology formed a basis for identifying built environment variables that were considered obesogenic. I used Carstairs and Morris's theories of social inequalities in the environment such as unemployment, socioeconomic status, lack of transportation, and overcrowding or congestion as a framework in this study. Carstairs and Morris posited that the higher the environmental inequities, the higher the health impact. Carstairs and Morris's deprivation indices were one of the first studies that incorporated an index of multiple deprivations (IMD, 2000) and the first national study of deprivation at the ward level; there were 8,414 wards across all the counties in England. This timely study provided the impetus for policy change based on deprivation indices and funding was subsequently provided to the top 10% of

the wards identified from the study. Those wards were identified as the most disparate on indices of deprivation. Additionally, Jarman's (1983) study on medical services and resource availability correlated with low access for children under the age of 5 living in families with unskilled, unemployed, or single parents or in overcrowded households and those classified as nonwhite.

Paquette and Ryan (2001), in their work on the socioecological theory, postulated that both the child's biological and physical development were linked to the quality of interactions they had with the primary ecology where they had the most exposure to the family and community environments. Paquette and Ryan suggested that the primary ecology fueled the child's development and had either positive or adverse effects on children (Bronfenbrenner, 1989; Proshansky, 1987). Bronfenbrenner's (1989) ecologies of the environment implied that the immediate microsystem had bidirectional influence on the quality of the parent–-child relationship, and though greatest at the primary microsystem level, the mesosystem (neighborhood) and the macrosystem (social conditions) had as much bidirectional influence on the child (Bronfenbrenner, 1989; Proshansky, 1987; Vaughn et al. 1979).

The idea that frequent exposures and forced interactions with detrimental environmental conditions contributed to disease was stipulated in the MEME model (Briggs, 1999). Proshansky (1987) theory on environmental stress implied that multiple deprivations such as violence, crowding, degradation, and space contributed to health outcomes. To this extent, neighborhood obseogenic conditions had adverse influence on the weight of children in low-earning families; obseogenic conditions had modifiable loci that could restore health (Black & Macinko, 2008; Bronfenbrenner, 1989). For purposes of this study, multiple deprivations included:

socioeconomic status, parent education, housing, safety, crime, and environmental degradation to name a few (Carstairs & Morris, 1991; IMD, 2000).

Neighborhood Factors

Neighborhood Socioeconomic Factors and Social Determinants of Health

I examined obesity in children and the poverty income ratio for this study. Published research placed urban children at greater health risk (Wang, 2001). Neighborhood socioeconomic status that was associated with children's health and environmental equity, and low socioeconomic status was particularly detrimental to racially minority children whose family income, unemployment, lack of transportation, and access to resources were limited (Saha, Eckert, Pratt, & Shanker, 2005). Talpade (2008), in a population study, posited that there were both similarities and differences found between Hispanic American (HA) and African American (AA) girls on disparities matrices such as food intake, obesity frequency, and Type 2 diabetes, and those similarities and differences crossed on several demographic variables to include SES, unemployment rates, and educational attainment. Talpade (2008) also postulated that food intake and body image between HA and AA girls had contextual influence based on culture. Lobstein, Baur, and Uauy (2004) posited that low-income children worldwide had disproportionate rates of obesity and were more vulnerable to chronic health disorders that were persistent and continuous. Lobstein et al. further indicated that though 10% of the world's children were overweight, the rates of excess weight in American children from urban environments increased 0.05% annually.

Of relevance was the excessive toll that family income had on early childhood obesity (Currie & Stabile, 2003; Wang, 2002, 2006). A review of the NHANES (2005–2008) survey

showed that the obesity gradient for children ages 2 to 19 increased as family income decreased. Here, obesity prevalence rates were higher for urban children whose family income was less than 130% of the poverty level as opposed to children whose family income was more than 350% of the poverty level: 21.1% of boys had family income less than 130% of the poverty level compared to 11.9% whose family income was more than 350% of the poverty level; 12% of girls had families whose income was less than 130% of the poverty level compared to 19.3% of girls whose family's income was at or above 350% of the poverty level.

Across race and ethnicity, the NHANES (2005–2008) survey showed that prevalence rates for obesity were less consistent for non-Hispanic Black and Mexican American boys and girls, though Mexican American girls showed obesity rates of 21.0% at or above 350% of the poverty rate and only 16.2% obesity rates less than 130% of the poverty level. Non-Hispanic white boys showed obesity rates at 10.2% at or above 350% of the poverty and 20.7% obesity rates below 130% of the poverty level. Non-Hispanic white girls' obesity rates were 10.6% at or above 350% of the poverty level and 18.3% obesity for girls living below 130% of the poverty level. Interestingly, NHANES reported that most 2- to 19-year-olds who were obese were not living in low-income households as per the poverty income ratio.

Studies on the demographics between neighborhood socioeconomic status and weight showed that the balance between healthy and unhealthy weight for urban children had predictive value for comorbid diseases including cardiovascular disease, Type 2 diabetes, and sleep apnea, and those diseases were associated with socioeconomic gaps in the neighborhood (Kumanyika & Grier, 2006). According to Brenner (1995) and Rothenberg (1988), socioeconomic status created unequal burdens that adversely affected health outcomes. In addition, the economic gradients in

urban neighborhoods showed health disparities that were skewed towards children whose parents were low on income, education, and age indices (Haas et al. 2003; Rothenberg, 1988). Babey, Hastert, Wolstein, and Diamant (2010), in their 2001–2007 study, used digital phone surveys and examined obesity rates by race and socioeconomic status in California adolescents (N = 17, 535) and concluded that income disparities and obesity were common factors for obesity in adolescent girls. Here, as the family's income decreased, obesity increased in both California Latino and African American adolescent girls, suggesting that differences in income gaps of low-income families adversely impacted weight in urban adolescents girls.

Case, Lubotsky, and Paxson (2002) posited in their secondary study that the gap in health status at birth for urban children widened in relation to their parent's socioeconomic status. Brenner et al. argued that there was a direct relationship between socioeconomic status and health where change in one affected change in the other. The effects of economic recessions, too, had considerable influence on health behavior particularly because of economic interactions between productivity and consumption (Case, Lubotsky, & Paxson, 2002; Krieger et al. 2005). This quasi-cyclical feature of the economy had a benefit, loss, and reward sequence that adversely impacted the health of urban children as a result of their family's income strata (Brenner 1998; Case, Lubotsky, & Paxson, 2002; Krieger et al. 2005; Rothenburg 1988; Turnock, 2004). Though similar on several outcome variables, socioeconomic in urban neighborhoods was most analogous in the literature with health outcomes in children than any other demographic factor (Johnson-Down, O'Loughlin, Koski, & Gray-Donald, 1997; Kimbro, Brooks-Gunn, & McLanahan 2007).

Much of the literature supported the relationship between the social, economic, and physical environment and obesity in urban children. Additionally, Gordon-Larsen et al. (2003) hypothesized from a STATA survey (SVY) series that there was an inversed relationship between income and parental education. Gordon-Larsen et al. found that differences in children's weight was highest at the middle income level for Asian (13.5 percentage points), African Americans (2.8 percentage points), and Hispanic (2.1 percentage points) children but decreased for Caucasian girls by 3.27 percentage points. High socioeconomic status and overweight prevalence increased for African American girls (5.6 percentage points) while it decreased for Hispanic and Caucasian girls the higher the socioeconomic status (−0.1 percentage points). Gordon–Larsen et al. also postulated that stressors found in the environment hindered adequate energy expenditure and activity levels that influenced weight gain.

Neighborhood Socioeconomic Factors and Stress

I examined studies on the relationship between socioeconomic status and childhood obesity in the literature together with children's exposures to environmental stress (Anderson & Butcher, 2006; Kumanyika & Grier, 2006; Proshansky, 1987; Wang, 2001). Kumanyika and Grier (2006) construed that environmental degradation, crime, and safety caused stress to children because it compromised their ability to play safely outdoors. Sallis and Glanz (2006) contended that socioeconomic disparities produced safety and stress issues for children that obstructed their ability to expend energy through outdoor physical activity and play. In addition, Sallis and Glanz posited that low-income families were often geographically concentrated in neighborhoods where walkability was limited because of higher rates of violence and crime that increasingly affected outdoor activity.

Studies that used census enumerated classifications for race and ethnicity across states such as New York, New Orleans, North Carolina, Ohio, and California showed that low-income children had higher rates of exposure to desolate neighborhoods where environmental stress and safety limited access to food and recreation resources (Graham 2005; Kumanyika & Grier, 2006). Those neighborhoods had similar demographics such as low socioeconomic status that impacted suitable life options (Burdette & Whitaker, 2004; Kumanyika & Grier, 2006; Lutfiyya, Garcia, Dankwa, Young, & Lipsky, 2008; Molnar, Gortmaker, Bull, & Buka, 2004). Molnar, Gortmaker, Bull, and Buka (2004), in a multilevel longitudinal study, concluded that disorder measures (evidence of trash, graffiti, empty beer bottles, condoms, and needles) in the environment were associated with parental stress particularly when the neighborhood was perceived as unsafe for outdoor play.

Likewise, Cohen, Evans, Krantz, and Stokols (1986) postulated that children's continuous exposures to environmental stress impacted their health and their parents as proxies also were equally affected by the same environmental stressors. Drentea and Lavrakas (2000) additionally, found in a comparative study on credit card use and debt stress, that the stress load on parents from debt was cumulative and impaired their health. As a result, the degree of difference between the costs of resources and the burdens from financing purchases caused prolonged stress for low-income parents / populations. Drentea and Lavrakas found that finance rates were inconsequential when the priority was to use credit cards to counterbalance life amenities. The spillover effect from financial distress had both socioeconomic and racial correlations (Drentea & Lavrakas, 2000).

Neighborhood Conditions and Opportunities for Outdoor Activity

Burdette and Whitaker (2004) hypothesized in their cross-sectional study of 7,020 poor 3- to 4-year-old children that their activity levels were related to their exposures to neighborhood crime and its proximity to playgrounds and fast food eateries. However, Burdette and Whitaker found no meaningful correlation between BMI levels and those proximal indices. Likewise, research from IOM (2005) contended that a correlation existed between the conditions of the neighborhood and obesity in children living in lower socioeconomic neighborhoods but identified that multifactor variables in the neighborhood overlapped and had equivalent impact on weight gain in children.

Degradation matrices such as no sidewalks or unwalkable sidewalks, debris, congestion, lack of open spaces, lack of street lights, insufficient parks, and inadequate places to exercise were verified in the literature as barriers for urban children outdoor activity (Burdette & Whitaker, 2004, CDC 1999; Graham, 2005; Kumanyika & Grier 2006; Sallis & Glanz, 2006). According to IOM (2004) and CDC, (1999), children's continuous exposures to those types of neighborhood conditions decreased their ability for outdoor activity and peer social integration.

The literature also suggested that low social gradients in neighborhoods had systematic deficiencies in quality of life resources (Powell, Slater, & Chaloupka, 2004). Kumanyika and Grier (2006) posited that the poorer the neighborhoods as a unit, the more juxtaposed it was against the presence of neighborhood parks, health care facilities, play grounds, and healthy food establishments. Hence, the absence of those amenities in neighborhoods not only disproportionately increased children's sedentary behavior, but affected their quality of life (Kumanyika & Grier, 2006).

Children born into urban families had statistically higher paucity of resources and quality of life amenities (Brenner 1998; Case, Lubotsky, and Paxson, 2002; James, 2008; Krieger, Chen, Waterman, Rehkopf & Subramanian, 2005; Rothenburg 1988; Sampson, Morenoff & Gannon-Rowley, 2002; Turnock et al. 2004). Those disparities in urban families showed contributory correlation to pediatric obesity (Cossrow & Falkner, 2004; Librett, Yore, Schmid, 2003; Wang & Zhang, 2006). Additionally, Haas et al. (2003) and Gomez et al. (2004) not only concluded that there was a correlation between obesity occurrences in children who were covered by WIC or public Medicaid insurance, but also concluded that those children were more sedentary and were treated more often for weight-related medical conditions.

Moreover, the frequency of childhood obesity was strongly associated with urban neighborhoods (Chang, 2006; Wang & Dietz, 2002; Wang & Zhang, 2006). Those neighborhoods often had high levels of obesity in African American and Hispanic children and adolescents (Miech et al. 2004). Contrarily, Wang and Zhang (2007) reported that income alone was not an indicator of obesity in their study since socioeconomic status for African American girls from higher economic brackets also had BMI greater than or equal to the 95th percentile. Wand and Zhang suggested that weight gain was not only influenced by income, but also by environmental, social, and cultural influences.

Disparity indices influence on the activity levels in children were referenced by Pickett and Pearl (2001) who argued that there was an analogous relationship between neighborhood degradation, socio - economic conditions and the weight of urban children. Darmon and Drewnowski, (2008) stated that the link between socioeconomic status weight was associated with the food sources available in the neighborhood and the nutritious quality of those foods.

More specifically, Sallis & Glanz (2006) CDC (1999) Kumanyika & Grier (200) Graham (2005) proposed that the lower the neighborhood income gradient, the higher the unfavorable neighborhood indices (conditions of homes, schools, parks, roads, playgrounds, crime levels and community aesthetics) decreased the chances of children's incidental and recreational outdoor play.

Obesity as Demographic Health Determinant

Scholarly perspectives on obese children included a mixture of exogenous and biological demarcations considered as health determinants (Stevens, Seid, Mistry & Halfon. 2006; Molnar, Gortmaker, Bull, Buka, 2004). Research studies suggested that urban children generally experienced substandard resources and circumstantial life conditions that precluded their access to adequate health care systems (James, 2008; Johnson-Down, O'Loughlin, Koski, Gray-Donald, 1997; Kumanyika, 2006; Kumanyika, 2008; Wilson, 2009,).

Obesity as a health determinant had disproportionate disease impact on urban children and had disparate impact between racial groups on indices of BMI, SES, comorbidities and resource access (Stevens, Seid, Mistry and Halfon. 2006). How urban children negotiated their daily neighborhoods was cited in the literature as being dependent on neighborhood crime rates, neighborhood drug use and exposures and gang activities and those factors were further considered to be health inequities (Ainsworth, & Addy, 2004; Graham 2005; James, 2008; Kumanyika, 2006; Kumanyika, 2008; Stevens, Seid, Mistry & Halfon. 2006; Wang & Dietz 2002; Wilson, Kirtland,; Molnar, Gortmaker, Bull, Buka, 2004). Sorof, Lai, Turner, Poffenbarger, Portman (2004) concluded that there was a disproportionate co-morbid disease impact on urban obese children than on their Caucasian obese counterparts. Likewise, a review

by The National Institute of Health [NIH] (2000) characterized inequities in obesity rates in urban children as empirically distinct in incidence rates, prevalence, mortality, and was considered more of a disease burden than those seen in like - aged non-Hispanic White children. The NIH applied the definition of health disparity to those differences since statistically they posed greater health risks for urban minority children.

Conditions cited in the literature at the neighborhood – level that were described as prohibitive or barriers to healthy behaviors were likewise called health disparities (Gregson, Foerster, Orr, Jones et al.; Solomons & Kumanyika, 2000). The schematic layout of inner-city neighborhoods was also described as an access to care health disparity since unequal access to resources and resource distribution was argued to produce health inequities (Bambra, Gibson, Wright, Whitehead, Petticrew, 2009; Kumanyika, 2002; Wilson, 2009).

Lastly, Deckelbaum and Williams (2001) surmised that urban children had biological indicators and genetic compositions that influenced obesity rates and that were considered health disparities. Predispositions for type II diabetes and metabolic syndrome were higher in minority urban children by virtue of their genetic composition and markers (Swinburn, Gill, Kumanyika, 2005; Wilson, 2009). Research from The Institute of Medicine (2005) also indicated that contextual differences at the neighborhood frequently precipitated genetic markers in urban children akin to the same disease precipitants in urban adults.

Social Integration

To the extent that children felt disconnected from their social network was cited in the literature as being proportional to their health and well - being (Falci & McNeely, 2009). In the same way, social integration was thought to be proportional to children's ability to become

socialized in their neighborhoods (Carstairs & Morris, 1989; CDC, 2004; Gomez, Johnson, Selva, & Sallis, 2004; Wang & Zhang, 2006).

Wilson, Kirtland, Ainsworth, & Addy (2004), and Coulton, Korbin, Su, (1996) argued that children had a need to develop natural social interactions with others in their environments and thus responded differently, both behaviorally and physiologically, when those did not happen. Falci & McNeely (2009) postulated in their study on social cohesion and belongingness that social integration was critical to children's friendship formation and connectedness that naturally increased when opportunities for socialization were available. Baranowski, Perry, & Parcel (2002) suggested that neighborhood resources affected children's social integration and social networking. Baranowski, Perry, & Parcel (2002) concluded that the decisions children (parents) made daily on whether to walk to school, play outdoors or go to the neighborhood parks enhanced or prevented opportunities for social integration.

Baranowski et al. (2002) further argued that the parent's decision - making was based on their socio-cognitive perceptions on the safety of the neighborhood. Lopez (2004) postulated that neighborhood sprawl attributed to parent's decisions to keep their children indoors thus interrupting opportunities for outdoor social integration, activity and play. Likewise, several studies showed that observable inequities in the neighborhood subsequently caused a perpetual pattern of social disintegration (CDC, 2004; Graham, 2005; Lopez, 2004; Stevens, Seid, Mistry, Halfon, 2006; Wang & Dietz, 2002).

Disease Burden of Obesity

Comorbidities and Childhood Obesity

The American Academy of Pediatrics [AAP] (2003) and the Institute of Medicine [IOM] (2005) described medical demarcations that were generally seen in early childhood obesity, but not seen in comparative preschoolers of healthy weight. Those differences included greater prevalence for adult - like health problems such as the early onset of coronary heart disease, gallbladder disease or cholecystitis, pancreatitis, non-alcoholic hepatic steatosis / fatty liver, hypertension, type II diabetes, metabolic syndrome, obstructive sleep apnea, muscular and degenerative joint disorders, dyslipidemia, endocrine abnormalities and impaired insulin tolerance (Burton, Lethbridge, Osberg, Phipps, 2006; Caprio, 2006; Droller, 2003; Graham 2005; Nesbitt et al. 2004).

In addition, minority children showed higher rates of insulin intolerance and resistance called acanthosis nigricans. Acanthosis nigricans was described as hyper pigmentation or dark markings around the neck commonly indicative of type II diabetes (Ogden, Carroll, Curtin, McDowell, Tabak, Flegal, 2006). Caprio (2006) suggested that certain diseases associated with childhood obesity also required medication and surgical interventions. Obese urban children who lived in certain geographic areas similarly showed clustered risk for secondary diseases (Cossrow & Falkner, 2004).

Urban children (combined sexes) were estimated to have a 15% higher comorbidity disease rate (Anderson & Whitaker 2009). Risk factors for two prevalent disorders; metabolic syndrome and type II diabetes predictably triggered early onset of cardiovascular and other associative diseases (Eyre, Kahn & Robertson, 2004). Children exposed to neighborhood

deprivations and SES inequities had higher rates of those two secondary diseases (Cook, Weitzman, Auinger, Nguyen, & Dietz, 2003; Gutman, McLoyd, & Tokoyawa, 2005).

In a four year analysis of NHANES data from 1999-2002, Hedley, Ogden, Johnson, Carroll, Curtin, Flegal (2004) found that obesity increased steadily over time in both adults and children. Here, children showed a continual and successive increase in weight gain over the study's four year analysis (Hedley, Ogden, Johnson, Carroll, Curtin, Flegal, 2004). A smaller but similar secondary analysis showed children's steady weight gain over a two year period from 1999 - 2000 and showed no indication of obesity leveling during that period (Ogden, Flegal, Carroll, Johnson, 2002).

A six - year secondary analysis by authors Ogden, Carroll, Curtin, McDowell, Tabak, Flegal, (2006), not only showed high prevalence rates of obesity in children that increased over time, but also showed remarkable correlations between obese children's race and ethnicity. Those studies showed linear trends for weight gain correlates similar to research noted for the adult-like diseases found in obese children (Ogden, Carroll, & Flegal, 2008). Deckelbaum & Williams (2001) cited Blount's disease as a comorbidity that caused the child's legs to bow outwards under the pressure of extreme weight. There was no cure indicated for Blount's disease and when not treated early caused continued leg bowing under the strain of increased weight gain.

Adiposity

Adipose tissue was a critical factor noted in the childhood obesity literature (Freedman, Mei, Srinivasan, Berenson, Dietz, 2007; Krekoukia, Nassis, Psarra, et al. 2007). Adipose tissue or the accumulation of body fat / fat mass had higher percentage rates in obese children and was

linked to metabolic disorders related to cardiovascular autonomic nervous system [cANS]; and inflammation and vascular irregularities in obese children (Kaufman Kaiser, Steinberger & Dengel, 2007). Increases in inflammation also caused the secretions of protein producing adipokines in tissues which showed a causal role in obesity associated disease and disorders (Freedman et al. 2007; Huang, Johnson, Figueroa-Colon, Dwyer, Goran, 2001).

Carnell & Wardle (2008) reasoned that children with adiposity were more susceptible to voracious appetites evidenced by lower satiation - rate conditions. Freedman, Thornton, Mei, et al. (2004) found that obese children were often tall for their age and height as compared to like – aged peers which correlated with higher levels of body fat. This research confirmed that adipose tissue was associated with BMI greater or equal to the 95th percentile. Hence, the best measure of adiposity in children given their changing body composition over time was BMI raw scores and BMI percentiles (Cole, Faith, Pietrobelli & Heo, 2005).

Morgan et al. (2002) similarly postulated that urban African American children were genetically more susceptible to adiposity and thus were less able to balance metabolic variations in energy expenditure. Researchers from Wake Forest University, Shively, Register, Clarkson (2009) contended that environmental stress correlated with the composition of visceral fats that accumulated in the abdomen and other visceral organs such as the liver, heart and blood stream. Health implications from that type of fat were more profound than typical subcutaneous fats found in other parts of the body since visceral fats tended to influence metabolism, sugar levels and energy expenditure. Carnell & Wardle (2008) in their study on appetite and adiposity found that urban children not only had susceptibility to being overweight because of an obesogenic environment, but also had susceptibility to being overweight due to factors of children's satiety

levels and their responsiveness to food cues. Here, children with low satiety levels and high responsiveness to food cues were at higher risk for obesity.

Johnson and Birch (1994) investigated parental adiposity levels and mother's feeding habits of their children and the children's satiation levels. Johnson & Birch (1994) postulated regardless of the mother's adiposity, when given healthy choices, children had the ability to self – regulate their food intake based on food choices and were able to maintain healthy weight levels with decreased fat stores. Gardner and Rhodes (2009) in their study of pre and postnatal disruption in appetite regulations theorized that as secretions and sensitivities to insulin increased, both adipose tissue and fat cells increased causing the propensity for childhood overweight and obesity.

Endocrine Disorders

Researchers also cited increased endocrine disorders in urban obese children (Kopelman, 2000, Landrigan, Garg, & Droller, 2003). Child populations showed increased thyroid and metabolic disorders exacerbated by weight gain, which ultimately interfered with metabolism binding and thyroid functioning (Galvez, Frieden, Landrigan 2003; Schmid 2004). Metabolic disorders also led to malaise, inactivity and subsequent weight gain in children (Schmid, 2004). A research study by Lustig (2006) suggested that neuroendocrine systems tended to become hyper-insulinemic in obese children which led to hedonistic types of eating patterns where eating behaviors were stimulated by the neuroendocrine systems and the satiety turn-off switch was inhibited. Similarly, Yanovski et al. (2000) postulated that obese children showed hyperinsulinemia and leptin resistance and showed elevated satiety levels. Yanovski et al. (2000) concluded that there was a decrease in children's energy expenditure the higher their

lipids levels (fats) were in their blood. This type of hyperlipidemia was also considered a precursor for heart disease (Hansen 2007).

In a study on glucose and lipid metabolism in children, Caprio (2002) used magnetic resonance to measure visceral and subcutaneous fat. Caprio (2002) surmised that the pattern and distribution of both visceral and subcutaneous fat increased glucose intolerance and metabolic deficiencies. Alberti, Zimmet, Shaw (2007) characterized the body composition of children with endocrine related metabolic syndrome as having a waist circumference descriptive of insulin resistance and GH deficiency. Here, subjects had abdominal adiposity, excess weight and higher fasting triglycerides than matched controls. Nathan & Moran (2008) study on pediatric metabolic deregulation in obese children found a clustering of diseases acquired by this population to include ovarian polycystic and fatty liver disorders.

Genetic Predispositions

Russell et al. (2001) surmised that genetic and metabolic predispositions added to childhood obesity when given favorable biological conditions that further contributed to non-modifiable genetic factors. The physiologic risk factors that influenced the gene encoding mechanisms to control and regulate body weight were irregular in genetically prone children. Russell et al. (2001) suggested that those children were genetically predisposed for weight gain particularly when paired with indulgent or obesogenic environments. Morgan et al. (2002) argued that adiposity and adipose tissue had a genetic component with predictive values for obese children that put them at greater risk for metabolic - imbalance and poor energy expenditures. Anderson & Whitaker (2009) found in their research on genetic epidemiology that genetic predispositions for obesity in children were generally exacerbated by obesogenic

exposures thus attributable to population – specific risk for obesity. Whitaker and Dietz (1998) postulated that developmental - intrauterine programming on energy sensing and energy expenditure; particularly where extra energy is stored due to excess, increased the propensity for postnatal obesity and comorbidities. Likewise, Small, Anderson & Melnyk (2007) purported that exogenous influences in the environment increased the propensity for the predictive value of adiposity and BMI ≥ 95% in urban children and those factors were beyond the child's control.

Biological Obesogenic Agents

Empirical studies on population genetics and obesity theorized that the relationship between obesity and genetics was measured by biological load (NIHCM Foundation, 2005; Salsberry & Reagan, 2005; Schwartz, 2003; Spear et al. 2007; Surgeon General, 2001; Teachman & Brownell, 2001). The biological load or genotype hypothesis (Neel, 1950) described the multiple genotype conditions of a population per its highest level of fitness to the emergence of genetic epidemiology. Frayling, Timpson & Weedon, et.al, (2007) suggested in their study on fat mass and obesity (FTO gene) that this particular gene underwent genetic variation influenced by the environment. Frayling, et al. hypothesized that obesogenic environments also predisposed at risk populations for polygenic obesity.

Rankinen & Bouchard (2008) study on heterogeneity and weight regulation suggested that biological predispositions to obesity were genetically expressed. Hence, a gene verses environment synergy occurred where weight would either become regulated or deregulated. Rankinen & Bouchard (2008) postulated that the gene – environment interactions influenced gene – nutrition and gene – physical activity responses. Lenard & Berthoud 2008 similarly

posited that obsegenic gene – environment interactions caused genetically – driven adiposity and inflammation which resulted in energy imbalance (Walley, Blakemore, Froguel, 2006).

Salsberry & Reagan (2005) contented that multiple biological factors influenced the obesity tendency in children that included the prenatal health of the mother, pre - pregnancy obesity, early exposures in utero and the overall quality of the intrauterine environment. Martonell, Stein and Schroeder (2001) submitted that high birth weights when related to gestational diabetes and the prenatal health of the mother was correlated with increased adiposity and early childhood obesity. Similarly, Dietz (1998) surmised that low birth weights were associated with overweight and obesity in children especially if prenatal under nutrition was prevalent in the first trimester. The "thrifty phenotype" concept coined by Hales and Barker (1992) caused conditions in utero that prompted the fetus to overcompensate and go into survival mode. Hence, intrauterine adaptations and changes that occurred at the metabolic and physiological levels were often to balance deficient prenatal sustenance. Hales and Barker (1992) surmised that this type of overcompensation while in utero led to permanent alterations in metabolism and adiposity later in life.

Energy Balance

Energy Intake and Energy Expenditures

Williams, Bing, Cai, Harrold, & Liu (2001) postulated in their research that the hypothalamus regulated feeding behavior and energy expenditure through a reciprocal interchange between satiety receptors and orexin neurons. Williams, Bing, Cai, Harrold, & Liu (2001) concluded that when the orexin neurons were imbalanced they would stimulate acute feeding behaviors. Variations in daily food intake and energy expenditure caused variable

homeostatic energy balances (Williams, Bing, Cai, Harrold, & Liu, 2001). According to DeLany, Bray, Harsha, Volaufova (2004) total energy expenditure [TEE] based on energy intake or food consumption decreased in obese children whose caloric intake was over a certain threshold. This notion suggested that the lower the expenditure of energy the higher the tendency for weight gain. The combination of excess calories over basal set - point also caused an energy imbalance that interfered with a stable base weight (DeLany, Bray, Harsha, Volaufova, 2004).

Anderson & Butcher (2006) used the metabolic rate measure [METS] to determine the energy expenditure of children during play and determined that it was equivalent to a metabolic rate of 1 MET per 1 kilocalorie per kilogram of the child's body weight per hour of activity (Savage, Toth & Ades, 2007).

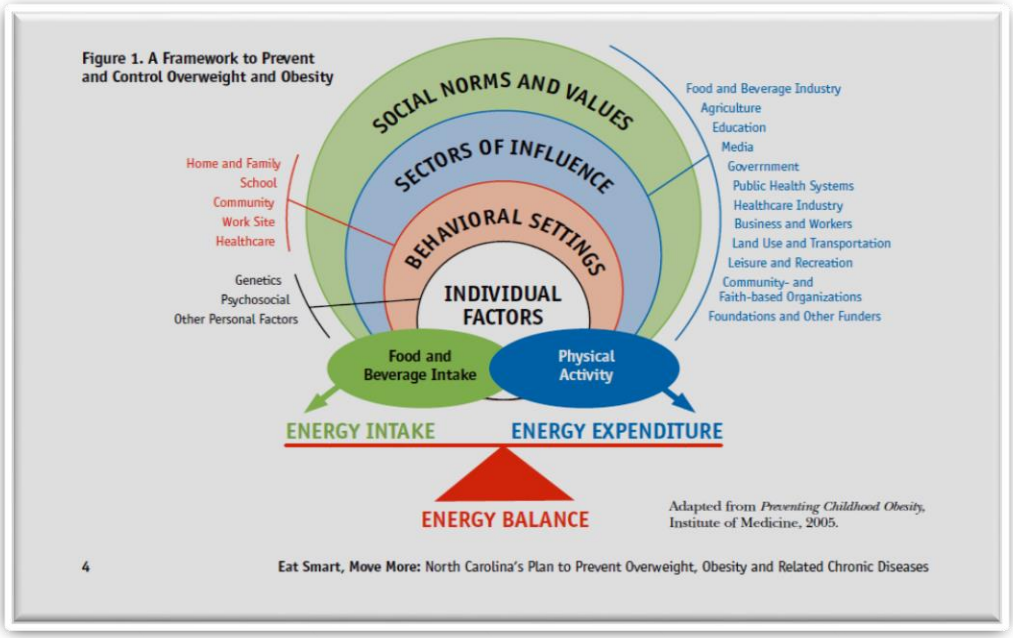

Figure 2: A Framework to Prevent and Control Overweight and Obesity. From Done, B. Eat Smart, Move More: North Carolina's Plan to Prevent Overweight, Obesity and Related Chronic Diseases.

Along the same thought, Kumanyika (2007) posited that the degree of difference between energy - intake, energy expenditure and energy decline showed an inverse relationship in the census study because of unmitigated harsh conditions found in urban neighborhoods. Temple, Legierski, Giacomelli, Salvy, Epstein (2008) found that overweight children responded to food as reinforcement and thus had higher energy intake levels than their leaner, non-overweight counterparts. Children in this particular study tended to respond to food as reinforcement even when given the choice to play hand-held video games or to read their favorite magazines or books. Figure2. showed the balance between energy intake and energy expenditure. Conversely, an imbalance in one would cause either weight gain or weight loss in the other.

Homeostatic Base – Weight

Notwithstanding, the clinical threshold for children's homeostatic base - weight equated with caloric intake not exceeding their energy expenditures (Burdette & Whitaker, 2005; Crowley, 2008). According to Ogden et al. (2002) children from lower SES who had base - weight irregularities also exhibited metabolic sluggishness, both precursors for lower basal metabolic rates [BMR]. Moreover, those children had lower basal set points and lower energy expenditures due to long periods of inactivity (Caprio & Tamborlane, 1999; Goran, 2001; Flegal & Troiano, 2000; Ogden et al. 2002; Rosenbaum & Leibel, 1998; Schmid 2003). The solution for weight regulation in children was a static basal metabolic rate and equivalent physical activity; both considered critical since imbalanced intake to expenditure was metabolically expressed through weight – gain (Caprio & Tamborlane, 1999; Molnar, Gortmaker, Bull, Buka,

2004; Popkin, Duffey, & Gordon-Larsen, 2005; Schmid 2003; Sothern et al. 1999; Swinburn, Jolley, Kremer, Salbe, Ravussin, 2006).

Neighborhood – Level Conditions

The Built Environment: Structures, Access, Proximity and Obesity

The design of the environment and the spatial use of land was vital deterrents to weight gain in children when designed to increase children's activity levels (Corti, Donovan, 2003). The built environment included those factors of the environment that influenced the ability for children's natural outdoor activity (Lopez, 2004). As part of the childhood obesity agenda, the built environment implied that environmental inequities worsened the health and weight of children (Saelens, Sallis, Black, and Chen, 2003)

When the environment had favorable conditions and deliberate design those factors were considered amenable to increased physical activity. The design of the neighborhood was noted in the literature to influence the energy expenditure and physical activity of urban children and was considered interrelated with children's optimal healthy weight (Chen 2003; Smart Growth America [SGA], 2006). The design of the neighborhood was considered either supportive and responsive or unsupportive and unresponsive to the physical activity needs of children (SGA, 2006). Thus, as a unit of study, the neighborhood, either intentionally or unintentionally encouraged social integration or disintegration by virtue of its design (Perdue, Stone, Gostin, 2003; Saelens, Sallis, Black, Chen 2003; SGA, 2006). Likewise, the land – use and layout of the neighborhood contributed to perceptions of safety and security akin to children outdoors activity (Perdue, Stone, Gostin, 2003; Saelens, Sallis, Black, Chen 2003; SGA, 2006).

Comparably, urban neighborhoods whose layout was not well planned were mostly incompatible with promoting children's outdoor activity (Corti and Donovan, 2003; Perdue, Stone, Gostin, 2003; Saelens, Sallis, Black, Chen 2003; Schimidt, 2003; Schmidt, Freedson, Chasan-Taber, 2003). Several studies revealed that health - compatibility from one neighborhood to another were likened to the socio-ecological theory that espoused opportunistic relationships between consequential health behavior, energy balance and the environment (Bronfenbrenner, 1989; Brownell, 2002; Saelens, Sallis, Black, Chen 2003). Researchers equally challenged that environmental aspects of the built environment such as neighborhood degradation, congestion, traffic and sprawl caused children to travel to most destinations by cars due to safety concerns, thus impeded their natural abilities to walk or ride bicycles (Bull & Buka, 2004; Molnar, Gortmaker, Yancy, Kumanyika, 2007; Schmid 2003).

Brownell (2002) construed that obesogenic (toxic) neighborhoods lacked designs with sidewalks, lacked open – unobtrusive spaces for children to play and had widespread degradation that caused increased sedentary activities in children as evidenced by increased and excessive use of computers, electronic games and television - viewing (Anderson & Butcher, 2006; Egger, Raza, 1999; Sothern et al. 1999; Swinburn). Researchers agreed that obesogenic neighborhoods adversely influenced the energy expenditure levels in children (Brownell, 2002; Caprio & Tamborlane, 1999; Ewing, 2005; Risica 2004).

Since urban neighborhoods were described in the literature as disproportionately affected by unfavorable environmental designs, researchers contended that salient conditions such as outdoor space, sidewalks, and playgrounds were needed to promote natural energy expenditure and outdoor play (Anderson & Butcher, 2006; Gordon-Larsen, 2005; Kumanyika & Grier, 2006;

Popkin, Duffey). Contrarily, sprawl and high – density designed neighborhoods reduced neighborhood "walkability" and increased safety issues of safety (Krizek, 2000; Ewing, Pendall, Chen 2002; Powell, Slater, Chaloupka, 2006). Lovasi, Hutson, Guerra, Neckerman (2009) study on marginalized population argued that obesity producing environments had multi-casual variability including poor neighborhood aesthetics. Other influences on obesity included an integration of socio – economic, demographic and socio-ecological factors.

Resource Scarcity

The literature supported the concept that resource surplus and resource scarcity had correlation with BMI levels (Björntorp, 2001; Yngwe, Fritzell, Lundberg, Diderichsen, Burström, 2003). Miller (1995) explained that the praxis for resource scarcity was associated with economic status that forced specific subgroups to make decisions based on necessities as opposed to superfluous choices. Powell, Slater, & Chaloupka (2004) postulated that scarcity of resources limited choices and restricted decision – making power within certain subsets of families. Miller et al. posited that resource scarcity was better understood when applied to shortages in terms of food choices. Rothenburg (1988) described resource scarcity as oppressive at its most fundamental level. Hence, neighborhood residents continuously forced to make choices between basic necessities of daily living and quality of life amenities experienced distress and health impairments such as disturbance in the autonomic nervous system and disturbance in the body's homeostasis or allostasis (Björntorp, 2001).

Low SES populations marginalized by resource scarcity also characteristically experienced barriers to adequate housing, healthy food, childcare, healthcare, education and transportation (Popkin, Duffey, Gordon-Larsen, 2005; Yngwe, Fritzell, Lundberg, Diderichsen,

Burström, 2003). Similarly, inequities in resources posed certain sanctions that kept urban residents from membership in organized recreational or fitness activities, from entry into certain health care organizations and had disqualifying implications on quality of life choices (Popkin, Duffey, Gordon-Larsen, 2005; Stevens, Seid, Mistry, Halfon, 2006). Schrecker (2008) espoused that resource scarcity was an artificial and consequential construct of policy choices and decisions made by public institutions that geographically produced resources and blocked access to those resources.

Economic Costs of Obesity

Adding to this public health quandary was the health care costs for obesity (American Academy of Pediatrics 2003**;** Caprio, 2006; Dietz, 1998). The burden to U.S. tax – payers for the general medical treatment costs of obesity related medical treatment was estimated at $180.00 annually per person. Likewise, in 1995 the total direct and indirect cost for obesity medical treatment was estimated at 99.2 billion dollars (Finkelstein, Fiebelkorn, Wang, 2003).

On the other hand, the estimated annual expenditure for obesity-related medical interventions for children and youth from1997 to1999 was an estimated 127 million dollars – rising some three – fold from a 35 million dollar annual cost seen twenty years earlier (Haas, Lee, Kaplan, Sonneburn, 2003; Wang & Dietz, 2002). The annual cost for treating urban obese children who were recipients of Medicaid Insurance neared $6,700 per child. The collective national costs to Medicaid Insurance was at 3 billion dollars as compared to an estimated annual cost of $3,700 per child or a national cost of 11 billion dollars for similar treatment collectively paid by commercial insurances (Finkelstein, Fiebelkorn, Wang, 2003; Marder & Chang, 2006; Thomson Medstat Research Brief 2006; Wolf & Colditz, 1998).

Approximately 50% of the medical cost for obesity was offset by the public sector and one half of overweight and obesity attributable medical spending was the responsibility of the public sector (Medicaid and Medicare) (Finkelstein, Fiebelkorn and Wang, 2003). Blue Cross / Blue Shield of North Carolina spent an estimated $83 million in 2003 for its members who required medical treatment for obesity (Harris, 2006). The medical cost for caring for obese children and youth in North Carolina based on 2003 figures was an estimated $16 million; this figure was both for direct and indirect costs (Finkelstein, Ruhm, Kosa, 2005).

Prescription drug use and healthcare expenditures were found to be particularly greater for children and adolescents who were obese than for those children and adolescents who were at healthy weight (Hampl; Carroll; Simon; Sharma, V. 2007). Inpatient hospital utilization for obese children on Medicaid was 2 to 3 times higher than for their healthy weight peers counterparts with private commercial insurances (Thomson Medstat Research Brief, 2006). Duke University Medical Center (2012) researcher Eric Finkelstein surmised that 42% of adults will be obese by 2030. This measure was based on a data from the Behavioral Risk Factor Surveillance System and from the Bureau of Labor Statistics and projected medical costs would be upwards of $550 billion.

Trasande & Chatterjee (2009) longitudinal study on utilization and expenditure data of children ages 6 - 19 (N = 19,613) and with BMI for overweight and obesity concluded that obesity related hospitalizations for the study populations was $237.6 million in 2005. When extrapolated just for outpatient services associated with overweight and obesity to include pharmaceuticals, costs were $14.1 billion (Trasande & Chatterjee, 2009).

Neighborhood crime & safety as barriers to outdoor activity

Crime impact on children's health

Weir, Etelson, and Brand (2006) surmised from their study on children's physical activity and crime that children who resided in urban neighborhoods had fewer opportunities for outdoor activity. Weir et al. (2006) studied and compared inner city children (*N*=204) ages 5 - 10 with like - aged suburban children (*N*=103) and surveyed their parents on how often their children participated in opportunistic outdoor activities. Weir et al. (2006) concluded that inner city children spent less time in outdoor activities relative to their parent's nervousness about neighborhood gang activity, other children's aggressive behavior and neighborhood crime occurrences.

Although, physical activity was one of the most agreed upon ways to alleviate childhood obesity, crime was one of the most common reasons why parent's kept their children indoors (Lumeng, Appugliese, Cabral, Bradley & Zuckerman (2006). Parent's actual fear of neighborhood crime appeared to largely influence why urban children had limited chances for outdoor activity (Gómez, Selva and Sallis, 2004; Lumeng et. al., 2006). In addition, the fear of crime also influenced parent's lifestyle decisions concerning their children (Gómez, Johnson, Selva, and Sellis, 2004).

Parental perceptions on safety

Parental perceptions of neighborhood safety were an overarching theme in the literature particularly in urban neighborhoods (Burdette & Whitaker, 2005; Gomez et al. 2004; Weir, Etelson, Brand, 2006). Parent perceptions about safety and security impacted children's ability to adequately expend energy through outdoor activity and subsequently affected BMI due to

calorie overload (Burdette & Whitaker, 2004; Lumeng et al.; 2006; Weir, Etelson, Brand, 2006). Timperio, Crawford, Telford, Salmon (2004) and Stafford & Marmot (2003) surmised that parental perceptions of neighborhood safety adversely influenced the use of neighborhood amenities such as parks, recreational facilities and bike trails by their children.

Additionally, Molnar, Gortmaker, Bull, and Buka, (2004) Harrison, Gemmell, & Heller, (2007) and Wilson et al. (2004) argued that parental perceptions on safety and security were more apparent in children whose families met the SES descriptors for poverty. Gomez et al. (2004) similarly asserted that parents of urban children whether they perceived their children were directly or indirectly exposed to violent crimes were less likely to let their young children play outside unattended. Gómez, Johnson, Selva, & Sallis, (2004) also concluded in their cross – sectional study on adolescent outdoor physical acivity [OPA] (N = 166) and crime / safety that adolescent's perceptions of neighborhood safety produced mixed results and surmised that the parent's of adolescents may be the better judges of safety for OPA on behalf of their adolescents relative to crime rates. Lumeng et al. (2006) random sample of 768 children also showed association between parent's perception of safety, OPA and BMI levels in children. Additionally, Satterthwaite (2014) suggested that safety posed risk to maternal health since the mother's own exposures to and her perceptions of violence were harmful to her health and to her ability to adequately provide for the safety of her children

Parental concern about neighborhood safety was a critical factor when considering if young children were allowed to play independently outdoors since the decision – making for outdoor activity was generally left with the parent (CADS 1999; Copperman and Jacobson 2004; Drentea and Lavrakas; Duke, Huhman, Heitzler, 2003; 2000, Kaplan et al. Lumeng et al. 2006;

Kumanyika and Grier 2006; Sallis and Glanz 2006). The literature showed that parents who kept their children in indoors because of heightened concerns about neighborhood safety created an on-going energy – imbalance in their children whereby the children's weight proportionately crept towards overweight and obesity with increased indoor sedentary activity (Burdette and Whitaker, 2005; Farley, Baker, Watkins, Johnson, and Webber 2007; Harrison, Gemmell, Heller, 2007).

Access and proximity to parks, playgrounds and recreational facilities

The favorably built environment, its homes, schools, parks, roads, playgrounds, environmental conditions and aesthetics were described in the literature as essential for fostering outdoor activity for children (Moore, Diez -Roux, Evenson, McGinn, Brines, 2008). Inasmuch as the environment accommodated the unique needs for children's social integration, those children were able to be active, healthy and lively members of their neighborhoods (Kogan, 2010). Outdoor activities were presumed to be the primary venue for social integration to occur, though such decisions were almost always under the control of the parent(s) or caretakers (Singh, Siahpush, Kogan, 2010). Early childhood outdoor play and activity should typically happened in parks, playgrounds, fenced yards for this population (Singh, Siahpush, Kogan, 2010). It is the inequitably distribution or absence of parks and play – areas in urban neighborhoods that was notably obvious in urban neighborhoods (Moore, Diez -Roux, Evenson, McGinn, Brines, 2008; Singh, Siahpush, Kogan, 2010).

Roemmich, Epstein, Raja, Robinson & Winiewicz (2006) study of 4 to 7 years old children (*N*=59) wearing accelerometers construed that the more dense the neighborhood the less likely there were neighborhood parks and recreational centers. However, neighborhoods with

dense housing and with parks had higher rates of children playing outside (Singh, Siahpush, Kogan, 2010). Kuo, Bacaicoa, & Sullivan (1998) stipulated that lack of urban greenery or green space in urban neighborhoods produced feelings of insecurity and negative perceptions of the neighborhood. On the contrary, planned landscaping, greenery and tree spatial topography were considered aesthetically pleasing and welcoming in urban neighborhoods (Kuo, Bacaicoa, and Sullivan, 1998).

Health Behavior

Parent Health Behaviors

According to Serra (1999) the physical activity - levels of parents tended to parallel the activity levels of their children. Hammer (2004) retrospective study of 150 children ages birth to nine ½ years old stipulated that the greatest indicator of childhood obesity was parent's obesity. The fitness of the parents, along with any deep – rooted cultural and ethnic indicators, also influenced the food habits, activity and health behaviors of children. The more active and fit the parent in spite of any cultural or ethnic markers the more active and fit the child (IOM 2004; Safer, Agras, Bryson and Hammer, 2001). Whitaker, Wright, Pepe, Seidel, and Dietz (1997) surmised that obese parents of both obese and normal weight children more than doubled the likelihood that their children would become obese adults.

Contrarily, Taveras, Gortmaker, Mitchell and Gillman (2008) postulated that parents with higher confidence levels were better decision – makers about making changes to their children's diet and eating behaviors, regardless if the parent was overweight (Wofford, 2008). Bluford, Sherry & Scanlon (2007) surmised from their study on obesity intervention programs designed for children younger than 6 years old that the inclusion of the parent was primary for the

successful behavioral changes desired in those children. Galuska, Will, Serdula, & Ford (1999) denoted in their study of 12,835 obese patients ages 18 and older with BMI >30 kg/m^2 and who were either a parent or non-parent, that 58% of them were never advised by health professionals to lose weight, diet or exercise. Hence, it was probable that a parent or adult caretaker who was obese to have a child (ren) who was also obese.

Food in-take behavior

Children were described as being currently heavier than seen in past years partly due to their easier access to high-density - high caloric foods, snacks, and beverages (Anderson & Butcher, 2006; Andersen et al.1998; DeLany et al. 2004; Luke, Dugas, Kramer 2007; Popkin, Duffey & Gordon-Larsen, 2005; Schneider & Brill 2005; Teachman & Brownell, 2001). Societal practices that influenced overeating and under activity in children were depicted in the literature as contributory to the dramatic upswing in early childhood obesity (Beaumont et al.; Chasan-Taber, 2003; Corti et al.; SGA, Librett, Yore, Schmid 2003; Schmid, Freedson). Passive activity, eating energy dense foods, fats and sugar were often described in the literature as reasons for early childhood obesity, as were cultural and ethnic food-habits and beliefs, genetic predispositions and other bio-ecological factors (AAP 2003; DeLuca, Counts 2001; Risica 2004; Young-Hyman, Schlundt, Herman). Kumanyika (2007) surmised that the environmental framework in which appetite patterns were practiced must be studied particularly for urban obese children since those added to the trajectory of how they became obese.

According to Cullen, et al. interactions among variables in the environment influenced patterns of eating and activity and therefore challenged implementing effective interventions (Bronfenbrenner, 1989; Epstein, Paluch, Gory and Dorn, 2000; Kumanyika 2007; Schmid 2003;

Schneider and Brill 2005). In addition, obese children's unhealthy eating practices were influenced by fast food and convenience store food items that were commonly low density, high fat and low -nutrient dense food products (Golan, Stewart, Kuchle, Dong, 2008; Fox, Pac, Devaney, Jankowski 2004; U. S. Census Bureau 2010). Davidson and Birch (2001) suggested that the child's dietary pattern was formed and influenced within the context of the neighborhood's resources and the parent's dietary patterns.

Hence, children living in households where the parent's or caretakers consumed and/or preferred certain food types and thus modeled those habits for their children who came to prefer and consumed those same types of food (Davidson & Birch, 2001; Fisher, Mitchell, Smiciklas - Wright & Birch, 2001). Davidson and Birch (2001) in Figure 4: posited that ecological systems influenced the parent's food choices which were further influenced by the parent's nutritional knowledge, the types of food that were available and accessible to them and their general parenting style and associated family and cultural influences.

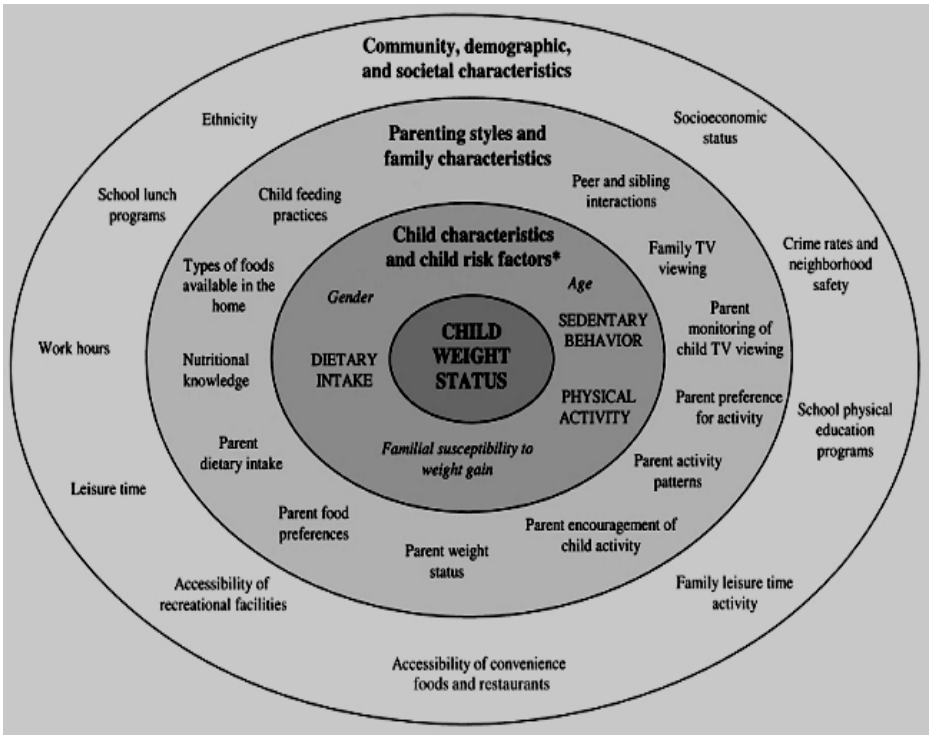

*Figure 3***:** From the Ecological Systems Theory (EST). "From Childhood overweight: a contextual model and recommendations for future research," by Davidson and Birch, 2001, *Obesity Reviews, 2*(3):159-171. doi:10.1046/j.1467-789x.2001.00036.x
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School Nutrition and Physical Activities

The literature supported the national economic trend that forced cuts to a variety of school programs in lieu of competing priorities (Popkin, Duffey, Gordon-Larsen, 2005; Wechsler, Brener, Kuester & Miller, 2001). Schools nationwide dissolved non-academic extracurricular activities like physical education and sport programs due to budgetary cuts (Popkin, Duffey, Gordon-Larsen, 2005). In addition, children participated in similar sedentary activities at their schools as they did in their homes: less moderate, less vigorous, less energy

expending such as sitting, participating in table games, participating in screened – based activities like computers, video games and watching video were thought to contribute to obesity in children (Pate, O'Neill & Lobelo, 2008).

The types of foods available to children in school were challenged for their nutritional value (Hayman et al. 2004). The nutritional value of school food products were considered as much of a health – risk factor to children's ability to maintain healthy weight as were the diminishing opportunities for them to expend energy through physical activity and exercise especially since children spent 50% of their awake time in school (IOM, 2004; Sallis et al. 2001; The American Academy of Pediatrics 2003). Hernandez, Francis, Doyle (2011) found in a longitudinal, secondary data analysis on urban children in national free lunch program that urban girls who participated in those lunch programs tended to have rapid weight – gain and increased BMI levels.

In addition, an estimated 8 million children in 2005 participated in the national School Breakfast Program and an estimated 28 million were enrolled in the National School Lunch Program. Both programs required children to meet the eligibility income - poverty threshold that 67% of elementary school children met (United States Department of Agriculture, 1990 - 2008). Based on those statistics pediatricians and health care professionals throughout the literature agreed that schools needed to re-prioritize dietary and activity opportunities for school-age children particularly for urban children in effort to battle childhood obesity (Hayman et al. 2004). Budgetary constraints, in combination with a food environment of high caloric lunches and food - choices, also were viewed as prescriptive for low energy expenditure and weight gain in

children during school hours (Cullen, Weber, Eagan, Baranowski, Owens, and Moor, 2000; Wecshler, Brener, Kuester, and Miller, 2001;).

Researchers recommended that formal physical education programs be re-instituted and more nutritious food choices be offered to help eliminate secondarily weight gain, improve academic performance and socialization skills and to enhance the overall health conditions of children (Whitaker, Wright, Finch & Psaty, 1993). Likewise, the National Institute of Medicine (2005) recommended that schools and families educate school – age children on the connection between energy expenditure and healthy – weight and teach them about the health benefits of making healthy food choices and about participating in robust, rigorous activity for a minimum of 30 minutes daily (Stubbs, Giles-Corti, Engelhard & Milat, 2003).

Early Childhood Obesity in North Carolina

One of the 38 priority areas of The Healthy People (2020) initiative included objectives on nutrition and weight status (The Healthy People, 2020). A number of The Healthy People (2020) nutrition and weight status goals for children were adopted by North Carolina to include:

- Reduce the proportion of children and adolescents who are overweight or obese.
- Increase the number of states with nutrition standards for foods and beverages provided to preschool-aged children in childcare.
- Reduce consumption of calories from solid fats and added sugars in the population aged 2 years and older.
- Increase the variety and contribution of fruits and vegetables to the diets of the population aged 2 years and older.

North Carolina was projected to spend a $95 billion on obesity by 2015. The CDC 2010 statistics on early childhood overweight and obesity in North Carolina [NC] indicated a need to

address early childhood obesity since 31.7% of 2 - 4 year old children across the State were obese or overweight (CDC, 2010). Of the 2,278,862 children who lived in North Carolina, 629,791 were under the age of 5 years old (Children's Defense Fund, 2013). Comparatively, a combined 31.8% of 2 - 4 year old children across the State who were seen in Public Health WIC programs and Child Health and/or School Based Health Clinics in 2010 were either obese or overweight (NC-NPASS, 2010). In addition, Durham County, NC, the geographic setting for this study, had a 19.7% obesity rate and a 15.8% overweight rate for the same age group in 2010 (NC-NPASS, 2010).

The Blue Cross and Blue Shield of North Carolina [BCBSNC] Foundation (2010) had begun the Shape NC: Healthy Start for Young Children to promote physical activity and healthy nutrition in licensed childcare centers across all 100 counties in North Carolina. BCBSNC (2010) contributed $3 million towards that initiative and collaborated with The North Carolina Partnership for Children, Inc. [NCPC], the North Carolina State University Preventing Obesity by Design (POD) program and the University of North Carolina Nutrition and Physical Activity Self-Assessment for Child Care [NAP-SACC]. The North Carolina Department of Health and Human Services (2010) stated that over 240,162 children under the age of 5 not enrolled in public schools were enrolled in over 8,000 child care centers across all 100 counties of the State. Hence, the core goals of the BCBSNC's / NAP-SACC collaborative initiative – Shape NC (2010) aimed to increase the numbers of young North Carolina children who started kindergarten to be at a healthy weight. The Shape NC (2010) initiative challenged childcare centers to provide this cohort of children the early experiences of healthy eating and age appropriate physical activity to reduce overweight and obesity (BCBSNC, 2010).

The Statewide initiative, Eat Smart, Move More North Carolina [ESMM-NC] (2010), outlined policy initiatives across the State and promoted opportunities for physical activity and healthy nutrition across the State to include remote, inner city and suburban living areas (ESMM-NC, 2010). The ESMM-NC (2010) had a University Collaborative comprised of both public and private universities in North Carolina that focused on researching disease management initiatives and developed health promotion strategies as best practices for statewide use. Researchers Dunn, Caldwell, Thaxton, Anderson, Hoggard, Thomas and Kolasa (2005) researched strategies to assist North Carolina public schools in making immediate changes to improve nutritious foods and healthy eating for its youth. The North Carolina Family Impact [NCFI] (2011) comprised of policy makers and practitioners proposed that local farmers and early childhood educators partner to provide cost – effective ways to make fruits and vegetables available to young pre – kindergarten children. Additionally, NCFI (2011) proposed that preschool educators and local farmers adopt a joint initiative for mobile farmer's market, preschool and daycare center gardens, and provide lesson plans and fun activities to expose and involve young children to fresh produce.

Beth, Boss, Kolasa, Newkirk, Thomas (2002) who comprised the Blueprint Leadership Team as part of the Shape – NC collaborative on early obesity developed a comprehensive guide to help childcare centers implement healthy eating strategies and physical activities as integral prototypes in their programs. Kolasa & Rickett (2010) founded in their research of physicians in North Carolina proportional differences between the referral habits of primary care physicians who gave advice and recommendations to parents of young children suffering from weight issues and those who did not. Kolasa & Rickett (2010) posited that primary care physicians frequently

failed make referrals for weight - related treatment to dietitians and further lacked a clear concept of the interdisciplinary clinical implications of nutrition on the obesity and overweight of young children in North Carolina (Kolasa & Rickett, 2010).

North Carolina researchers Pratt, Lamson, Lazorick, Parker – White, et al. (2011) surmised that pediatric treatment programs for early childhood obesity neglected to provide integrated - interdisciplinary collaboration between providers. Lazorick, Parker – White, et al. and (2011) founded that the financial, clinical and operational aspects of pediatric obesity services posed barriers for patient's access to comprehensive care when not integrated. Ward, Benjamin, Ammerman, et al. (2008) suggested that the fidelity implementation of the NAP-SACC Statewide initiatives required design improvements to show consistent intervention effects in child care centers addressing early childhood obesity. Moreover, Buescher, Whitmire, & Plescia, (2008) used data from the NC–NPASS and demonstrated correlation between federal Medicaid expenditures and paid claims for obesity related medical services for low income North Carolina youth. Buescher, Whitmire, & Plescia, (2008) further argued that Medicaid expenditures for overweight and obese North Carolina low income youth were associated with comorbidities that were higher than those for normal-weight North Carolina adolescents.

Geographical Information Systems (GIS) and The Built Environment

Unlike this study that used Geographical information systems (GIS) based data as an emerging technology to measure obesogenic and environmental conditions influence on childhood overweight and obesity, there were fewer studies in the literature that used this technology (Nuckols, Ward and Jarup, 2004; Witten, Pearce and Day, 2011; Shaw, 2012). Geographic spatial data of the built environment for purposes of this study used topologically

and mapped coordinates of the build environment to analyze and provide statistical correlations that impeded children's access to their natural outdoor environments (Buliung & Kanaroglou, 2006; Leslie, Coffee, Frank, Owen, Bauman, Hugo, 2007). GIS variables associated with risk for early childhood weight and the built environment included aspects of SES levels, crime, property values, parks, playgrounds, day cares, restaurants, healthcare entities to name a few.

Koleilat (2010) used GIS measures and determined that spatial autocorrelations between the distribution of neighborhood parks, food outlets and conditions were associated with early childhood obesity in 3 - 4 year old children. Zhang, Christoffel, Mason & Liu (2006) also used GIS geospatial mapping and analysis of risk conditions of 412 school neighborhoods and determined where community based resources needed to be initiated to thwart childhood. Witten, Pearce & Day (2011) employed geospatial tools to measure the capacity for neighborhoods to support outdoor physical activity, while McLafferty (2002) suggested in her annual review on public health that practitioners needed to understand how the geography impacted health outcomes and the general relationship between the environment and health. McLafferty espoused that GIS spatial analysis was a useful tool in examining and evaluating the needed health care delivery systems and their locations based projected need and potential utilization. Meier and Kuykendall (2013) used GIS technologies and structured observations and catalogued difference between urban and suburban neighborhoods and concluded that socioeconomic factors contributed to health disparities greater the diversity (minorities) the fewer the resources (Meier and Kuykendall, 2013).

Wridt (2010) used a qualitative GIS spatial analysis with low income inner city children in Denver Colorado and determined that the children perceived risk factors in the environment

that impeded their outdoor physical activity. Wridt (2010) concluded that although children perceived certain risk to be aligned spatially with the built environment, they did not correlate reported crime as an impediment. Wridt (2010) postulated that research on the built environment and physical activity ought to consider the children's perceptions since those help shape their activity levels in their neighborhoods.

Liu, Colbert, Wilson, Yamada & Hoch (2007), likewise postulated in their study using GIS and cumulative logit analysis that the built environment influenced the physical activity and diets which were contributory factors to childhood obesity. Zhang, Christoffel, Mason & Liu (2006) likewise used GIS and hierarchically clustered analysis to assess environmental data sources across several neighbourhoods in Chicago to determine neighbourhoods best suited for physical activity and those neighbourhoods that were not.

Summary

The literature on neighborhood – level conditions and obesity in urban children was resolute and persuasive and argued that of millions of children considered overweight in America, rates were higher for urban children, particularly those living in obesogenic neighborhoods (The National Statistics for Health, 2004). The literature tended to vary on scholarly usage and reference points, but consistently intersected on intermediary variables such as SES, race, obesogenic factors and contributory disease prevalence. Kumanyika & Grier (2006) Stevens, Seid, Mistry and Halfon (2006) Ahn, Juon & Gittelsohn, (2008) summarized that lower – income children were significantly impacted by environmental variables that influenced overweight and obesity and espoused a critical need for practitioners to understand both distal

and proximal neighborhood indices and their roles in influencing high body mass indexes in children.

In addition, association between comorbidities and SES gradients were described as conduits for adult obesity in the census study (Chambers, Duarte, Yang, 2009; Graham, 2005; Flower, Perrin, Vidor & Ammerman, 2007**;** Miller, Rosenbloom & Silverstein, 2004). Researcher also described stressors from environmental factors such as crime, congestion, physical design and socioeconomic scarcities as particularly significant to the disproportionate prevalence of obesity and comorbidity diseases founded in the research population. King and Williams (1995) further surmised that demographic profiles of neighborhoods particular to SES and race acted as proxy for features that adversely affected the health of children. This argument implied that race and SES both directly and indirectly impacted children's health behavior, which was supported by the socio-ecological model used as the premise for this research.

Corin (1995) suggested that urban populations by their collective membership were exposed to adverse stressors and experiences in the environment that impacted health - behavior. When applied to the research population, Corin's (1995) postulation showed correlation between demographic predictors and BMI levels. Likewise, Storey, Foresheet, Weaver & Sans (2002) determined in their review of The National Health and Nutrition Examination Survey III that complex statistical patterns of obesity existed to the extent that SES, race and obesity were intertwined particularly in minority children.

Earlier research founded that urban children experienced quality of life inequalities at higher rates than like - age peers and were described by Stewart & Naples-Springer (2003) as having a pattern of cultural dimensions. Stewart & Naples-Springer (2003) analyzed collective

matrices and catalogued those on demographic indices and when applied to low income children founded them at a health disadvantage. In addition, population specific pathophysiological diseases and comorbidities indicated that race and SES were legitimate indicators in determining prevalence rates in BMI levels ≥ 95% expressly in African American and Hispanic American children (Corin et al. 1995; Kumanyika, 2002; Solomons & Kumanyika, 2000; Flower, Perrin, Vidor & Am merman, 2007).

A number of reviews showed that African American children were exposed to chronic psychosocial stressors more often and were more likely saddled with adverse health outcomes than their like aged Caucasian peers (Damon & Drewnowski, 2008; Gordon-Larsen, Adair and Popkin, 2003; Miech, Kumanyika, Stettler, Link, Phelan and Chang, 2006; Kumanyika and Grier, 2006). Over the long term, exposures to interrelated environmental stressors impeded the ability for those children to achieve healthy weight (Kumanyika and Grier, 2006). The notion that no - single hypothetical model was indicative of childhood obesity; too, that no single neighborhood stressor neither explained the multiple nexus' related to BMI and obesity in urban children, SES was consistent throughout the literature as contributory (Bartholomew, Parcel, Kok and Gottlieb, 2001).

The literature differentiated cultural and demographic dimensions particular to population specific studies on both quantitative and qualitative fronts; many studies however showed both direct and indirect quantitative and qualitative impact on BMI levels (Bambra, Gibson, Amanda, Wright, Whitehead, Petticrew, 2009). Consequently, neighborhood – level stressors were consistently described as having cumulative health effects and health disadvantages that disproportionably affected the census studies (Lutfiyya, Garcia, Dankwa, Young, Lipsky, 2008).

Qualitative schemes depicting adverse neighborhood – covariates were threaded throughout the research and were tied to BMI ≥ 95% in resident children **(**Rundle, Hanson and Chen, 2007). Consequently, race and poverty were used interchangeably as a qualitative condition of urban neighborhoods related to childhood obesity (Gordan – Larsen and Popkin, 2003; Lovasi, Neckerman, Quinn, Weiss, Rundle, Hanson & Chen, 2009**).** Neighborhood quality indicators further showed inverse relationships throughout the research between neighborhood obesogenic features and socioeconomic correlates and childhood obesity (Wang and Zhang, 2006).

Pickett and Pearl (2001) Wang and Zhang (2006) Gordon – Larsen and Popkin (2003) surmised that SES conditions in low – income neighborhoods were correlated with degradation and crime rates that posed inherent safety concerns for parents thus decreasing opportunistic outdoor activity and energy expenditure for children. Environmental constraints on urban children chances to participate in outdoor physical activities were also analyzed and founded to be proportional to available community resources and recreational facilities, which often showed deficient in urban neighborhoods (Kumanyika and Grier, 2006; Powell et al. 2004; Wang and Dietz, 2002).

Likewise, Saelens, Sallis, Black and Chen (2003) concluded that neighborhood walkability was perceived by residents as having favorable conditions that would increase outdoor activity levels such as perceptions of safety, aesthetics, sidewalks and mix – land use. Residents in those types of neighborhoods tended to spend more time outdoors walking and were physically leaner than those residents in neighborhoods less suitable for walking. Parental perceptions of safety, too, was described as being a critical factor in determining whether

neighborhoods were favorable for safe - outdoor physical activity for children (Saelens, Sallis, Black and Chen, 2003; Singh, Siahpush, Kogan, 2010). The strain, stress and perception of safety affixed to poverty – stricken neighborhoods affected the health and activity levels of children to the extent that parents kept their children indoor (Chambers, Duarte and Yang, 2009; Gordon-Larsen, Adair, Poplin, 2003; Gutman, McLoyd and Tokoyawa, 2005; Kumanyika and Grier, 2006; Saelens, Sallis, Black & Chen, 2003; Sallis & Glanz, 2006). Lopez (2004) and Cubbin & Winkleby (2005) summed that neighborhoods with persistent deprivation indices showed a broad spectrum of inequalities that by virtue of children's exposure limited their outdoor activity. Gaps in access and availability of neighborhood parks, playgrounds, recreation centers were supported built environment barriers. The absence of sidewalks, walking paths and greenery produced a propensity for weight gain in children and consequently increased their sedentary activities and lowered energy expenditures (Burdette, Wadden, Whitaker, 2005; Gordon-Larsen and Popkin, 2003; Floyd, Taylor, Whitt-Glover, 2009; Powell et al. 2004; Singh, Siahpush, Kogan, 2010).

Arguably, the literature review had sufficient resources for this researcher that qualified the hypothesis that: 1). Obesogenic conditions in urban neighborhoods are associated with early childhood BMI levels; 2). Obseogenic neighborhood conditions are spatially more prevalent in neighborhoods where young obese children reside; and 3). There are prevalent built environment variables in urban neighborhoods that influence early childhood obesity (Gordon-Larsen, Adair, Popkin, 2003; Molnar, Gortmaker, Bull & Buka, 2004; Nesbitt et al. 2004).

Researchers used a variety of study designs and analytical approaches throughout the literature to support different hypotheses and research questions on childhood obesity. Research

studies conducted by Kumanyika (2008); Gordon-LarsenAdair & Popkin (2003); Ogden, Carroll & Flegal (2008); Swinburn, Jolley, Kremer, Salbe, & Ravussin (2006); Wang (2001); Wang & Dietz (2002); Kumanyika & Grier (2006); Gordon-Larsen, Adair & Popkin (2003) used quantitative regression models to advance this area of study particularly in reference to correlations between environmental variables and the BMI of low – income children.

Accordingly, Wang (2006), used logistical analysis to study the correlation between childhood obesity and socioeconomic status on a more global level.Wang (2006) analyzed data for children aged 6 through 18 years old from the United States, Russia and China and and concluded that the link between childhood obesity and SES had international indications. Kumanyika (2008) used correlation and comparison designs while Gordon-Larsen, Adair & Popkin (2003) used meta-analysis and regression models. Saelens, Sallis, Black, Chen (2003) used ANCOVA analysis and logistic regression to measure the outcome variable and predictor variables in neighborhood "walkability" and neighborhood weight – status differences. Here, Saelens, Sallis, Black & Chen (2003) evaluated environmental self – report surveys of adults (N=107) across two neighborhoods on the neighborhood's walkability. Those researchers surmised through comparison analysis that the weight status and activity levels of neighborhood residents in low – walkability neighborhoods were associated overweight prevalence.

Gordon-Larsen, Nelson, Page, & Popkin (2006) used logistic-regression analyses to test the relationship between access to recreational facilities and physical activity in low income adolcents at the community level. Powell, Slater & Chaloupka (2006) comparatively used multivariate analysis in their study on neighborhood conditions and recreational facilities by zip code and showed the absence of those in African Amercian and Hispanic neighborhoods.

Franzini, Elliott, Cuccaro, Schuster, Gilliland, Grunbaum, Franklin & Tortolero (2009) examined the relationship between the activity levels and obesity of 650 children using cross – sectional analysis with structural equation models in a multisite community based study. Franzini et al. (2009) suggested that social factors were associated with activity levels in the study population and physical factors were adversely associated with obesity in those children. In addition, Perkins, Meekes & Taylor's (1992) quantitative study used regression analysis of resident surveys and environmental assessments (N=412) and explained that the relationship between crime and fear were linked to environmental incivilities.

Weden, Brownell & Rendell (2012) research on early life disparities in childhood obesity used logistic regression models and pooled survey models and determined that there were underlying racial differences in high body mass index between African American and Caucasian children when exposed to early life risk factors as opposed to early life protectors. Weden et al. surmised that those risk or protector factors accounted for the differences between race – related propensities for early childhood obesity. Likewise, Singh, Siahpush and Kogan (2010) used logistic regression models and postulated that there was a relationship between overweight and obesity and neighborhood conditions. To the extent that the food environment was linked to higher BMI levels, Shier & Strum (2012) longitudinal survey surmised that food outlet data when merged with the study population's residential census tracts showed significant association between the number of different types of food outlets and higher BMI levels.

A number of sampling strategies were used in the literature to investigate the causes of childhood obesity in urban children which included regression models, longitudinal and sample studies to measure childhood obesity correlations with SES, ethnic, racial, and demographic

variables, this research emphasized those aspects and also whether neighborhood structural / obseogenic variables were associated with early childhood obesity by virtue of their limitations on opportunistic outdoor activity (Baker et. al., 2006; Cohen, et. al., 2006; Gorden- Larsen, et. al., 2000; Morland, et. al. 2006). This researcher used the analysis of secondary data to demonstrate those burdens of the obseogenic environment on the early onset of obesity in low income children.

A number of North Carolina initiatives were begun in 2010 to address statewide early childhood obesity particularly since the CDC (2010) named North Carolina the 5th highest obese state in the nation with over 31% of its 2 – 4 year olds either overweight or obese. Programs were launched across the State that evaluated and measured, recommended and provided a statewide obesity prevention plan. The BCBSNC Foundation-Shape-NC program (2010), the NAP SACC (2010) and the ESMM-NC (2010) and multiple University and local organizations aimed to provide strategies and policies for regulated standards for childcare centers on obesity prevention and forward best practices that were proposed to increase the activity level and nutrition value of children under 5 years old with the goal for those settings to reach a healthy weight in children by start of kindergarten.

The emergence of GIS mapping as a technology enabled the geospatial assessment of the built environment and offered opportunity for researchers to derive data on obesogenic conditions of the built environment through structured observations of particular domains of the environment overlaid in maps (McLafferty, 2002; Thornton, Pearce and Kavanagh, 2011; Zhang, et al. 2006). Of the 33 quantitative studies used by Davidson and Lawson (2006) in their literature review to determine if the environment influenced children's physical activity, six of

those studies measured qualities of the environment using GIS methodologies and founded positive relationships between the infrastructure of the environment and outdoor opportunistic physical activity and play. Neighborhood conditions such as recreational facilities, sidewalks, and controlled intersections had favorable influence on physical activity while congestion, density, crime, and degradation had a negative impact on children's physical activity levels (Davidson and Lawson, 2006).

The use of GIS data informed and provided demographic analysis to researchers and policy makers on where and when to impose new structures, designs, resources, interventions in the built environment to enhance health and well - being. In addition, GIS spatial statistic software tools such as the Ordinary Least Squares [OLS] and Geographically Weighted Regression [GWR] allowed for convenient statistical testing of the researcher in the literature geospatial hypotheses between conditions of the built environment and early childhood obesity.

Chapter 3: Research Method

The method section provides a synopsis of the study, its design, its study population, ethical considerations, and the approach used in examining associations between childhood obesity and the built environment data analysis.

Study Purpose

The purpose of this research was to determine early childhood obesity influences by examining urban neighborhood conditions and BMI levels greater than or equal to the 95th percentile by zip code in children 2- to 4-year-old from secondary data from the NC-NPASS (2010) Durham County, North Carolina and linking those with built environment data from Miranda, Edwards, Anthopolos, Dolinsky, & Kemper (2012) study from the 2011 Children's Environmental Health Initiative [CEHI] and from the 2010 U.S. Census data that was further overlaid by zip code and mapped using GIS. Neighborhood conditions thought to be obesogenic in urban neighborhoods used in this study included: neighborhood safety, neighborhood conditions, housing conditions, community resources, food and healthcare access, and demographics and were hypothesized to have impact on the healthy weight of children (CEIH, 2011; Census, 2010; James, 2008).

Research Questions

The following research questions and hypothesis directed this research study:

1. Are obesogenic conditions of urban neighborhoods associated with early childhood obesity as measured by BMI $\geq$ 95%?
    - H_{01}: Children living in obesogenic environments have no discernible difference in BMI levels.

- H_{a2}: Children living in obesogenic environments have BMI levels ≥ 95%.

2. Are obesogenic neighborhood characteristic spatially more prevalent in neighborhoods where obese children live?
    - $H_{02:}$ Adverse obesogenic characteristic are not more spatially concentrated in urban neighborhoods where obese children live.
    - H_{a2}. Adverse obesogenic characteristic are more spatially concentrated in urban neighborhoods where obese children live.
3. What are the most prevalent obesogenic neighborhood conditions that are significant explanatory variables that categorically contribute to BMI levels ≥ 95^{th} percentile?
    - H_{03}: Urban children's exposures to neighborhood conditions have no predictive value or increase in BMI levels
    - H_{a3}: Neighborhood conditions have explanatory values that influence the BMI levels in urban children.

I used measures from the meta-analysis CEHI (2011) GIS data in addition to supplemental data from the following sources: the Durham County tax assessor's office, the Durham Police Department, and the U.S. Census 2010 to examine if exposures to neighborhood-level conditions affect the activity levels in urban children as measured by BMI levels at or equal to the 95^{th} percentile.

The BMI distribution of 2-4 year old obese children from the NC – NPASS (2010) records were overlaid by zip code with GIS data by census tract. The assumption was the higher the number of observations of obesogenic conditions from the GIS data, the less likely the neighborhood was conducive to opportunistic outdoor activity. Here, the pooled GIS data was

analyzed by conducting regression analysis to determine assumptions on the quality of the neighborhood for outdoor activity.

Role of the Researcher

I attest that there were no physical or psychological risks to human subjects since I used secondary data to answer questions about whether obesogenic conditions of the neighborhood influenced BMI in the research population. I adhered to ethical guidelines when extracting data from NC-NPASS (2010) and CEHI Community Assessment Project (2011) with attention given to privacy protections of personal information and identifiers. Subjects otherwise had adequate protections from risk since no subject identifiers were linked to the subject population in this study. I was committed to protecting and preserving the privacy of all individually-identifiable health information and to uphold both federal and state guidelines about privacy practices. In accordance, I used only the minimum necessary information to carry out this research study as recommended by Broome, Horton, Tress, Lucido, & Koo (2003) and the Health Privacy Project (2005).

Child-Participants Protections

I assured that the data on 2- to 4-year-old obese children used in this study were not exploited since the data was from secondary data sources. In addition, I believed that ample benefits will be reached from completing this study in combating early childhood obesity. The original data collection followed federal regulations policy and guidelines on the use of children in research, and sufficient practices of beneficence were used in protecting the target population during the original data gathering. I verified that the original child participants in the secondary analysis were neither recognizable nor identifiable and therefore were protected by anonymity

and rules of confidentiality (NIH, 1998). In addition, no direct or indirect contact with child - participants was made for purposes of this study. Ethical clearance from Walden University's Institutional Review Board (IRB) for this secondary data source study was received on June 26, 2013 and approval number 06-27-13-0013697 was assigned to this study. The IRB approval expiration date for this study was on June 26, 2014.

Population and Sampling

The study used secondary analysis of archival data collected by the NC-NPASS (2010) of 2- to 4-year-old children who had received WIC services from the Department of Public Health in Durham County, North Carolina who met the 185% benchmark for the federal poverty threshold and who were considered obese by measure of their BMI levels equal to or greater than the 95^{th} percentile based on gender and age. The aim of this secondary analysis was to determine the extent to which there were any correlations between the high BMI levels ($\geq$ 95%) in low-income early childhood (ages 2 to 4) and obesogenic conditions of the neighborhoods that could explain early childhood obesity.

Demographics of Durham County, North Carolina

Durham, County, North Carolina was the sixth most densely inhabited county in North Carolina (282,641) and had a 108 square mile span in size at the time of this study (U.S. Census of Population and Housing, 2010). Durham County had a 41,500 (20%) increase in population from 2000–2010 to 267,593. Of the total population, 19,815 (7.4%) were under 5 years old in 2010 (U.S. Census of Population and Housing, 2010). Between 2008 and 2013, 18.0% of Durham County residents were below the poverty level, and 19% of children under the age of 18 lived in poverty in 2010 with this percentage increased to 28% in 2014 (Robert Wood Johnson

Foundation, 2010–2014). Durham, North Carolina's demographics population by race and ethnicity are further depicted in Figure 4.

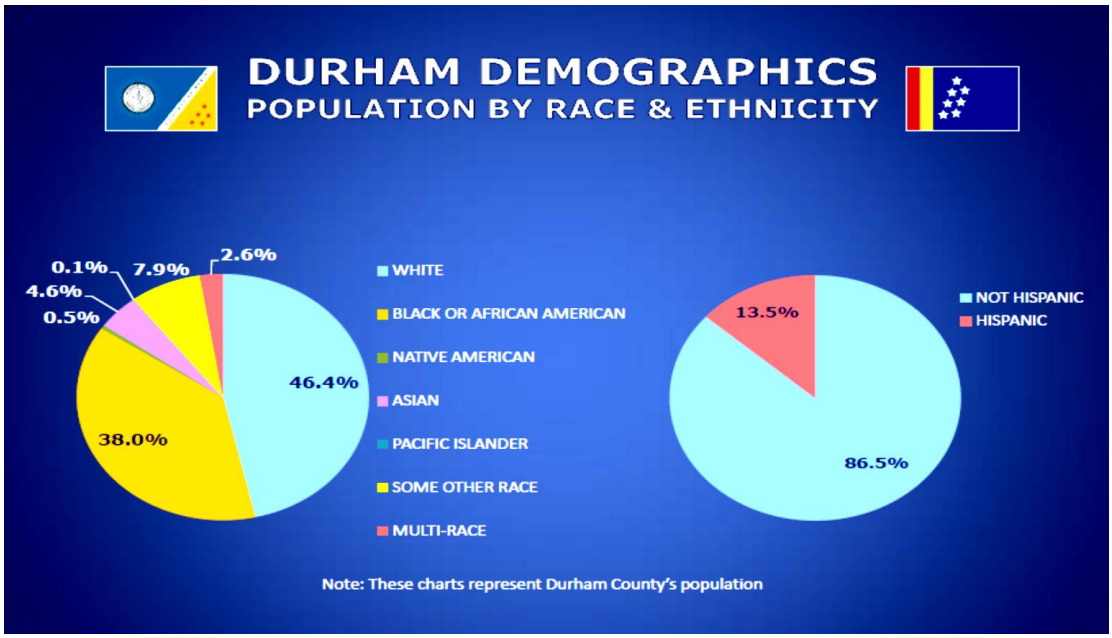

FIGURE 4. Durham Demographics: Population by Race & Ethnicity. From U.S. Census Bureau, 2000 and 2010 U.S. Census of Population and Housing. Retrieved April 14, 2012 from durhamnc.gov/ich/cb/ccpd/Documents/Demographics/RACE-ETHNICITY.pdf

Research Design and Approach

Validity and Reliability

The validity and reliability of the NC-NPASS and its repeated administration since 1995 suggested that this surveillance instrument provided accurate and timely information relevant to children's weight status. The NC-NPASS collected prevalence data from across counties in

North Carolina on the healthy weight, overweight, underweight, and obesity of children in this age group as measured by BMI for age and gender (NC-NPASS, 2010). Data were collected and submitted annually by designated North Carolina public health, child health clinic, and WIC professionals since 1995 as indicated in Figure 5.

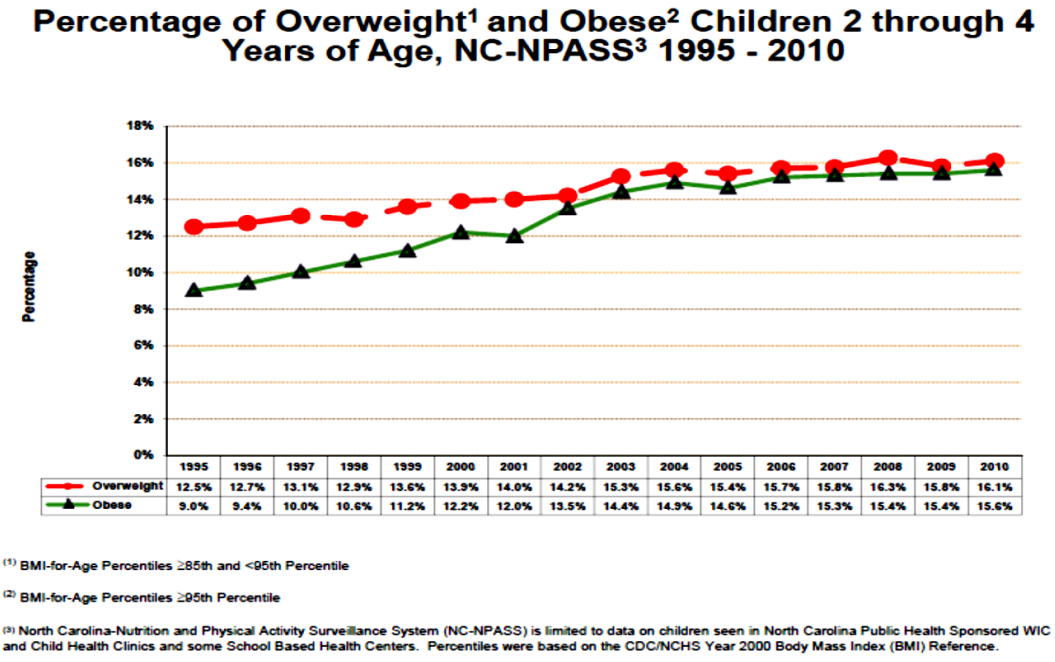

FIGURE 5: Percentage of Overweight and Obese Children 2- 4 Years of Age, NC-NPASS 1995 – 2010. From NC-NPASS. (2010). *Data on Childhood Overweight*. Retrieved October 22, 2011, from http://www.eatsmartmovemorenc.com/Data/ChildAndYouthData.html.

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Of the 105,410 total BMI records for the 2 - 4 year old children collected from counties across North Carolina, 16,478 (15.6%) were considered obese (NC-NPASS, 2010). By race and ethnicity across all counties of North Carolina as depicted in Figure 6: 4,416 (12.9%) White non-Hispanic 2 - 4 year old of both genders were obese; 4,243 (13.4%) non-Hispanic Black children; 208 (16.6%) American Indian; 133 (10.3%) Asian / Pacific Islander and 7,323 (20.4%) Hispanic 2 - 4 year old children of both genders were considered obese (NC-PASS, 2010). Within the Durham County (as the unit of study), 433 children of all races and ethnicities and of both genders were considered obese (NC-PASS, 2012).

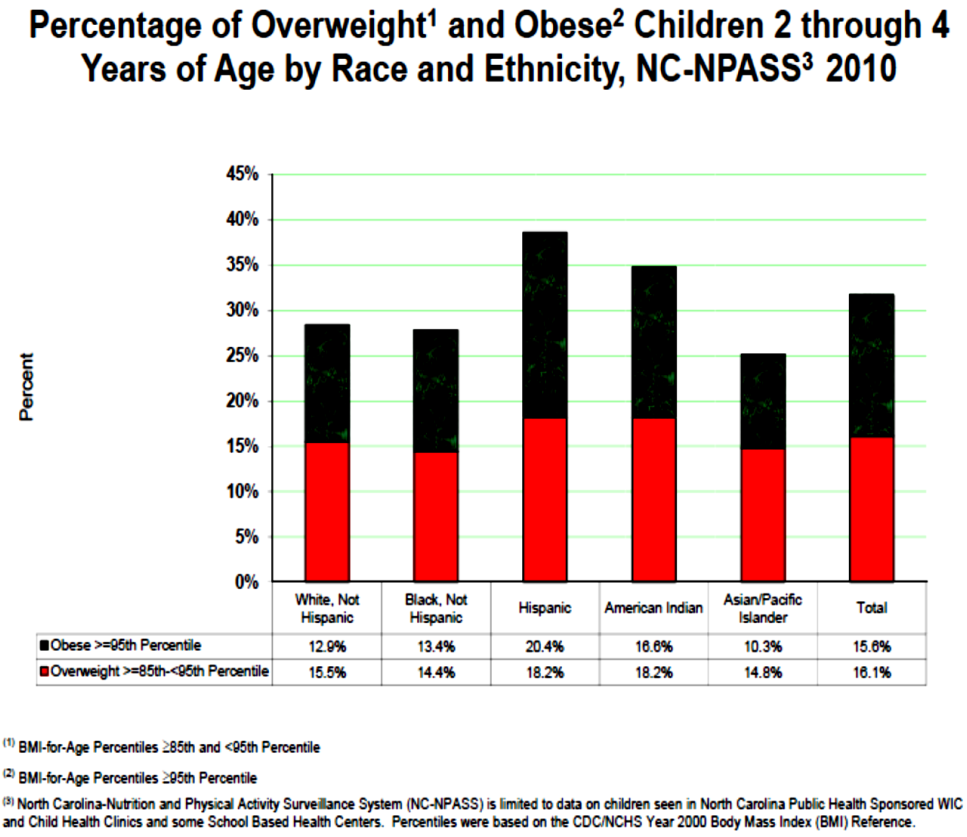

*FIGURE 6***.** The % of Overweight and Obesity Children 2- 4 Years of Age by Race and Ethnicity, NC-NPASS, 2010. From NC-NPASS. (2010). *Data on Childhood Overweight*. Retrieved October 22, 2011, from http://www.eatsmartmovemorenc.com/Data/ChildAndYouthData.html.

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Statistical power calculation analysis

The three areas that influenced statistical power to determine if any real or detectable difference in treatment effect existed in the sample population were the alpha level, the effect level and the sample size (MacCallum, Browne, Sugawara, 1996). The power of the statistical test determined the probability that the test would invalidate the null hypothesis as a type II error as determined by parameters of the alternative hypothesis (MacCallum, Browne, Sugawara, 1996). The type II error also referred to as the false negative rate (β) power is equal to 1- β. The general value for power is .80 (80%).

Table 2

Statistical Hypothesis Testing Theory

Null Hypothesis (H_0)		
Decision	**True**	**False**
Fail to reject H_0	**Correct decision** **$p = 1 - \alpha$**	**Type II error** **$p = \beta$**
Reject H_0	**Type I error** **$p = \alpha$**	**Correct decision** **$p = 1 - \beta$**

Note. Tabachnick, B.G., & Fidell, L.S. (2001). *Using multivariate statistics (4th ed.).* Needham Heights, MA: Allyn & Bacon. Used with permission.

For this research to be considered statistically informative, an a - priori power analysis was used to determine the sufficient sample size needed to run multiple regression analyses of the data to examine the predictive nature between the predictor variables and the criterion variable BMI (Lindell & Whitney, 2001). A statistical power calculator was used to determine

the minimum number of participants needed for the study and was calculated by using parameter values for the probability level (0.01), anticipated effect size (0.15), the desired statistical power level (.80), and the number of predictor variables (6). Based on the aforementioned parameter values, the minimum required sample size for this study was calculated to be (N = 134). Additionally, the probability level was increased to 0.01 from the standard .50 in order to increase the power of the test and to decrease the chance of a type II error; and additionally to increase the chances of rejecting the null hypotheses (MacCallum, Browne, Sugawara, 1996). To increase the confidence level of the study to 95%, this researcher used a larger sample size (N = 433).

Explanatory Variables

Twenty – six explanatory environmental variables were used to determine the influence of the built environment on early childhood obesity as described in Table5. below. Each explanatory variable was considered a direct extension of simply regression and added one term to the structural equation. An interaction between any two explanatory variables where a relationship was determined due to the effects of one on the outcome, based on the value of the other was recorded.

Table 3

Explanatory Environmental Variables

Explanatory Environmental Variables		
Name	*Variable Number*	Variable Description
EdLess9th	(X_1)	Education: Less than 9th Grade
Ed912NoD	(X_2)	Education: 12th Grade; no diploma
EdHighSch	(X_3)	Education: High School Diploma
EdSomColl	(X_4)	Education: Some College
EdAssoc	(X_5)	Education: Associates Degree
EdBachlrs	(X_6)	Education: Bachelor's Degree
EdGradDgre	(X_7)	Education: Graduate Degree
EdPctHSHir	(X_8)	Education: High School or Higher
EdPctBchHi	(X_9)	Education: Bachelor's Degree & Higher
PctVac	(X_{10})	Percent Unoccupied
PctOcc	(X_{11})	Occupancy Rate
PctOwnOcc	(X_{12})	Housing Percent Owner Occupied
PctRentOcc	(X_{13})	Percent Renter Occupied
VALUEMEDN	(X_{14})	Housing: Median Home Value
PctYr39Ear	(X_{15})	Housing: Homes Built 1939 or Earlier
PctYr90_99	(X_{16})	Homes Built 1990-1999
CTRelig	(X_{17})	Religious Institution
CTFFood	(X_{18})	Fast Food Establishment
CTConvSt	(X_{19})	Convenience Store
CTDayCare	(X_{20})	Day Care Facilities
CTGroc	(X_{21})	Grocery Store
CTHealthC	(X_{22})	Health Care Facility
CTHosp	(X_{23})	Hospitals
CTSchools	(X_{24})	Schools (K-12)
CTRecArea	(X_{25})	Park Facility
CTCrime	(X_{26})	Crimes

Note: Description of Explanatory Variables Used in This Quantitative Research Study.

Data Acquisition

The primary step in the method section of this study was to acquire the secondary datasets. Those were needed to perform analysis, conversion and to produce built environment data for GIS mapping. Numerous attempts were made to acquire the full sets of secondary GIS data from the University of Michigan researchers who completed the CEHI (2011) GIS project (secondary data) planned for use in this study. A version of the census data from the CEHI (2011) was eventually obtained by extracting it from an earlier GIS web based map project retrieved from the website http://cehi.snre.umich.edu/share/capmap and was then copied into a GIS format as a layer and stored as a geodatabase file. The data for the CEHI (2011) maps were collected at individual tax parcel levels then aggregated and displayed at a Census block level. The attribute data for each layer (census block) was manually updated to reflect five equal percentile classification intervals to coincide with the previous online web map version located at http://cehi.snre.umich.edu/share/capmap. This data only reflected a range or average of values; therefore no accurate and reliable systematic analysis could be performed on the data. A GIS map was created for each of those layers for purposes of demonstrating the geographic distribution of the specified resources for the study area in Durham County, Durham North Carolina.

All other Census data for this study was acquired from the U.S. Census Bureau American Fact Finder website for the year 2010. The study's sixteen zip codes for Durham County were narrowed down to seven due to the small sample size in those zip codes that could compromise the anonymity of those children who participated in the WIC program in

those zip code areas. Zip Codes having less than ten children in the obese category were redacted (Table 6.). The remaining seven zip codes were displayed in aggregated ZIP Code Tabulation Areas [ZCTAs] which better matched the census tract areas inside Durham County NC. All Census tabular datasets downloaded were for Durham County, Durham, North Carolina (2010) at the census tract level. Once downloaded those tables were imported into the project file geodatabase then associated and joined to the Census Tract polygon features so data could be represented on GIS maps.

Table 4

Number and Percent of Obese Children 2 through 4 Years of age in Durham County by Zip Code, North Carolina-NPASS 2010

Durham County Zip Code of Child's Residence	Obese	Children on WIC at or below 185% Federal Poverty Level	
	>=95th Percentile	Total Number *	Percent Rate
27701	55	294	18.7%
27703	91	447	20.4%
27704	78	422	18.5%
27705	61	273	22.3%
27707	89	423	21.0%
27712	12	69	17.4%
27713	33	197	16.8%
County Total*	433	2,201	19.7%

Note. Chart Created From Data Retrieved from the NC-NPASS 2010 of 2 -4 Year Old Children Who Visited WIC Clinics in Durham County, North Carolina During WIC Certification Visit.

Crime data for this study was obtained from the Durham Police Department's website (http://www.durhampolice.com/crimemapper.cfm).

The data was extracted as text then placed into a database format to import into a GIS. There were 14,471 crime incidents responded to by the Durham Police throughout 2010. Once in GIS, the databases of records were geocoded based on incident address fields and given a spatial coordinate for each record. This iterative process was dependent on the integrity of the address field and of the 14,471 crime incidents recorded, geographic locations were found for 13,323 crime incidents. The incidents were summarized per count of occurrences per census tract, mapped and were associated with each census tract accordingly.

Data analysis testing inside GIS

The intent of this study was to use quantitative regression analysis to account for the multiple explanatory environmental variables that were hypothesized to influence (BMI) obesity. The regression method was also thought to offer the flexibility in examining the data for relationships with more than one explanatory variable (Cohen, Cohen, West, & Aiken, 2003). In this study, observed relationships could be either linear or nonlinear; the explanatory variables could be either quantitative or qualitative in nature and the effects of a solitary variable or several variables were taken into account by using the multivariate regression method with or without effects on other predictive variables **(**Cohen, Cohen, West, & Aiken, 2003).

One of the goals of the project was to use Spatial Statistic tools within Esri ArcGIS 10.2 software to determine relationship and spatial correlation between childhood obesity data and certain environmental variables. The software package was an application product from the Environmental System Research Institute (ERSI). Datasets from the environmental

variables were first projected and imported into a single file geodatabase the Exploratory Regression Tool (ERT) which evaluated the dependent variable (PCTObese95: Percent obese children 2-4yrs old greater or equal to 95 percentile) and informed which explanatory variables could be considered to yield a properly specified regression model. Results from this test consistently showed a very weak model determined to be from the different data sample sizes. Hence, the dependent (secondary obesity data) was only available at a larger/coarse ZCTA area, in comparison to all explanatory (environmental) variable were at a smaller/finer, census tract level. The obesity data (grouping variable) was also only available at a zip code level, therefore too coarse a data to perform proper GIS spatial analysis with the finer census tract level data (test variables).

Summary

In summary, this researcher purported that a multiple variant regression model was the best statistical method to model the risk of neighborhood obesogenic factors on early childhood obesity in Durham, County, and North Carolina. That statistical method was determined over other statistical models because of the inferences that could be drawn from the concurrent observation and examination of more than one outcome variable at a time. The application of multivariate statistics to analyze secondary data from the 2010 NC – NPASS and from the Durham, County 2011 CEHI geographic information systems surveillance was presumed to demonstrate the burdens and correlations of obesogenic neighborhood conditions on urban children's BMI that impeded their natural opportunities for outdoor activity (Gorden- Larsen, et. al., 2000; Baker et. al., 2006; Cohen, et. al., 2006; Morland, et. al., 2006). Consequently, regression analysis testing within GIS Spatial Statistic tools within Esri ArcGIS 10.2 software

indicated that the population sample size was too small and thus too weak to utilize regression outcomes to determine relationship and spatial correlation between childhood obesity data and certain environmental variables. When all datasets were properly projected and imported into a single file geodatabase the Exploratory Regression Tool was used. This tool evaluated the dependent variable (PCTObese95: Percent obese children 2-4yrs old 95th percentile) and reported which candidate explanatory variables could be considered to yield a properly specified regression model. Results from this test consistently showed a very weak model due to the variance from the different data sample sizes. The dependent (obesity data) was only available at a larger/coarse ZCTA area, in comparison to all exploratory (environmental) variables which were at a smaller/finer, census tract level.

Hence, this researcher chose to use the county specific secondary data from Durham, County North Carolina 2010 NC – NPASS and the North Carolina 2011 CEHI GIS surveillance of neighborhood conditions early in the study and confirmed its viability at a point in time. The North Carolina 2011 CEHI GIS surveillance had become unattainable well into the study. The researcher examined the viability of using other children's health datasets such as the National Longitudinal Survey of Adolescent Health, Area Resource File, Behavior Risk Factor Surveillance System, National Health and Nutrition Examination Survey, National Health Interview Survey, National Longitudinal Survey of Youth, Socioeconomic Monitoring System, Youth Risk Behavior, but those were not specific to the Durham County geographic area of study on the built environment.

Child -participant protections in this study were decidedly not needed because this study used only secondary data and public GIS data; no human participants were contacted and or

identified as part of this study. In addition, the statistical power calculation analysis parameter values calculated the minimum required sample size for this study to be (N = 127). Hence, this researcher chose (N = 433) as the subset of 2 – 4 year old children from the NC – NPASS (2010) included in this study. Although the researcher chose to use the higher sample size, that too proved to be too small a number when extrapolated over larger/coarse ZCTA area; the dependent (obesity) was only available at a larger/coarse ZCTA area, in comparison to all exploratory (environmental) variables which were at a smaller/finer, census tract level.

Lastly, this researcher assumed that the secondary data used for this study accurately represented children age 2 - 4 that were at the 185% level of poverty; that the children had BMI greater or equal to the 95 percentile for gender and age; and that the children were subsequently considered obese. The research also presumed that the archived data originally collected had met all of the ethical criteria: respect for person, beneficence and justice (The Belmont Report, 1979). The assumption that confidentialities and protection of child participants had been upheld in this secondary research study was inherent in the nature of the archived data (NIH, 1998). In addition, the use of BMI for age as a standard formula for childhood obesity was purported to be a valid measure that could be replicated across like populations to determine childhood obesity.

Chapter 4: Results

The scope of this project was to use GIS tools and software support research to determine correlations between early childhood obesity and environmental variables within Durham County, North Carolina. I used the GIS geocoding process to organize, visualize, analyze, present, and comprehend complex layers of built environment data that were further overlaid by ZCTA locations in map form. The intention of the GIS process was to explore the public health relational trends between early childhood obesity and built environment variables which were not readily apparent in traditional research on the built environment and early childhood obesity.

The study scope of the GIS project used 7 of the originally intended16 zip code tabulation areas (ZCTA); 27701, 27703, 27704, 27705, 27707, 27712, and 27713. The zip codes 27702, 27706, 27708, 27709, 27710, 27711, 27715, 27717, and 27722 were not converted to ZCTAs since the numbers of 2- to 4-year-old obese children in those geographic areas were too limited with fewer than 10 obese children and were redacted to protect the confidentiality of those children. The intended frequency count of the sample size of 2- to 4-year-old children who resided in Durham County by zip code and whose BMI for age was more than 95th percentile during their WIC Clinic visit in census year 2010 was (N=433), which was reduced to an actual (N=419). Fourteen (N=14) children were redacted due to the discretionarily small geographic zip code area which could have compromised and breeched their confidentiality.

Data from the NC-NPASS (2010) collected in WIC clinics during WIC certification visits in Durham County were analyzed for correlations with geographic environmental variables, defined at the census tract level from the 2010 census bureau and from manually updated GIS

study on the build environment conducted by the Children's Environmental Health Initiative (CEHI) and University of Michigan.

Test for Homogeneity of Variances

BMI for age and gender was the criterion variable in this study. Associations between 26 neighborhood-level conditions (independent environmental variables) and BMI were determined through use of test for homogeneity of variances: the Levene's test for equality of variances { $e_{ij} = x_{ij} - \bar{x}_j$ } between variables and *t* test hypothesis testing for the differences between two means (Table 5). The independent environmental variables included a combination of census tract (2010) data and selected data collected by the CEHI Community Assessment Project (2012) overlaid on GIS mapping by zip code (*N*=7/16) and was further populated with BMI levels (≥ 95%) from the study population (*N*=419/433) who participated in the Durham County, North Carolina (2010) WIC program.

The Levene's Test for Quality of Variance

Neighborhood – level conditions that were assumed to adversely affect BMI levels in the research population were categorized by non-spatial Grouping Samples t-Tests using IBM SPSS software. That test compared the means between two unrelated groups on the same continuous, grouping variable. The Levene's Test for Equality of Variances was used to determine if the two conditions had about the same or different amounts of variability between scores was also used. An Alpha value greater than.05 indicated that the variability in the two conditions was about the same. That the scores in one condition did not vary too much more than the scores in the second condition indicated that the variability in the two conditions was not significantly different. The Levene's Test for Equality of Variances determined if any assumptions of the t-test were met.

The t-test assumed that the variability of each group was approximately equal. If the significance (Sig. or p value) was less than Alpha $\alpha = .05$, then the null hypothesis was rejected, hence if the variability of the two groups was equal, that implied that the variances were unequal.

Ordinary Least Squares and Exploratory Regression Tool

The study's 26 demographic obesogenic variables were tabulated by ZCTAs per the Durham County neighborhood zip codes used in this study. The obeseogenic variables were further quantified and codified for purposes of cross-tabulation and comparison of variance, as recommended by Singleton and Straits (2005). Contingency tables included the ordinary least squares [OLS] spatial statistic test results in ArcGIS 10.2 software and the exploratory regression tool [ERT]. The OLS provided a global model to gain an understanding of the variables. The dependent variable was set as (PCTObese95: Percent obese children 2-4yrs old 95^{th} percentile) and all other data/layers were inputted as explanatory variables. The results from the OLS test demonstrated that the probability and robust probability *p* values were higher than expected; therefore, accepting the null hypothesis showed that coefficients from the variables were not helping the model. Those results coincided with and confirmed earlier spatial testing that the dependent variable data was too small a sample size to perform effective spatial analysis with larger census level tract explanatory variable data. The histograms below and scatter plots whereby $Y' = A + B_1X_1 + B_2X_2 + B_3X_3 + B_4X_4 + B_5X_5 + B_6X_6$, etc., were considered as a direct extension of simply regression and interactions between any two explanatory variables where a relationship was recorded (Figures 7 to 10).

Histograms and Scatterplots

The ERT histograms and scatterplots showed a very low R^2 value that denoted a weak model performance for linear regression analysis. Hence, the R^2 value suggested the expected correlations and explanatory values for the dependent variable BMI levels greater than or equal to the 95^{th} percentile failed and failed to provide the strength of relationship between the explanatory variables and the dependent variable desired for this study. Consequently, strong relationships failed to appear as diagonals in the regression line as shown in the histogram and scatterplot figures (Figures 7, 8, 9, 10).

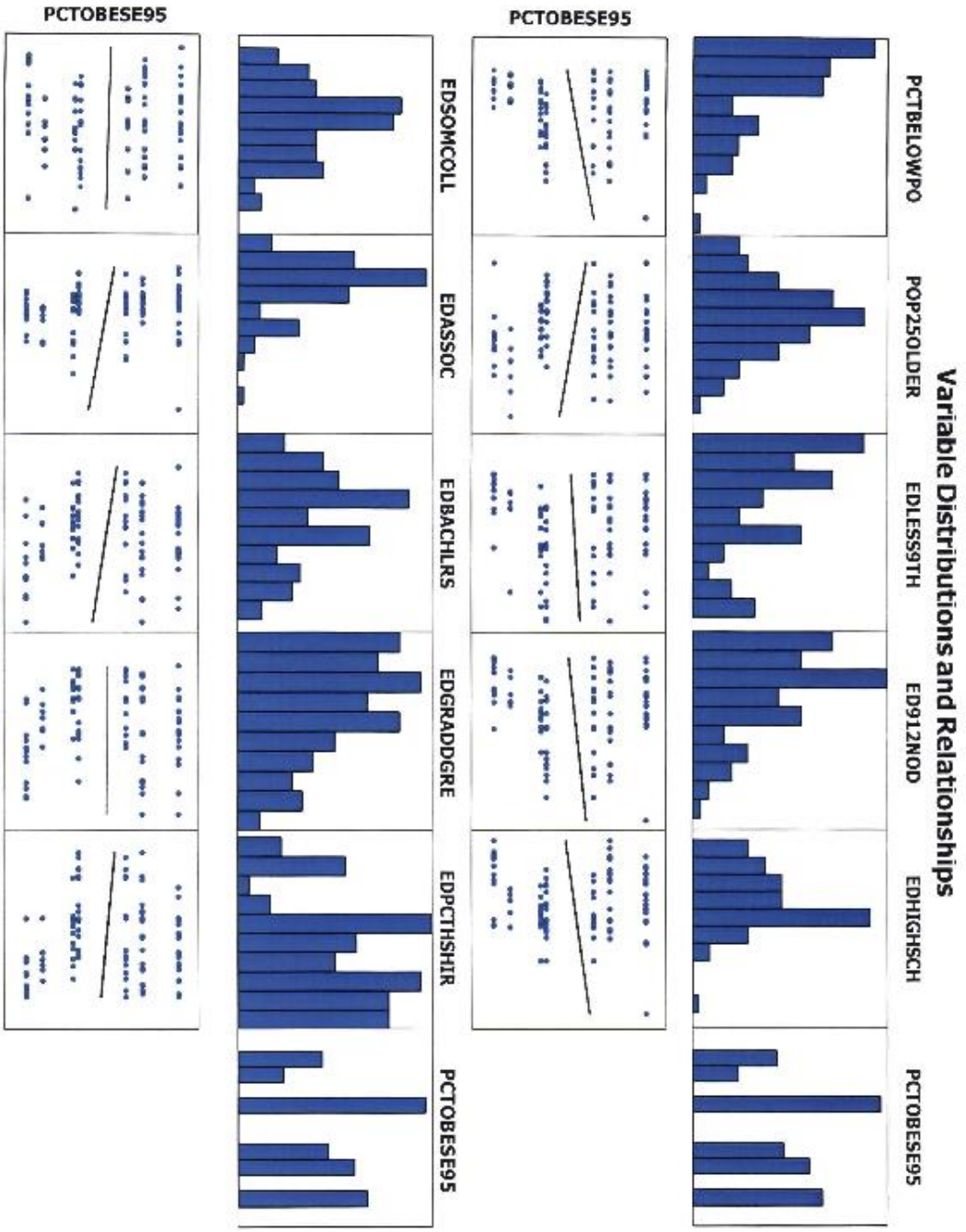

Figure 7. **“**Variable Distributions and Relationships Using Spatial Statistic Tools Within Esri ArcGIS 10.2 Software to Determine Relationship and Spatial Correlation Between Childhood Obesity Data and Certain Environmental Variables,” by Eureka C. Daye

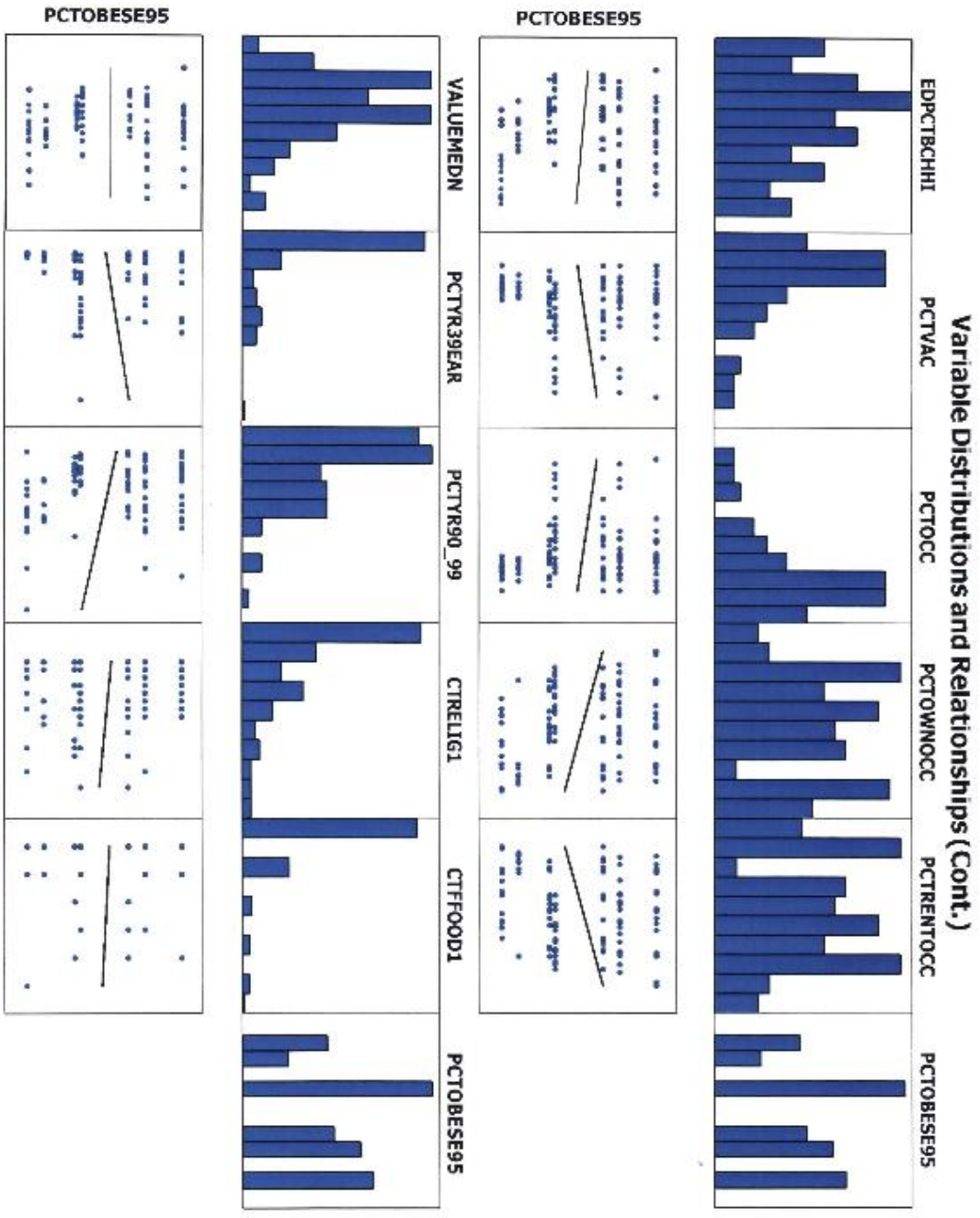

Figure 8. **"**Variable Distributions and Relationships Using Spatial Statistic Tools Within Esri ArcGIS 10.2 Software to Determine Relationship and Spatial Correlation Between Childhood Obesity Data and Certain Environmental Variables," by Eureka C. Daye

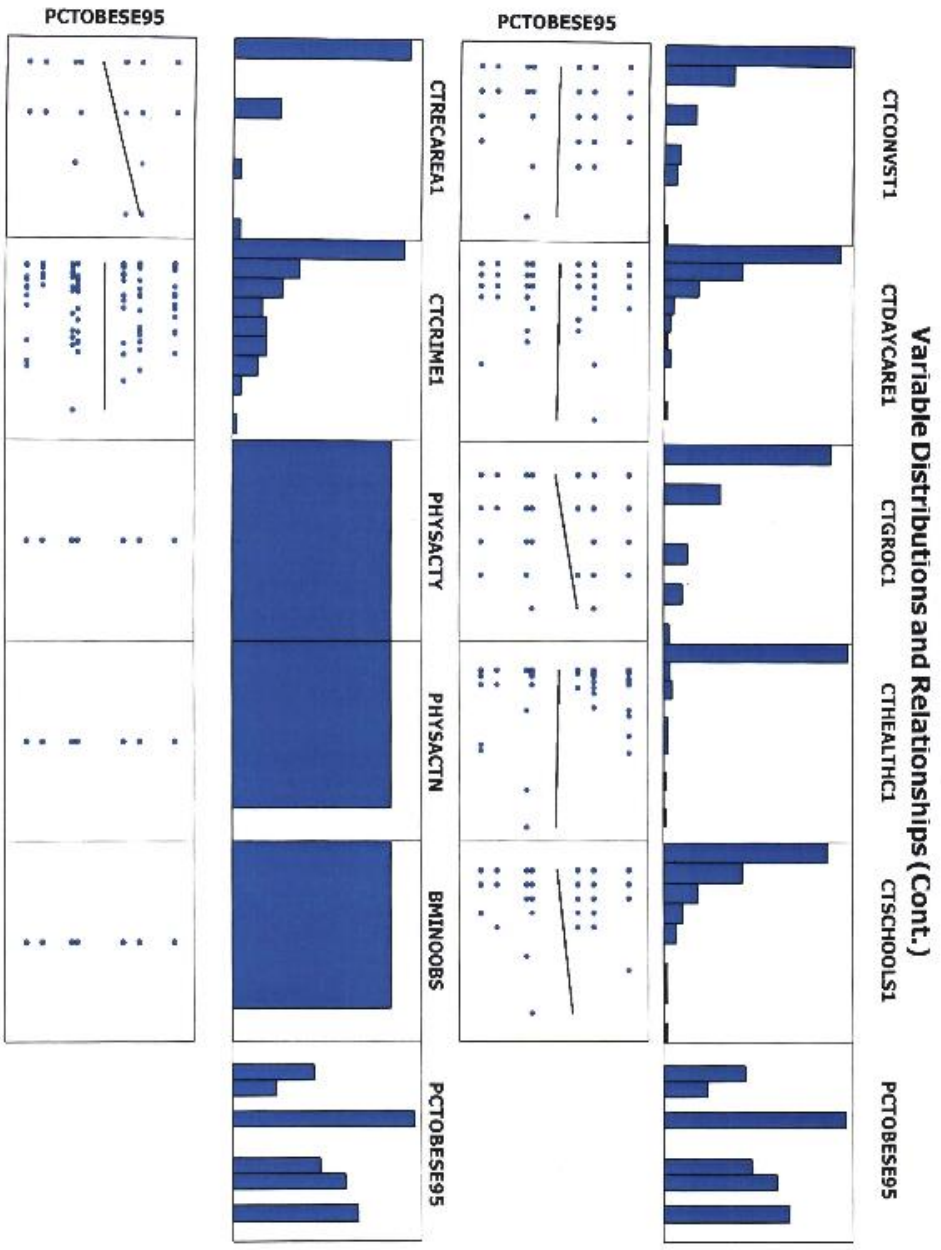

Figure 9. **"**Variable Distributions and Relationships Using Spatial Statistic Tools Within Esri ArcGIS 10.2 Software to Determine Relationship and Spatial Correlation Between Childhood Obesity Data and Certain Environmental Variables," by Eureka C. Daye

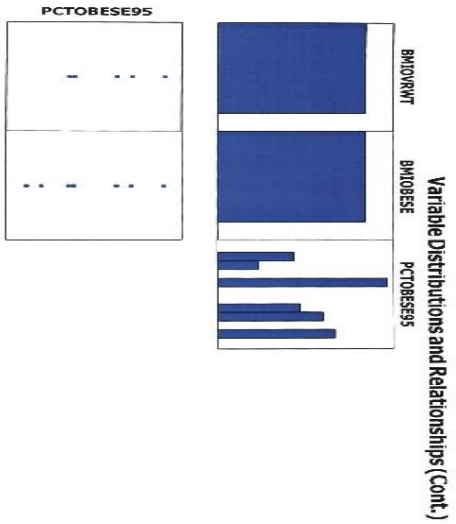

Figure 10. **"**Variable Distributions and Relationships Using Spatial Statistic Tools Within Esri ArcGIS 10.2 Software to Determine Relationship and Spatial Correlation Between Childhood Obesity Data and Certain Environmental Variables," by Eureka C. Daye

Below (Table5) showed the results from the Grouping Samples t-Tests which were configured with the cut-point of 18.7%, the median value of the seven ZCTA areas.

Table 5

Group Statistics

Group Statistics					
	PCTOBESE95	N	Mean	Std. Deviation	Std. Error Mean
EdLess9th	>= 19.3	29	3.96	4.88	.907
	< 19.3	35	7.44	5.19	.877
Ed912NoD	>= 19.3	29	7.30	6.69	1.242
	< 19.3	35	11.71	7.24	1.224
EdHighSch	>= 19.3	29	19.40	11.22	2.083
	< 19.3	35	21.52	8.06	1.362
EdSomColl	>= 19.3	29	16.17	5.13	.954
	< 19.3	35	18.56	5.56	.940
EdAssoc	>= 19.3	29	6.72	4.41	.818
	< 19.3	35	6.11	3.01	.508
EdBachIrs	>= 19.3	29	25.40	12.08	2.243
	< 19.3	35	19.33	10.18	1.721
EdGradDgre	>= 19.3	29	21.08	11.05	2.051
	< 19.3	35	15.31	11.48	1.941
EdPctHSHir	>= 19.3	29	88.74	9.79	1.817
	< 19.3	35	80.85	11.52	1.947
EdPctBchHi	>= 19.3	29	46.46	20.50	3.807
	< 19.3	35	34.63	20.92	3.536
PctVac	>= 19.3	29	8.97	8.28	1.538
	< 19.3	35	13.76	9.68	1.636
PctOcc	>= 19.3	29	91.03	8.29	1.538
	< 19.3	35	86.24	9.68	1.636
	PCTOBESE95	N	Mean	Std. Deviation	Std. Error Mean

PctOwnOcc	>= 19.3	29	56.44	31.54	5.856
	< 19.3	35	50.94	24.80	4.192
PctRentOcc	>= 19.3	29	43.56	31.54	5.856
	< 19.3	35	49.06	24.80	4.192
VALUEMEDN	>= 19.3	29	192848.28	88666.44	16464.945
	< 19.3	35	155131.43	68597.98	11595.176
PctYr39Ear	>= 19.3	29	4.52	9.17	1.703
	< 19.3	35	11.65	15.24	2.576
PctYr90_99	>= 19.3	29	19.55	12.64	2.348
	< 19.3	35	14.21	12.89	2.1788
CTRelig	>= 19.3	29	4.83	3.73	.693
	< 19.3	35	6.80	4.44	.751
CTFFood	>= 19.3	29	1.07	1.438	.267
	< 19.3	35	.66	1.027	.174
CTConvSt	>= 19.3	29	1.03	1.322	.246
	< 19.3	35	1.46	1.704	.288
CTDayCare	>= 19.3	29	2.38	2.527	.469
	< 19.3	35	2.86	2.534	.428
CTGroc	>= 19.3	29	1.14	1.217	.226
	< 19.3	35	1.00	1.188	.201
CTHealthC	>= 19.3	29	5.48	8.288	1.539
	< 19.3	35	5.17	12.874	2.176
CTHosp	>= 19.3	29	.17	.539	.100
	< 19.3	35	.17	.747	.126
CTSchools	>= 19.3	29	1.59	2.027	.376
	< 19.3	35	1.69	2.139	.362
CTRecArea	>= 19.3	29	.38	.820	.152
	< 19.3	35	.69	.796	.135
CTCrime	>= 19.3	29	167.24	150.247	27.900
	< 19.3	35	242.09	146.480	24.760

Note. Chart Created From Statistical Data Used in this Study.

Grouping Samples t-Test Results List and Narrative

The below list is a narrative of the Grouping Samples t-Tests used in this study to determine significance. The first Independent Samples t-Test was configured with obesity percentage rate as "Grouping Variable" with the cut-point of 18.7%, the median value of the seven ZCTA areas. The 26 independent variables were input as the "Test Variable", each with an Alpha of .05. This configuration produced "statistical significance" only on test variables (% Vacant, % Occupied, % Owner Occupied, % Renter Occupied, % Housing built 1939 or earlier). It was then configured with the "mean" rate of 19.3%. This produced "statistical significance" on test variables (% Education less than 9th grade, % Education 9-12 no degree, % Education Bachelor's Degree, % Education Graduate Degree, % Education High School or higher, % Education Bachelor's Degree or higher, % Housing Vacant, % Housing Occupied, % Housing built 1939 or earlier, Count of Crimes).

- H_0: $\mu_{\text{HIGHER OBESITY TRACTS}} = \mu_{\text{TESTED VARIABLE TRACTS}}$
- H_1: $\mu_{\text{HIGHER OBESITY TRACTS}} \neq \mu_{\text{TESTED VARIABLE TRACTS}}$

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95th percentile)

Test Variable: Education: Percent with Education less than 9th grade (EdLEss9th)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = 1.035; p = .313

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t' Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

M = Mean, s = Standard Deviation, *t* = Degrees of freedom, p = p value associated with test, α = alpha level 95% conf.

An equal variances *t* test **PASSED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = 3.959, s = 4.883) and lower Obese children per census tract (M = 7.443, s = 5.186), *t* (62) = 2.747, *p* = .008, α = .05.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95th percentile)

Test Variable: Education: Percent with Education 9th – 12th Grade No Degree (Ed912NoD)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = .896; p = .348

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t' Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **PASSED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = 7.297, s = 6.686) and lower obese

Children per census tract (M = 11.714, s = 7.240), *t* (62) = 2.515, *p* = .015, α = .05.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95th percentile)

Test Variable: Education: Percent with High School Diploma (EdHighSch)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = 2.220; p = .141

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **FAILED** to reveal a statistically reliable significance

between the mean percentage of higher obese children (M = 19.403, s = 11.216)

and lower obese children per census tract (M = 21.523, s = 8.055), *t* (62) = .878,

TS = F = .356; p = .553

ARE assumed at the Alpha = 0.05 level.

Results for Pooled t Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative *p* = .383, α = .05.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95th percentile)

Test Variable: Education: Percent with Some College Education (EdSomColl)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

Equal Variances Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **FAILED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = 16.166, s = 5.134) and lower obese children per census tract (M = 18.557, s = 5.557), *t* (62) = 1.773, $p = .081$, $\alpha = .05$.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95^{th} percentile)

Test Variable: Education: Percent with Associates Degree (EdAssoc)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = 3.090; p = .084

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **FAILED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = 6.721, s = 4.407) and

lower obese children per census tract (M = 6.114, s = 3.007), t (62) = .652,

$p = .517$, $\alpha = .05$.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95th percentile)

Test Variable: Education: Percent with Bachelor's Degree (EdBachlrs)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = .645; p = .425

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **PASSED** to reveal a statistically reliable significance

between the mean percentage of higher obese children (M = 25.397, s = 12.080)

and lower obese children per census tract (M = 19.326, s = 10.182), *t* (62) = 2.182,

$p = .033$, $\alpha = .05$.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95th percentile)

Test Variable: Education: Percent with Graduate Degree (EdGradDgre)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = .277; p = .600

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **PASSED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = 21.079, s = 11.045) and lower obese children per census tract (M = 15.306, s = 11.484) t (62) = 2.037, p = .046, α = .05.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95th percentile)

Test Variable: Education: Percent with High School Diploma or higher (EdPctHSHir)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = 1.232; p = .271

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **PASSED** to reveal a statistically reliable significance

between the mean percentage of higher obese children (M = 88.741, s = 9.785) and lower obese children per census tract (M = 80.849, s = 11.517), *t* (62) = 2.918, *p* = .005, α = .05.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95th percentile)

Test Variable: Education: Percent with High School Diploma or higher (EdPctBchHi)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = .007; p = .935

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **PASSED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = 46.455, s = 20.502) and lower obese children per census tract (M = 34.629, s = 20.916), *t* (62) = 2.272, *p* = .027, α = .05.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95th percentile)

Test Variable: Housing: Percent Vacant Homes (PctVac)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = 1.380; p = 0.245

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t' Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **PASSED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = 8.966, s = 8.284) and lower obese children per census tract (M = 13.758, s = 9.680), t (62) = 2.102, *p* = .040, $\alpha = .05$.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95^{th} percentile)

Test Variable: Housing: Percent Occupied Homes (PctOcc)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = 1.380; p = .245

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t' Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **PASSED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = 91.033, s = 8.284) and lower obese children per census tract (M = 86.241, s = 9.680), *t* (62) = 2.102, $p = .040$, $\alpha = .05$.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95th percentile)

Test Variable: Housing: Percent Owner Occupied Homes (PctOwnOcc)

Results for Levene's Test for Equality of Variances:

$H_0: \sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

$H_1: \sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = 3.739; p = 0.058

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t' Test for Equality of Means:

$H_0: \mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

$H_1: \mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **FAILED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = 56.438, s = 31.537) and lower obese children per census tract (M = 50.937, s = 24.801), *t* (62) = .781, $p = .438$, $\alpha = .05$.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95th percentile)

Test Variable: Housing: Percent Owner Occupied Homes (PctRentOcc)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = 3.739; p = .058

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t' Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **FAILED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = 43.562, s = 31.537) and lower obese children per census tract (M = 49.063, s = 24.801), t (62) = .781, *p* = .438, $\alpha = .05$.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95^{th} percentile)

Test Variable: Housing: Median Value of Housing ($) (ValueMedn)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = .392; p = .533

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t' Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **FAILED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = 192848.28, s = 88666.440) and lower obese children per census tract (M = 155161.43, s = 68597.984), *t* (62) = 1.918, *p* = .060, α = .05.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95th percentile)

Test Variable: Housing: Percent Homes Built in 1939 or Earlier (PctYr39Ear)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = 8.190; p = .006

Equal Variances ARE NOT assumed at the Alpha = 0.05 level.

Results for Welch t' Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **PASSED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = 4.517, s = 9.1696) and lower obese children per census tract (M = 11.654, s = 15.240), t (62) = 2.311, *p* = .024, α = .05.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95th percentile)

Test Variable: Housing: Percent Homes Built between 1990 - 1999 (PctYr90_99)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = .002; p = .962

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t' Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **FAILED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = 19.548, s = 12.644) and lower obese children per census tract (M = 14.214, s = 12.889), t (62) = 1.665, p = .102, α = .05.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95^{th} percentile)

Test Variable: Housing: Count of Religious Institutions in Census Tract (CTRelig)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = 1.833; p = .181

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t' Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **FAILED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = 4.83, s = 3.733) and lower Obese children per census tract (M = 6.80, s = 4.444), t (62) = 1.898, *p* = .062, α = .05.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95th percentile)

Test Variable: Housing: Count of Fast Food Establishments in Census Tract (CTFFood)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = 1.090; p = .301

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t' Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **FAILED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = 1.07, s = 1.438) and lower Obese children per census tract (M = .66, s = 1.027), t (62) = 1.334, *p* = .187, α = .05.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95th percentile)

Test Variable: Housing: Count of Convenience Stores in Census Tract (CTConvSt)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = 1.030; p = .314

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t' Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **FAILED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = 1.03, s = 1.322) and lower Obese children per census tract (M = 1.46, s = 1.704), t (62) = 1.091, p = .280, α = .05.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95th percentile)

Test Variable: Housing: Count of Day Care Centers in Census Tract (CTDayCare)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = .476; p = .493

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t' Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **FAILED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = 2.38, s = 2.527) and lower obese children per census tract (M = 2.86, s = 2.534), t (62) = .752, *p* = .455, $\alpha = .05$.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95th percentile)

Test Variable: Housing: Count of Grocery Stores in Census Tract (CTGroc)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = .283; p = .597

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t' Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **FAILED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = 1.14, s = 1.217) and lower obese children per census tract (M = 1.00, s = 1.188), t (62) = .457, *p* = .649, $\alpha = .05$.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95th percentile)

Test Variable: Housing: Count of Health Care Centers in Census Tract (CTHealthC)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = .652; p = .422

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t' Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **FAILED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = 5.48, s = 8.288) and lower obese children per census tract (M = 5.17, s = 12.874), t (62) = .112, p = .911, α = .05.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95^{th} percentile)

Test Variable: Housing: Count of Hospitals in Census Tract (CTHosp)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = .009; p = .923

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t' Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **FAILED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = .17, s = .539) and lower obese children per census tract (M = .17, s = .747), t (62) = .006, p = .995, α = .05.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95th percentile)

Test Variable: Housing: Count of Schools in Census Tract (CTSchools)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = .004; p = .951

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t' Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **FAILED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = 1.59, s = 2.027) and lower obese children per census tract (M = 1.69, s = 2.139), t (62) = .190, p = .850, α = .05.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95th percentile)

Test Variable: Housing: Count of Recreational Areas in Census Tract (CTRecArea)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = .530; p = .469

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t' Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

An equal variances *t* test **FAILED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = .38, s = .820) and lower obese children per census tract (M = .69, s = .796), t (62) = 1.512, p = .136, α = .05.

- **Grouping Variable:** Obesity percentage rate of 2-4 year old children (95^{th} percentile)

Test Variable: Housing: Count of Crimes in Census Tract (CTCrime)

Results for Levene's Test for Equality of Variances:

H_0: $\sigma_1^2 = \sigma_2^2$ Null Hypothesis: Variance one IS equal to Variance two

H_1: $\sigma_1^2 \neq \sigma_2^2$ Alternative Hypothesis: Variance one is NOT equal to Variance two

TS = F = .459; p = .501

Equal Variances ARE assumed at the Alpha = 0.05 level.

Results for Pooled t' Test for Equality of Means:

H_0: $\mu_1^2 = \mu_2^2$ Null Hypothesis: Mean one IS equal to Mean two

H_1: $\mu_1^2 \neq \mu_2^2$ Alternative Hypothesis: Mean one is NOT equal to Mean two

and equal variances *t* test **PASSED** to reveal a statistically reliable significance between the mean percentage of higher obese children (M = 167.24, s = 150.247) and lower obese children per census tract (M = 242.09, s = 146.48), t (62) = 2.011, p = .049, α = .05.

Overall, the 26 independent variables of the built environment were inputted as the "Test Variables" and each with an Alpha of .05 produced "statistical significance" on a small subset of test variables % Vacant, % Occupied, % Owner Occupied, % Renter Occupied, % Housing built 1939 or earlier. The test was also configured with the "mean" rate of 19.3%. That produced "statistical significance" on test variables % Education less than 9th grade, % Education 9-12 no degree, % Education Bachelor's Degree, % Education Graduate Degree, % Education High School or higher, % Education Bachelor's Degree or higher, % Housing Vacant, % Housing Occupied, % Housing built 1939 or earlier, Count of Crimes.

One of the most difficult parts of this study was to acquire the proper datasets GIS data from the University of Michigan and CEHI (2011), the entity involved in an original GIS project for the same location, Durham County, North Carolina. Those datasets were needed to perform analysis, conversion and to import individual layers for mapped GIS. Many attempts were made to acquire the full sets of GIS data from the University of Michigan and CEHI (2011) but to no advantage. Therefore, a consolidated version of the data was obtained by extracting those

geographic data (points, lines, polygons) in KMZ - zip file format from an earlier CEHI (2011) GIS web based mapped project (http://cehi.snre.umich.edu/share/capmap).

The KMZ data provided limited information in the associated attribute fields. Each KMZ file extracted was copied into a GIS format as a layer. Those layer files from the earlier CAP-CEHI study were stored in a feature dataset inside a file geodatabase. The data were originally in a Geographic Coordinate System with a North American 1983 Datum. Each layer needed to be spatially re-projected into a "projected" coordinate system. The final projected coordinate system was NAD 83 State Plane North Carolina. To perform accurate spatial analysis in GIS data, layers needed to be in the same projected coordinate system that could transfer geographic data from the Earth's three-dimensional spheroidal surface and transform it to create a flat map. The data for those maps were collected at individual tax parcel levels then aggregated and displayed at a Census block level. The attribute data for each layer (census block) was manually updated to closely reflect five equal percentile classification intervals or quantiles to coincide with the previous online CEHI (2011) web map version. That data only reflected a range or average of values; therefore no systematic regression analysis could be performed on the data.

Notwithstanding, those manually updated built environment variables from the Children's Environmental Health Initiative CEHI (2011) which included convenience stores, day care facilities, fast food establishments, grocery stores, health care facilities, park facilities, religious institutions and schools (k- 12) were plotted and mapped using GIS showed significant clustering in several ZCTAs per BMI which negated the researcher's assumption that Durham County had diminished resources in the study's prescribed ZCTAs. Those structured observations in specific ZCTAs did not support the researcher's hypothesize on early childhood

obesity since they showed numerous resources when compared to either lower or higher BMI percentages from the NC – NPASS (2010) data (see Appendixes 29 through 37).

Chapter 5: Discussion, Conclusions and Recommendations

Discussion

The purpose of this research project was to examine the influence of 26 built environment explanatory variables on the independent early childhood obesity variable through GIS analysis with converted and imported individual data layers and produced GIS geospatial maps. Secondary data analysis from the Children's Environmental Health Initiative (CEHI) (2011) and the North Carolina Nutrition and Physical Activity Surveillance System (NC-NPASS, 2010) as well as from 2010 U.S. Census bureau were data sources mapped through the geospatial methodology. The most noteworthy complication of the study was to obtain the original GIS data sets from the study to perform analysis, conversion and to import individual layers of data to produce GIS maps.

Although the CEIH (2011) study was public retrospective information for a period of time during the course of this dissertation process, the principals of the study from the University of Michigan in the end pulled and made proprietary their metadata from their GIS project contingent on impending grant monies. Many attempts were made to acquire full sets of GIS data from the University of Michigan CEHI (2011) GIS study that was already configured at the census block level and already in a spatial GIS format and would have provided thorough results as well as would have reduced upfront costs for GIS software and consultation. Hence, a consolidated version of the data was eventually obtained by extracting it in KMZ or zip drive format from an earlier CEIH (2011) GIS web-based map project. Compiling the extracted KMZ data through this approach provided limited information in the associated attribute fields which was another learned challenge in using GIS in the acquisition of detailed data from secondary sources.

Method Implications

The use of secondary geospatial data sources for this public health study proved challenging since the metadata sets needed for this study required access to CEIH (2011) complex aggregate and embedded spatial data and coordinates. Not knowing all of the steps taken by the original researchers in their GIS project particularly in effort to precisely replicate and to meet the intentions of this dissertation study on secondary data analysis of childhood obesity and the built environment in GIS was hampered. The secondary data from CEHI (2011) was subsequently copied into a GIS format as layers. Those layer files were stored in a geodatabase and each layer was further spatially re-projected into a "projected" geographic coordinate system.

Moreover, to perform accurate spatial analysis in GIS data/layers required the same projected coordinate system used by CEIH (2011) to transfer geographic data from the Earth's three-dimensional spheroidal surface and to transform it to create flat maps. The data for the maps designed for this study were collected at individual tax parcel levels then aggregated and displayed at a Census block level. The attributed data for each layer (census block) was then manually updated to closely coincide with the previous CEHI (2011) online web map version data retrieved from http://cehi.snre.umich.edu/share/capmap. This data reflected a range or average of values; therefore no systematic analysis could be performed on the data. GIS maps were however created for each of these layers of built environment domains.

The census data for the study was acquired from the U.S. Census Bureau (2010) American Fact Finder website the census tract level. The projects seven ZCTA's coincided with the Durham County, North Carolina NC – NPASS (2010) BMI for children 2 - 4 years old. Two

ZCTAs areas, however, were slightly overlapped into Orange County and into Chatham County which potentially skewed the study area. The census tract data per ZCTA was downloaded and imported into the project's geodatabase then joined to the census tract polygon features and represented inside a GIS.

The 14,471 crime data records reported throughout 2010 and used in this study were acquired from Durham Police Department's website. This data was extracted as text then introduced into a database format to import into a GIS. Once in GIS, the database of records was geocoded based on incident address fields and given a spatial coordinate for each record. This iterative process weighted heavily on the integrity of the addresses from whence crime reports originated and the repetitious approach located and created geographic points for 13,323 of the 14,471 crime incidents in Durham County reported during 2010. 1,148 crime reports were not verifiable based on missing information from the physical address. The crime incidents were further summarized to obtain a count of occurrences per census tract, and associated with each tract accordingly.

GIS Statistical Implications and Conclusions

In order to project and import all datasets into a single geodatabase for statistical analysis, the Exploratory Regression Tool (Figures 6 - 9) was used to determine which regression analysis testing inside the GIS format would best evaluate the relationships and spatial correlation between the dependent variable childhood obesity (PCTObese95: Percent obese children 2-4yrs old 95th percentile) and explanatory variables to yield a specified regression model. However, because the dependent variable (obesity data) was at a larger/coarse ZCTA area, in comparison to all explanatory (environmental) variables which were at a smaller/finer, census tract level, the

Exploratory Regression Tool inside the GIS project continually showed a very weak model for regression analysis, hence substantiation of cause and effect could not be determined between the criterion and explanatory variables.

On the whole, statistical significance for a subset of built environment variables were determined through the use of non-spatial Grouping Samples t-Tests (Table.7) which compared the means between two unrelated groups on the same continuous, grouping variable and The Levene's Test for Equality of Variances (Table.6) was used to determine if any two conditions had about the same or different amounts of variability between scores. The IBM SPSS software was used for those purposes with an Alpha value greater than.05. The first Independent Samples t-Test was configured with obesity percentage rate as "Grouping Variable" with the cut-point of 18.7%, the median value of the seven ZCTA areas. The 26 independent variables were inputted as the "Test Variable", each with an Alpha of .05. This configuration produced "statistical significance" on test variables: % Vacant, % Occupied, % Owner Occupied, % Renter Occupied, % Housing built 1939 or earlier. When configured the with the "mean" cut - point of 19.3% "statistical significance" was produced on test variables: % Education less than 9^{th} grade, % Education 9-12 no degree, % Education Bachelor's Degree, % Education Graduate Degree, % Education High School or higher, % Education Bachelor's Degree or higher, % Housing Vacant, % Housing Occupied, % Housing built 1939 or earlier, Count of Crimes.

GIS Spatial Mapped Data Observations and Conclusions

The researcher's structured observations of similar CEHI (2011) build environment and manually updated variable - polygons showed evidence of spatial clustering although those were

not overly interpreted since reliable regression assessments were beyond the scope of this study. The following observations were made:

Appendix A29: GIS was used to map crime as an obesogenic variable by census tract. Observations showed spatially significant clustering where crime may be associated with early childhood obesity in the ZCTA 27701 with 18.7% of children on WIC at or below the 185% Federal Poverty level with BMI ≥ 95%; ZCTA 27707 with 21.0% of children on WIC at or below the 185% Federal Poverty level with BMI ≥ 95%; and ZCTA 27705 with 22.3% of children on WIC at or below the 185% Federal Poverty level with BMI ≥ 95%.

Appendix A30: GIS was used to map convenience stores as an obesogenic variable by census tract. Observations showed spatially significant clustering where convenience stores may be associated with early childhood obesity in ZCTA 27701 with 18.7% of children on WIC at or below the 185% Federal Poverty level with BMI ≥ 95%; ZCTA 27707 with 21.0% of children on WIC at or below the 185% Federal Poverty level with BMI ≥ 95%; ZCTA 27703 with 20.4% of children on WIC at or below the 185% Federal Poverty level with BMI ≥ 95%; and ZCTA 27705 with 22.3% of children on WIC at or below the 185% Federal Poverty level BMI ≥ 95%.

Appendix A31: GIS was used to map day care facilities as an obesogenic variable by census tract. Observations showed spatially diminished numbers of day care facilities that may be associated with early childhood obesity in ZCTA 27712 with 17.4% of children on WIC at or below the 185% Federal Poverty level with BMI ≥ 95%. Appendix 32: Fast Food Establishments appeared to have unremarkable clustering in the 7 ZCTAs studied and conclusions could not be drawn that fast food establishments were associated with the BMI ≥ 95% of children ages 2 - 4.

Appendix A32: GIS was used to map fast food establishments as an obesogenic variable by census tract. Observations showed spatially significant clustering of grocery stores that may be associated with early childhood obesity in ZCTA 27705 with 22.3% of children on WIC at or below the 185% Federal Poverty level with BMI ≥ 95% and in ZCTA 27707 where 21.0 % of children on WIC at or below the 185% Federal Poverty level with BMI ≥ 95%.

Appendix A33: GIS was used to map grocery stores as an obesogenic variable by census tract. Observations showed spatially diminished numbers of grocery stores that may be associated with early childhood obesity (2) in ZCTA 27712 with 17.4% of children on WIC at or below the 185% Federal Poverty level with BMI ≥ 95% and in ZCTA 27713 which had (5) grocery stores where 16.8 % of children on WIC at or below the 185% Federal Poverty level with BMI ≥ 95%.

Appendix A34: GIS mapped health care facilities as an obesogenic variable by census tract which showed a spatially diminished number of health care facilities which may be associated with early childhood obesity (4) in ZCTA 27712 with 17.4% of children on WIC at or below the 185% Federal Poverty level with BMI ≥ 95% and in ZCTA 27703 which had 7 healthcare facilities (5 located at the remote southern end of the ZCTA) where 20.4 % of children on WIC at or below the 185% Federal Poverty level with BMI ≥ 95%.

Appendix A35: GIS was used to map park facilities as an obesogenic variable by census tract. Observations showed spatially diminished numbers of parks that may be associated with early childhood obesity 1 park in the ZCTA 27712 with 17.4% of children on WIC at or below the 185% Federal Poverty level with BMI ≥ 95%; 5 parks in ZCTA 27705 with 22.3% of children on WIC at or below the 185% Federal Poverty level with BMI ≥ 95%; ZCTA 27704

with 4 parks and 18.5% of children on WIC at or below the 185% Federal Poverty level with BMI ≥ 95%; and ZCTA 27703 with 4 parks and 20.4% of children on WIC at or below the 185% Federal Poverty level with BMI ≥ 95%; and ZCTA 27713 with 1 park and 16.8% of children on WIC at or below the 185% Federal Poverty level with BMI ≥ 95%.

Appendix A36: GIS was used to map religious institutions as an obesogenic variable by census tract. Observations showed spatially significant clustering of religious institutions across all 7 ZCTAs which could be surmised as not associated with the BMI of children on WIC at or below the 185% Federal Poverty level.

Appendix A37: GIS mapped school (k-12) as an obesogenic variable by census tract which showed a spatially diminished number of schools (5) which may be associated with early childhood obesity in ZCTA 27712 with 17.4% of children on WIC at or below the 185% Federal Poverty level with BMI ≥ 95%. The additional 6 ZCTAs had a clustering or scatter of schools where conclusions could not be drawn about any association with the BMI ≥ 95% of children ages 2 - 4.

Comparison studies on GIS and the built environment on childhood obesity

Although an emerging technology, GIS when used in studies on the build environment and childhood obesity had it challenges to include spatial lags between polygons, errors in data replication, problems with precision and accuracy of data and fundamental criteria and conceptual problems (Liu, Colbert, Wilson, Yamada & Hoch, 2007; Davidson and Lawson, 2006; McLafferty, 2002). Accuracy in the data seemed to be an inherent issue in using GIS and the build environment. McLafferty (2002) found in her annual review on public health that practitioners not only found structural barriers when using GIS data but also found significant

problems with the consistent utilization and availability of data across practitioners. McLafferty (2002) suggested that practitioners needed to understand that spatial analysis was a useful tool in examining and evaluating the needed health care delivery systems and their locations based projected need and potential utilization, but also needed to understand that data was not always interpreted and applied every time. Hence, the use of secondary data had a tendency to be misinterpretation and therefore not reliable.

Several studies found that there was a lack understanding in the industry when using GIS in the study of the built environment and childhood obesity that there are both scale and autocorrelation problems (Thornton, Pearce, and Kavanaugh, 2011). Similar to this study completed by the undersigned, several secondary studies found statistical correlations from environmental variables that were thought to impede children's activity levels, however, concluded that secondary studies were often non – replicable (McCrorie, Fenton, & Ellaway, 2014; Krieger et al. 2001; Zhang, Christoffel, Mason & Liu (2006). GIS variables associated with risk for early childhood weight and the built environment included aspects of SES levels, crime, property values, parks, playgrounds, day cares, restaurants, healthcare entities to name a few. Koleilat (2010) used GIS measures and determined that spatial autocorrelations between the distribution of neighborhood parks, food outlets and conditions were associated with early childhood obesity in 3 - 4 year old children, but suggested that misinterpretation of data relationships could occur due to autocorrelation and non-stationarity problems.

In addition, a study on the distribution of fast food restaurants by Block, Scribner, & DeSalvo, (2004) found that many restaurants were undercounted due to the unique geographic areas of the neighborhoods. Krieger et al. (2001) also compared geocoding firms and found

widely varying geocoding success rates as well as large differences in the accuracy of census tract assignments which impeded her study's accuracy. Zhang, Christoffel, Mason & Liu (2006) likewise used GIS and hierarchically clustered analysis to assess environmental data sources across several neighbourhoods in Chicago to determine neighbourhoods best suited for physical activity and those neighbourhoods that were not and found that obesogenic conditions The built environment data between neighborhoods was very complex when aggregated and when represented in polygons and thus widely varied in accuracy and was missing precise GIS spatial relationship data . Matthews (2012) postulated that public health researchers should be careful when using the GIS technologies due to the high potential of discrepant use and interpretation of data and outcome findings.

Limitations

This GIS study had several limitations particularly in establishing causality when using secondary spatial data to generate objective measures for large geographic areas. The intended use of the CEHI (2011) GIS data was not fully assessable nor could the manually updated data be accurately interpreted since all of the steps taken by the original researchers in their GIS study were not known and therefore could not be replicated to the extent needed for this dissertation study. The built environment in GIS was also limited due to the unforeseen proprietary nature of the CEIH (2011) data particularly for grant purposes. In addition, the linear regression model based on environmental variables from the CEHI (2011) showed a weak R^2 indicative of nonlinear relationships and constraints of the secondary data's latitude and longitude coordinates and its spatial digital files and GIS settings and thus could not entirely meet the needs of this researcher. The cost of using GIS and spatial analysis is a limitation since the expense in not

only the software, but tutorial course for skill attainment and consultative services can range in the thousands of dollars and in this author's case was over $6,000.

Recommendations

GIS is an important technology in the measure of place variant obesogenic factors of the build environment and their influence on children's BMI and ought to be considered when policy priorities related to early childhood obesity are made. GIS surveillances and interactive maps have the potential to drive policy and decision making on neighborhood design and resource deployment in geographic areas where SES and race are considered obesogenic variants of the built environment (McGinn, Brines, 2008). GIS could help policy makers and public health practitioners better understand spatial and obesogenic cluster patterns in underserved neighborhoods to the extent of informing policy and social change. Federal and state legislation also ought to support community leaders and community tasks groups when modifiable obesogenic built environment barriers are identified through GIS geospatial mapped analysis and those changes can be made to neighborhoods to enhance children's physical activity, healthy diet and general wellness by installing for instance playgrounds, day cares, grocery stores, sidewalks and open spaces in the neighborhoods to encourage daily exercise (Roemmich, Epstein, Raja, Robinson & Winiewicz, 2006; Kuo, Bacaicoa, and Sullivan, 1998; McGinn, Brines, 2008).

Standardized policies, prototypes, definition and GIS user – friendly software are recommended to increase the knowledge adaptation, the utilization and ways to operationalize data outcomes from this innovative technology. Cost also is a factor in utilizing GIS and can largely forestall active research and current studies and ought to be considered. Funding sources should be developed and assessable to public health and industry researches in real time. Grant

monies and research dollars may not vastly apply to this fairly new research technology and thus may thwart the widespread use and dissemination of GIS data due to limited resource dollars. In addition, GIS can be extrapolated to map interdependent disease clusters, crime clusters, health program utilization, and mix land use which all are associated with the complexities of early childhood obesity.

Since methodological challenges posed significant challenges in the literature in studies that used GIS technology, the glossary of terms and definitions as they apply to public health should be taken into account. In addition, standardized protocols, too, should be considered which could provide processes and systems on how to functionally utilize GIS in public health studies.

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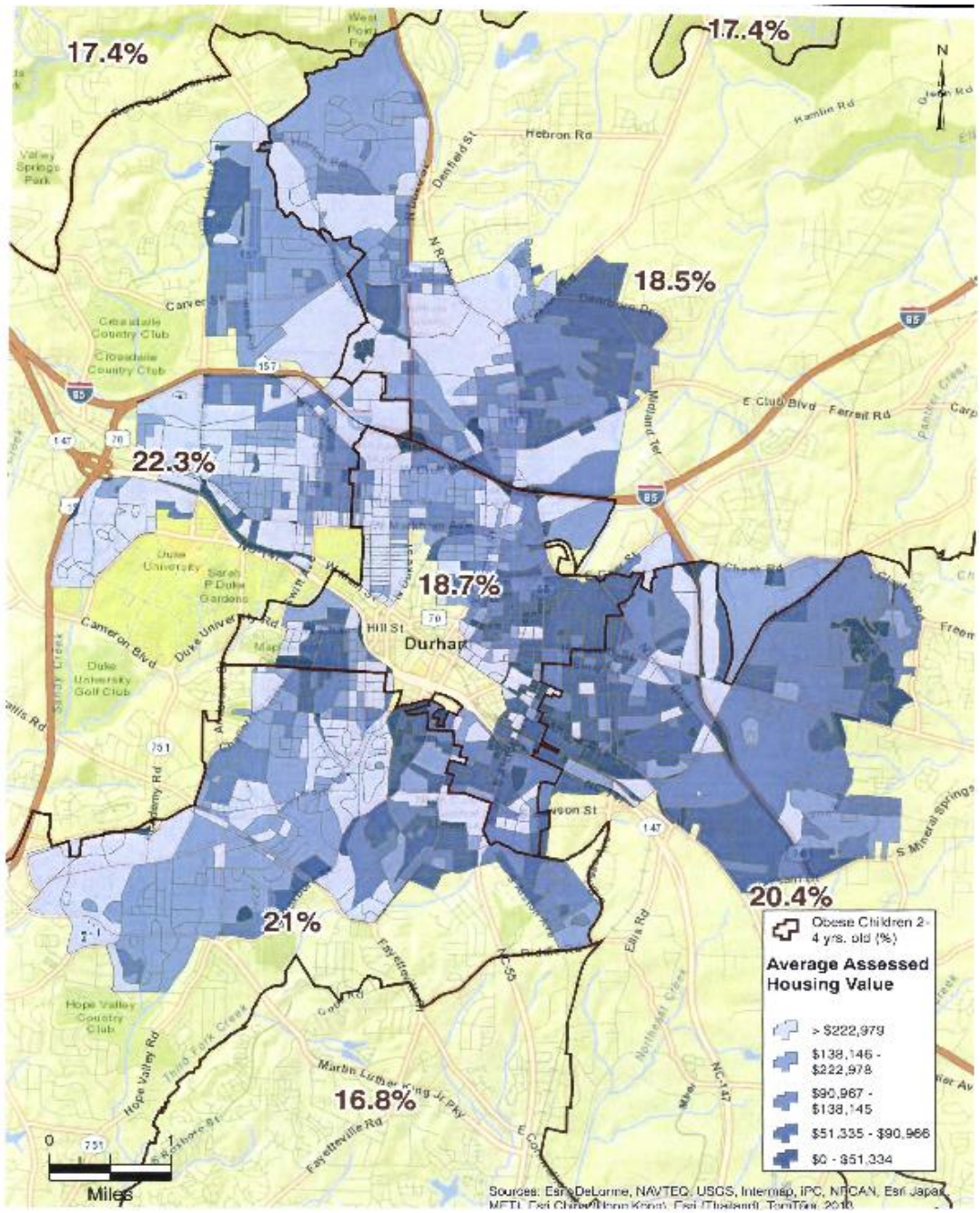
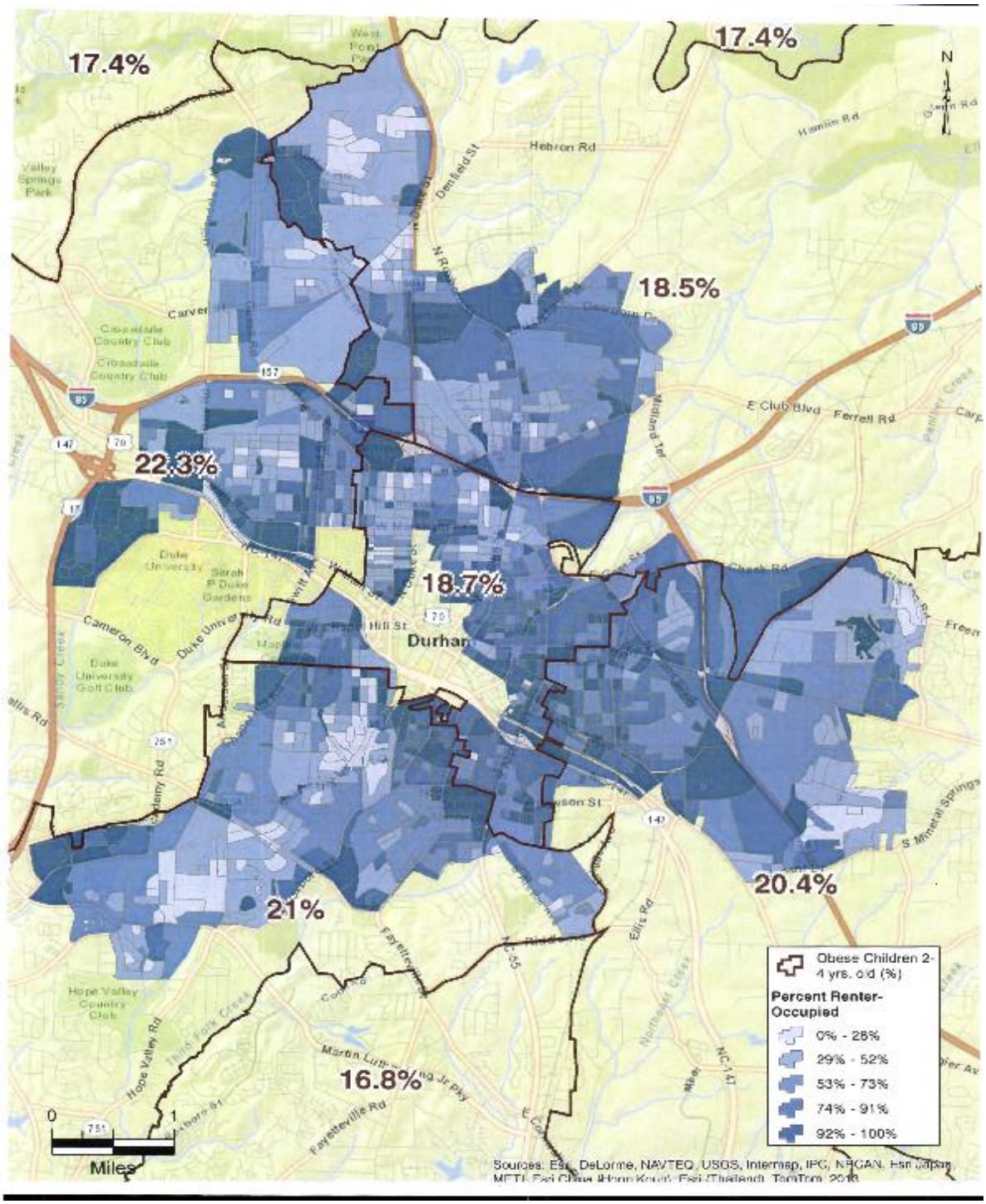
Appendix A1

Average Assessed Housing Value



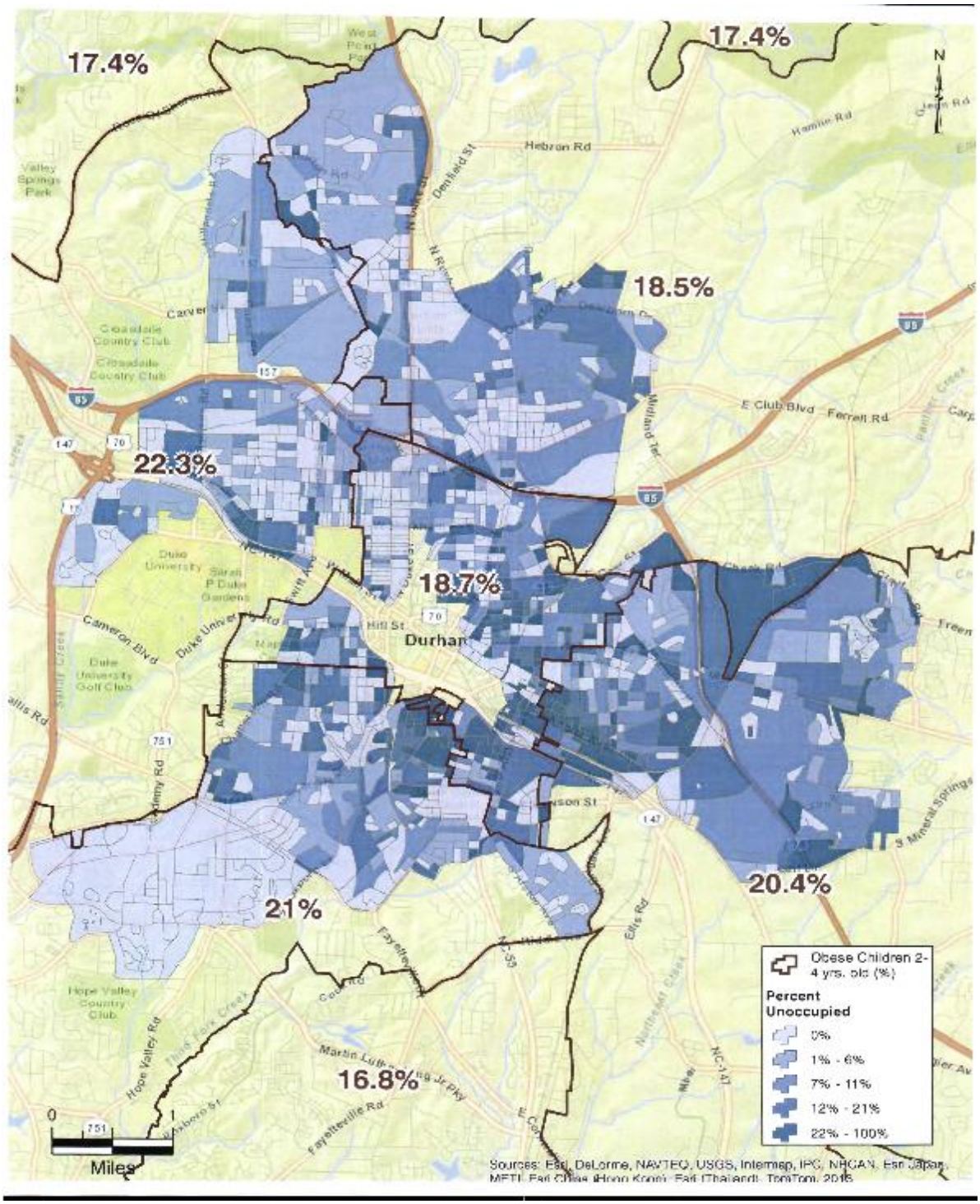
Appendix A2

Percent Renter Occupied



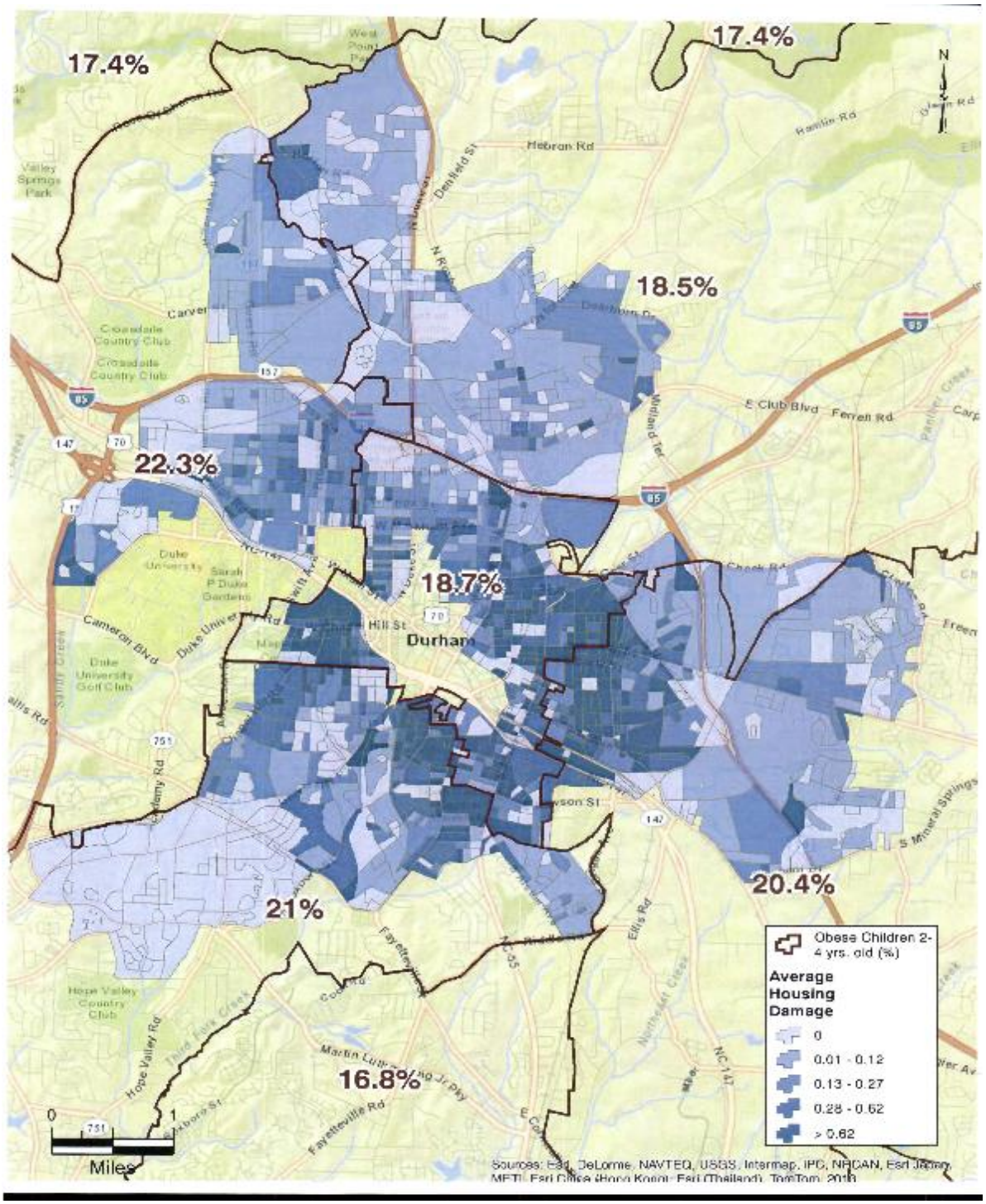
Appendix A3

Percent Unoccupied



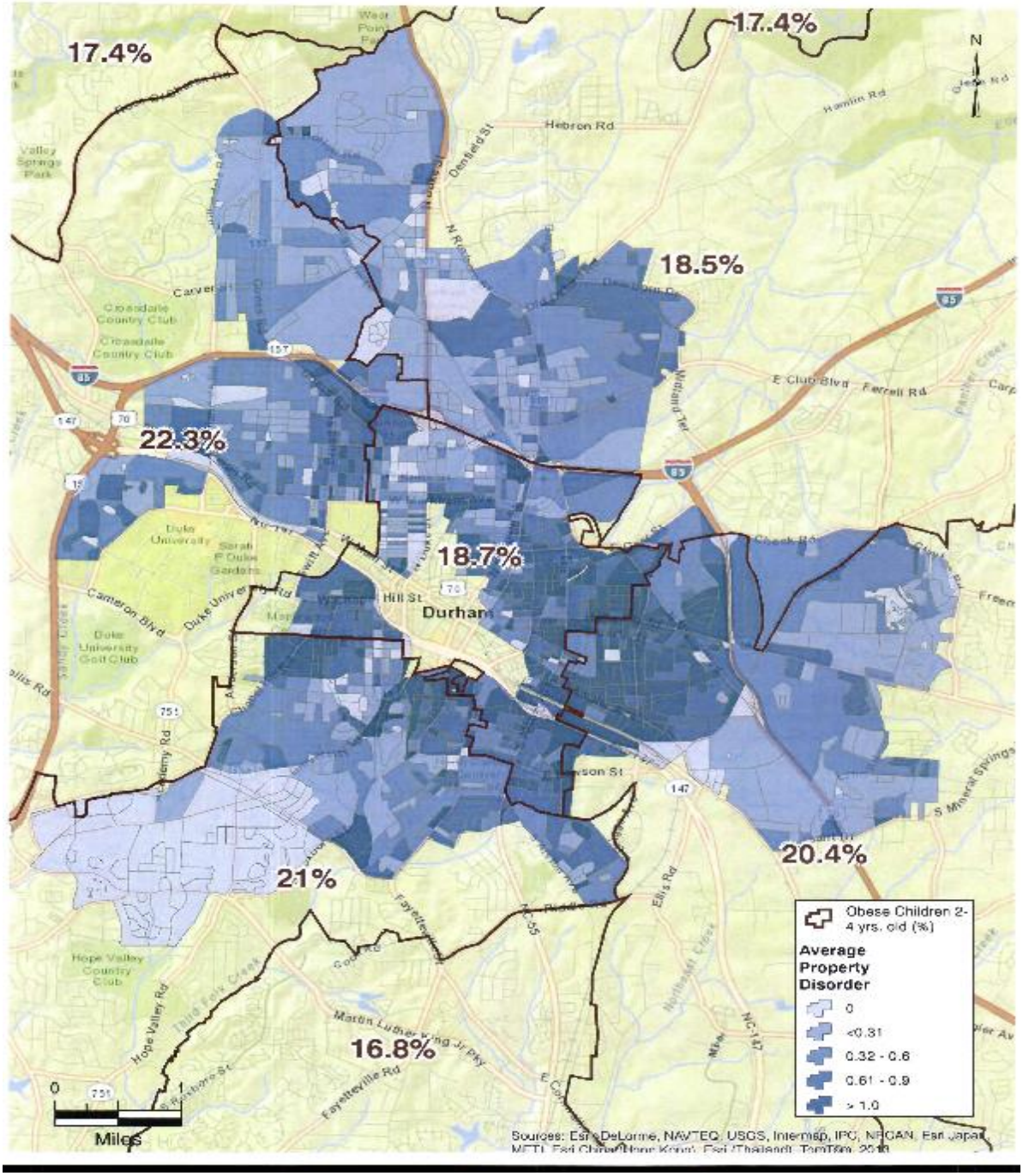
Appendix A4

Average Housing Damage



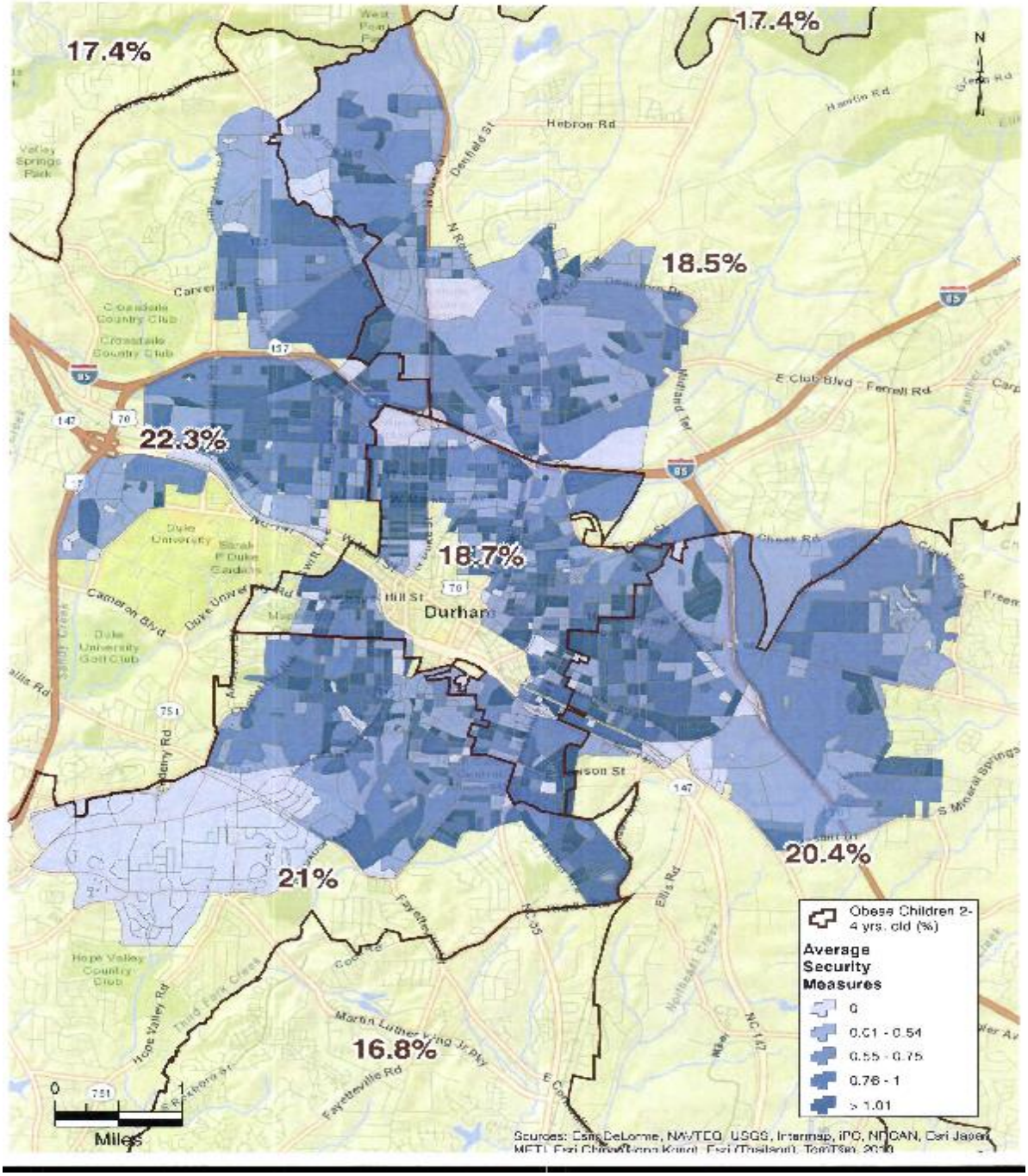
Appendix A5

Average Property Disorder



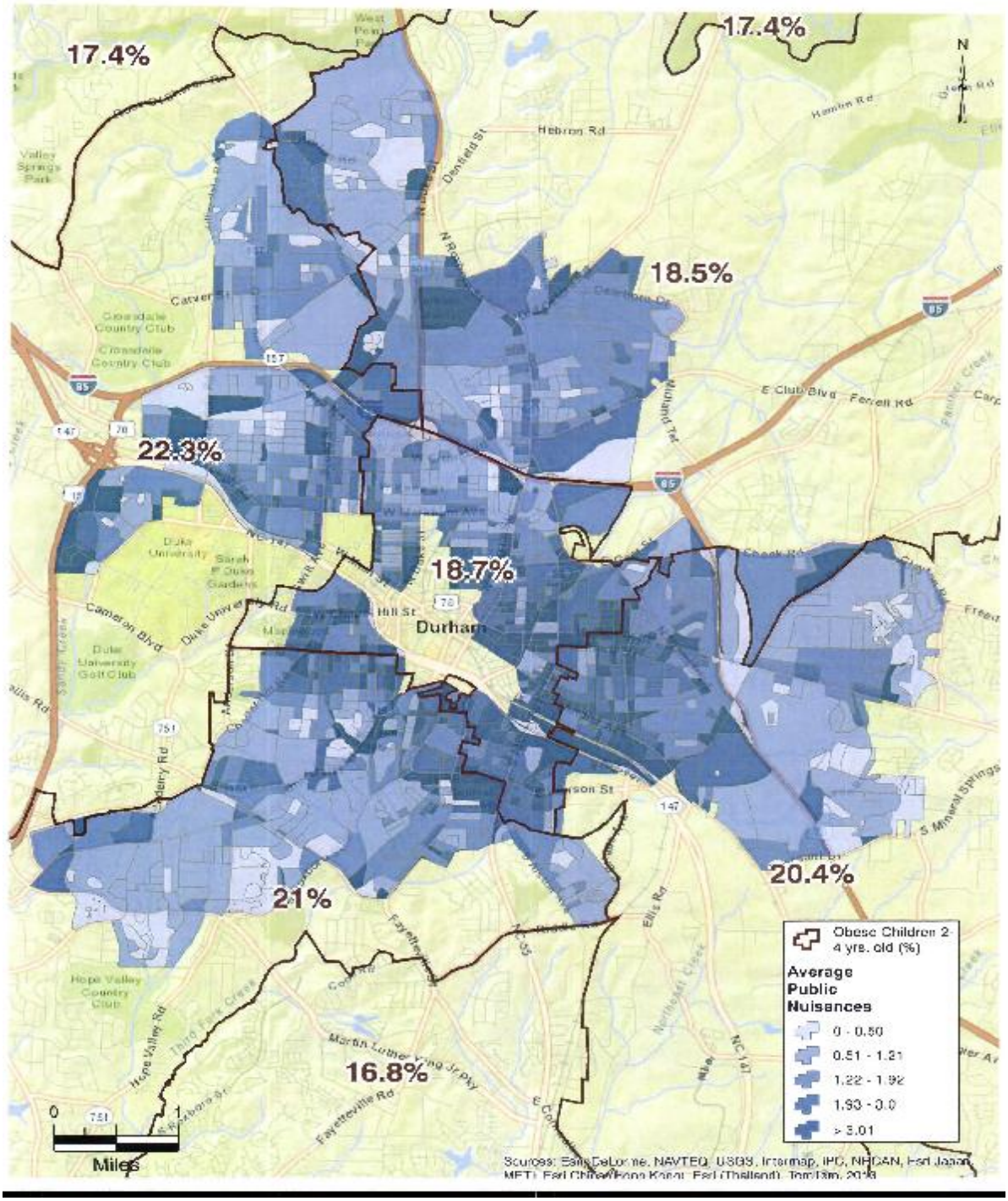
Appendix A6

Average Security Measures



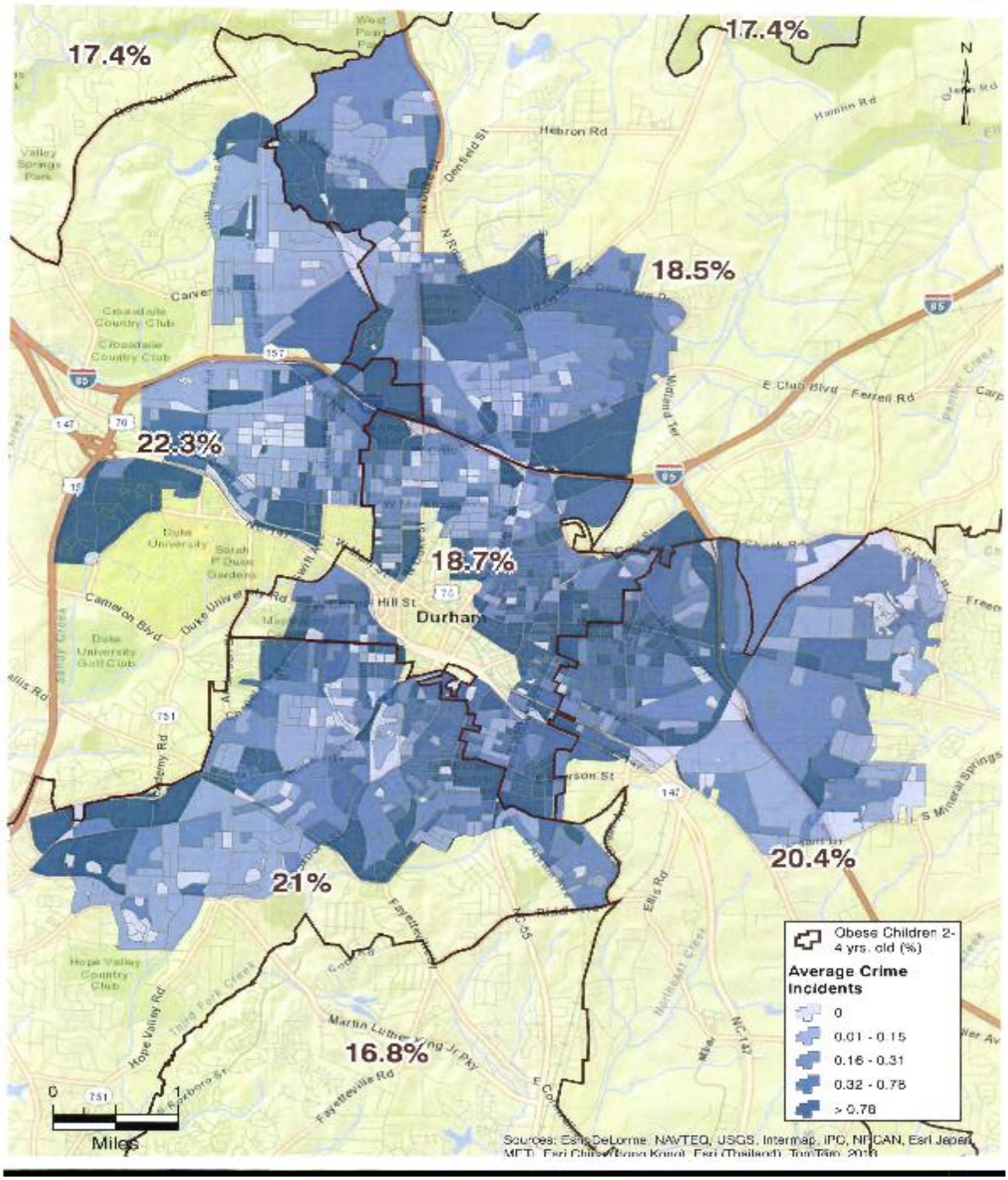
Appendix A7

Average Public Nuisances



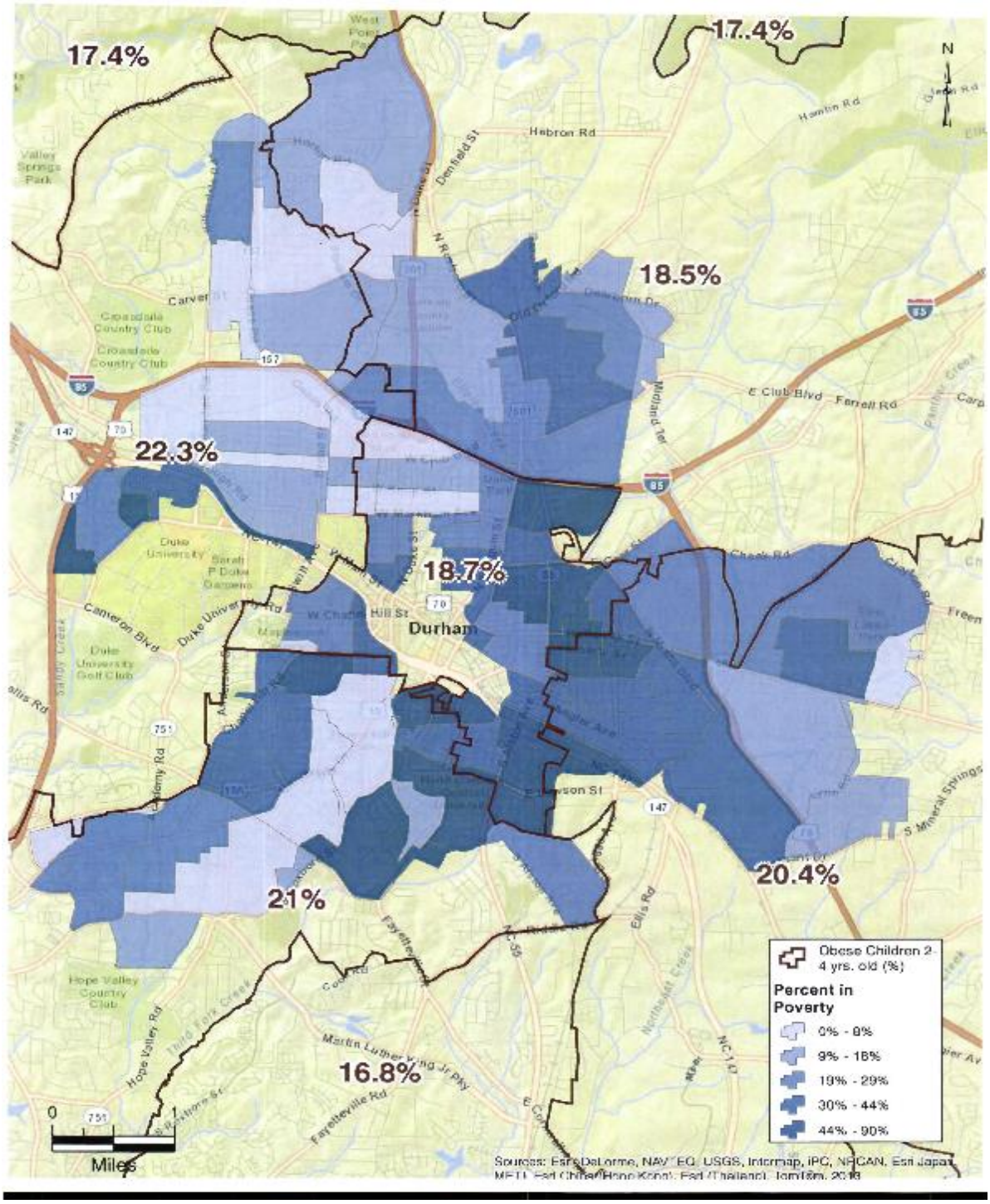
Appendix A8

Average Crime Incidences



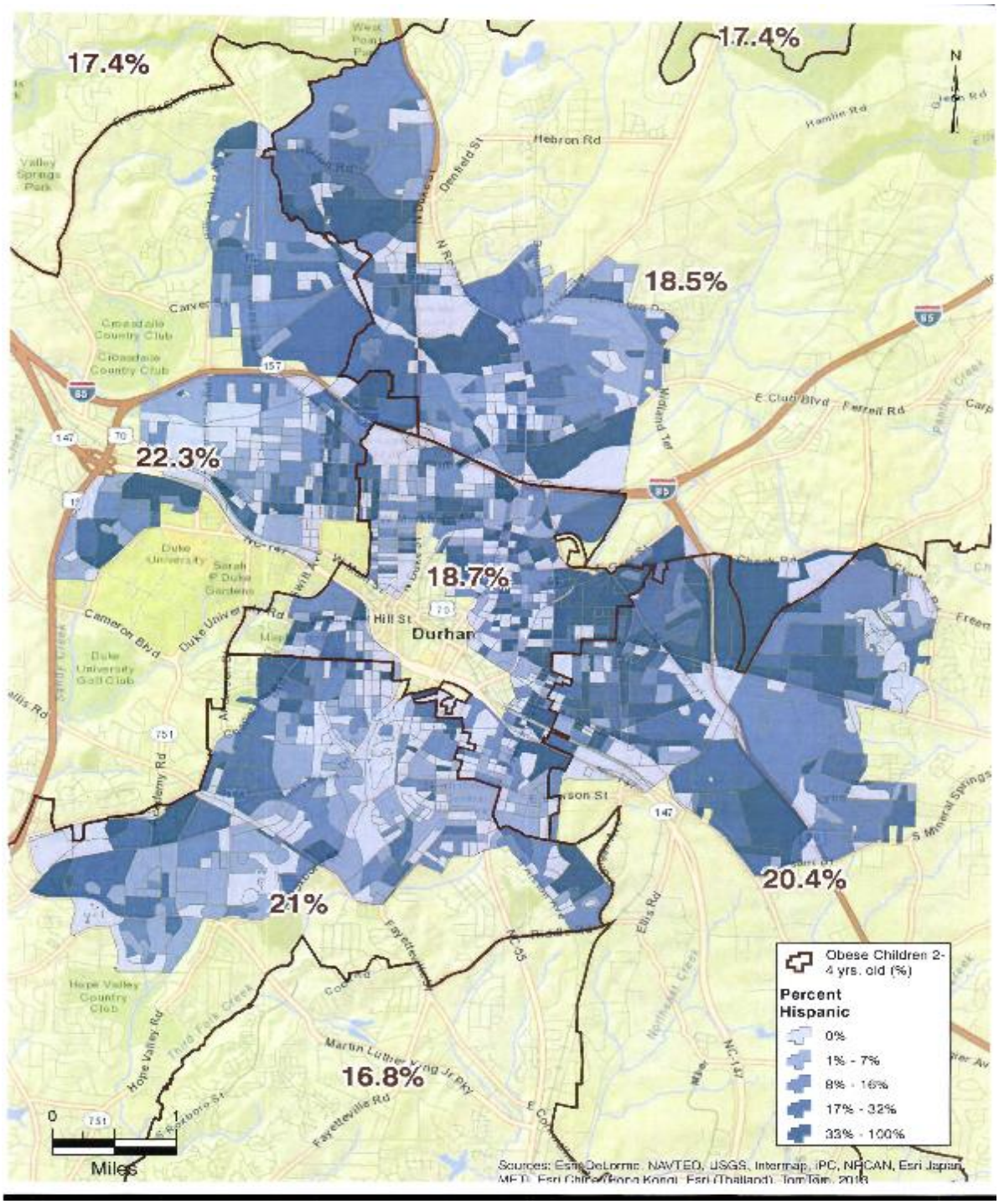
Appendix A9

Percent in Poverty



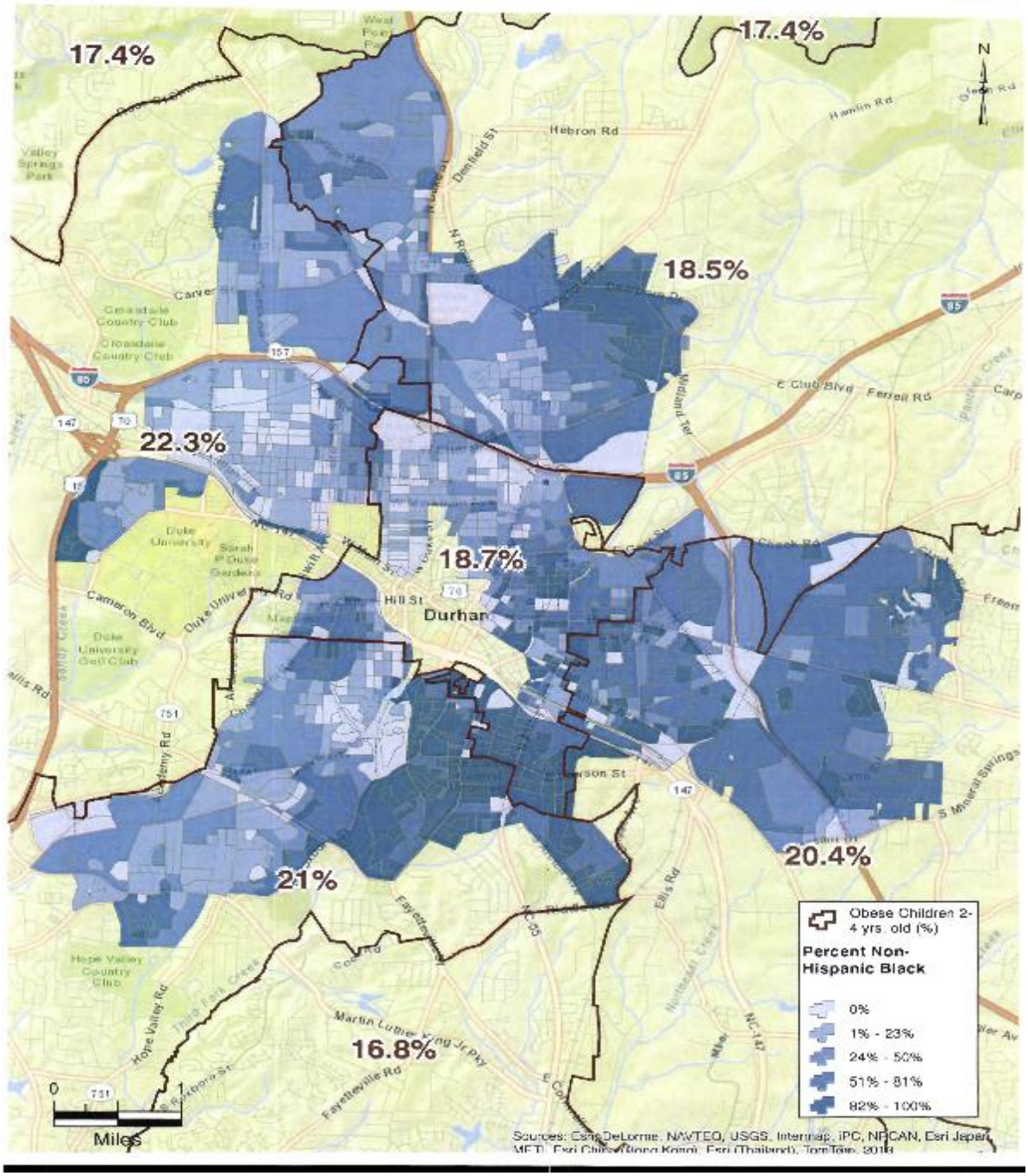
Appendix A10

Percent Hispanic



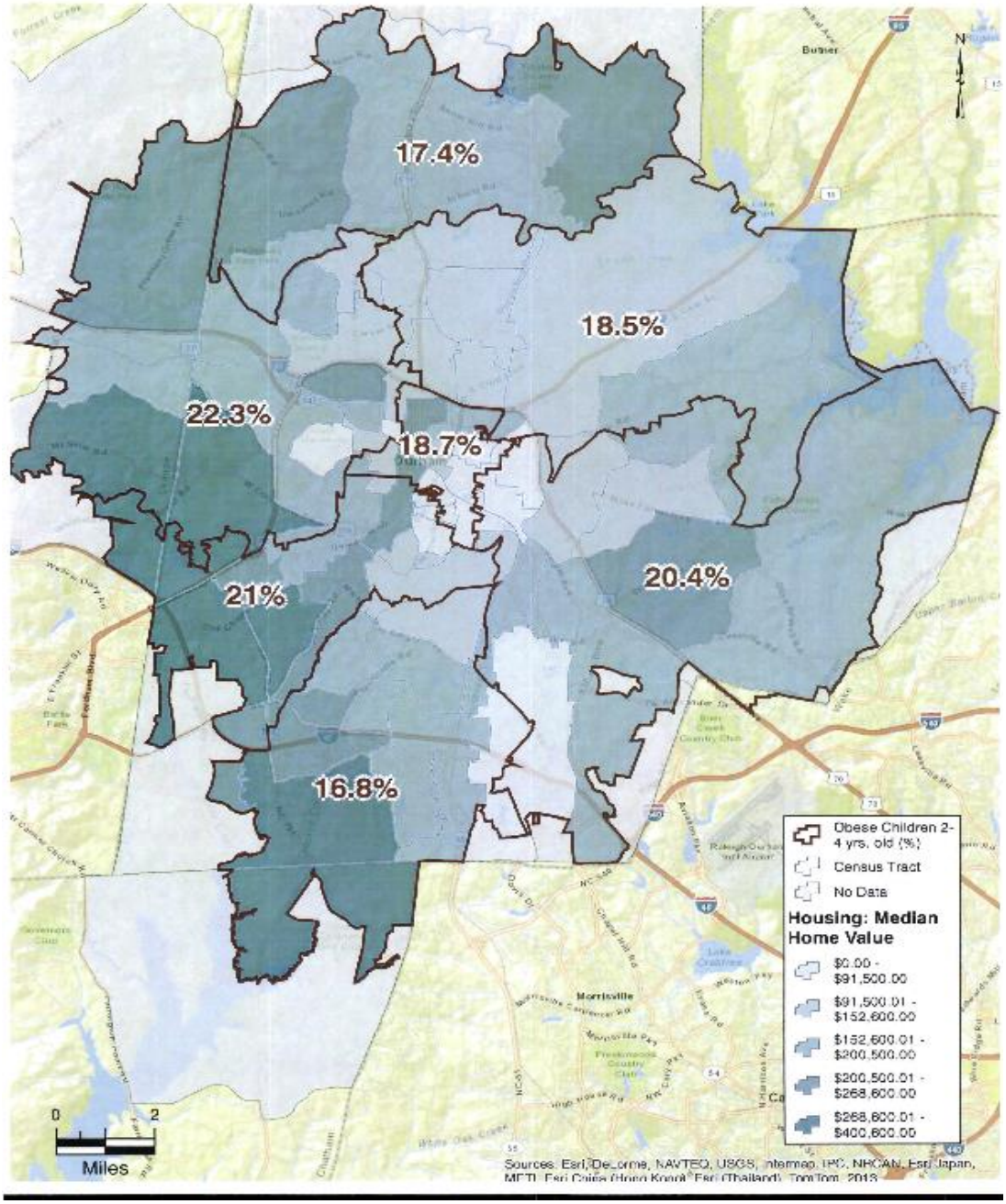
Appendix A11

Percent Non-Hispanic Black



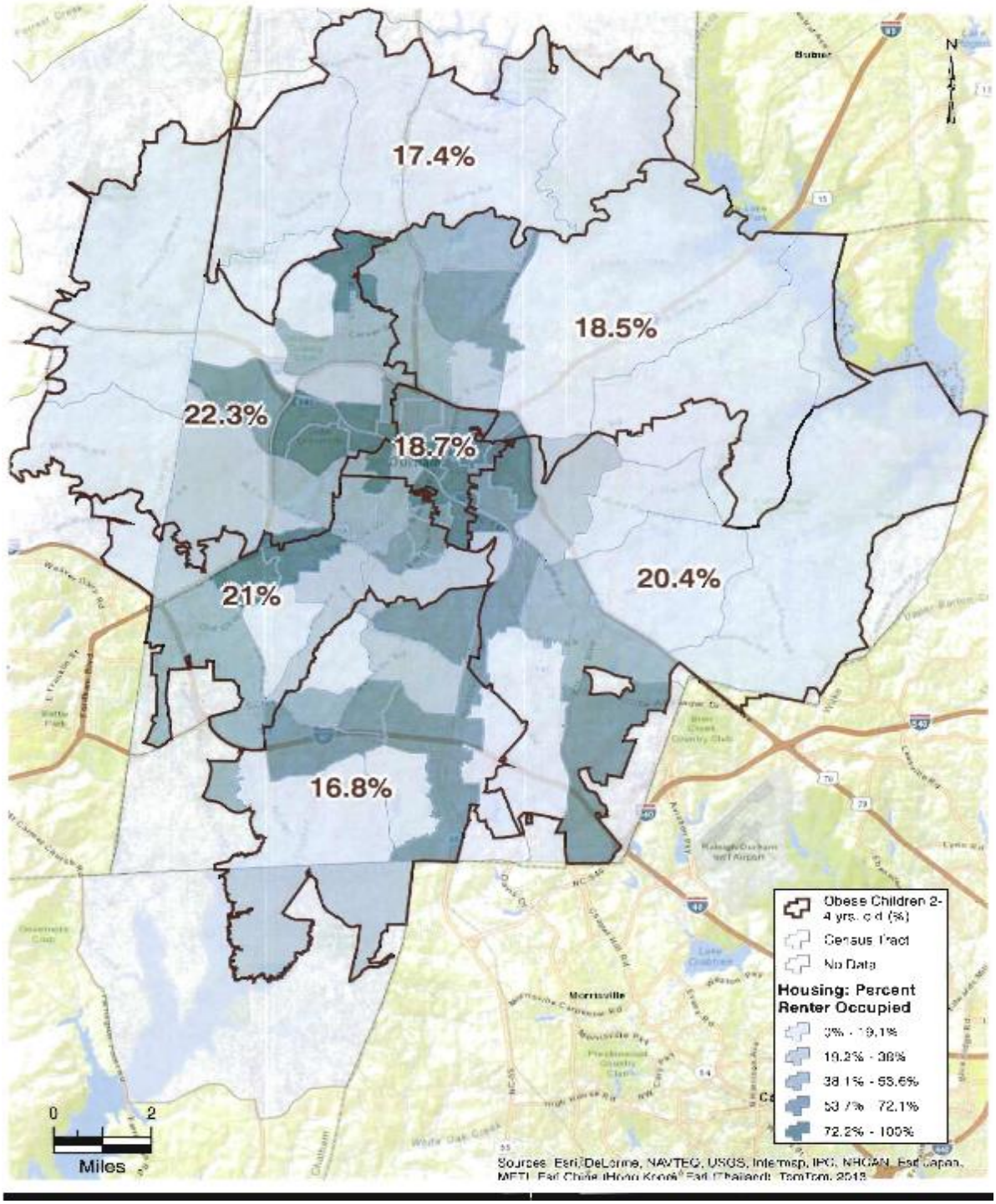
Appendix A12

Housing: Median Home Value



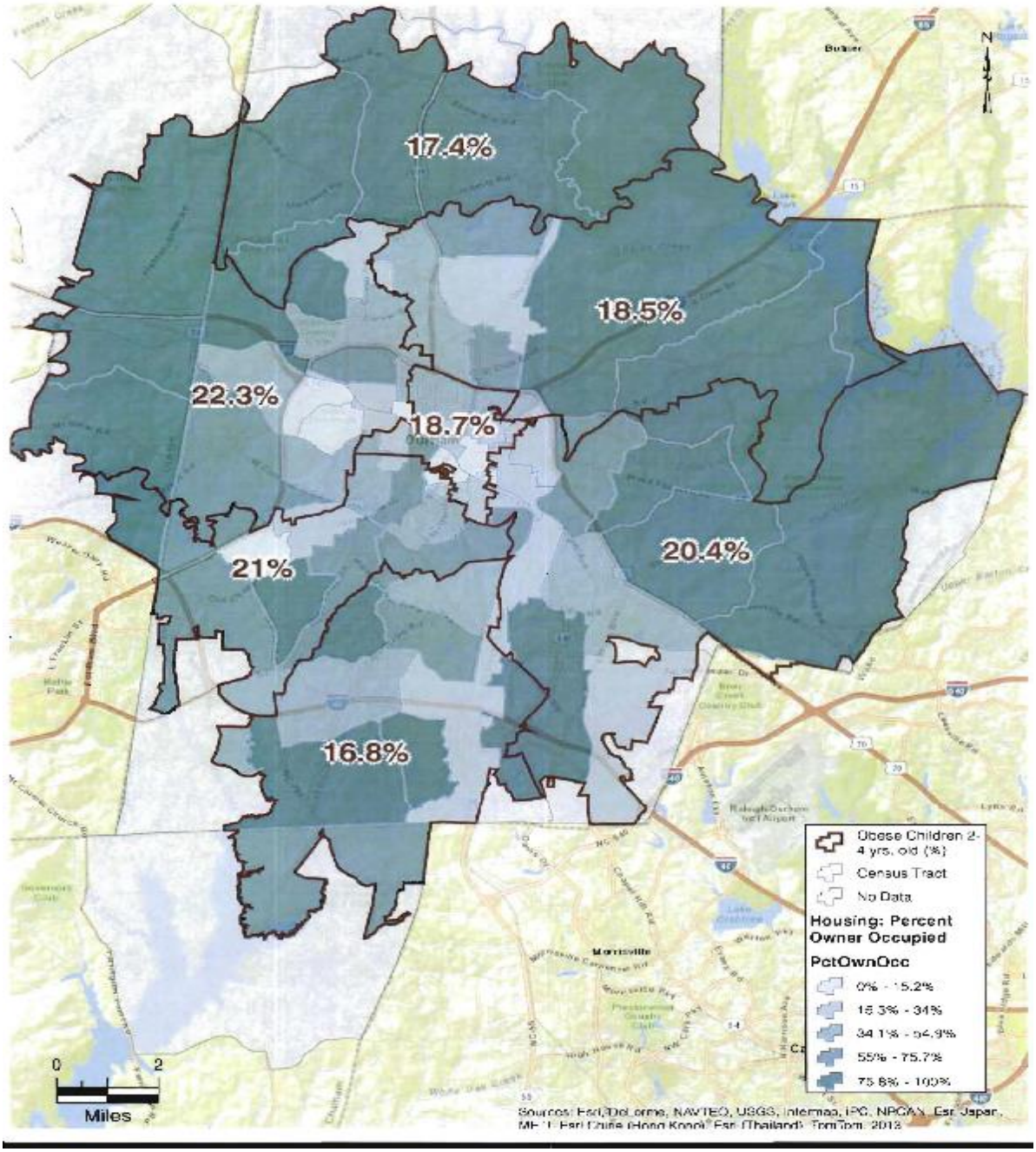
Appendix A13

Housing: Percent Renter Occupied



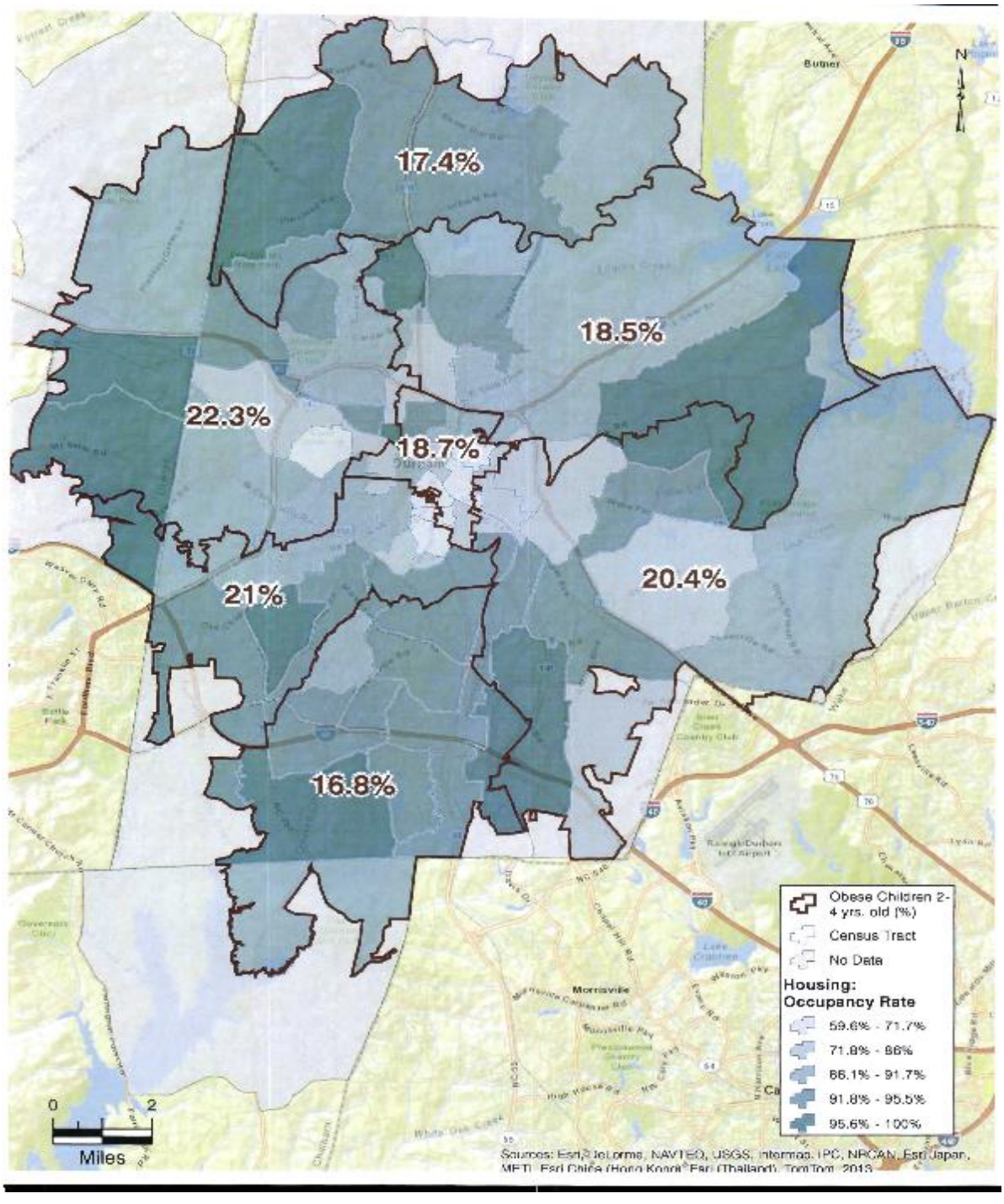
Appendix A14

Housing Percent Owner Occupied



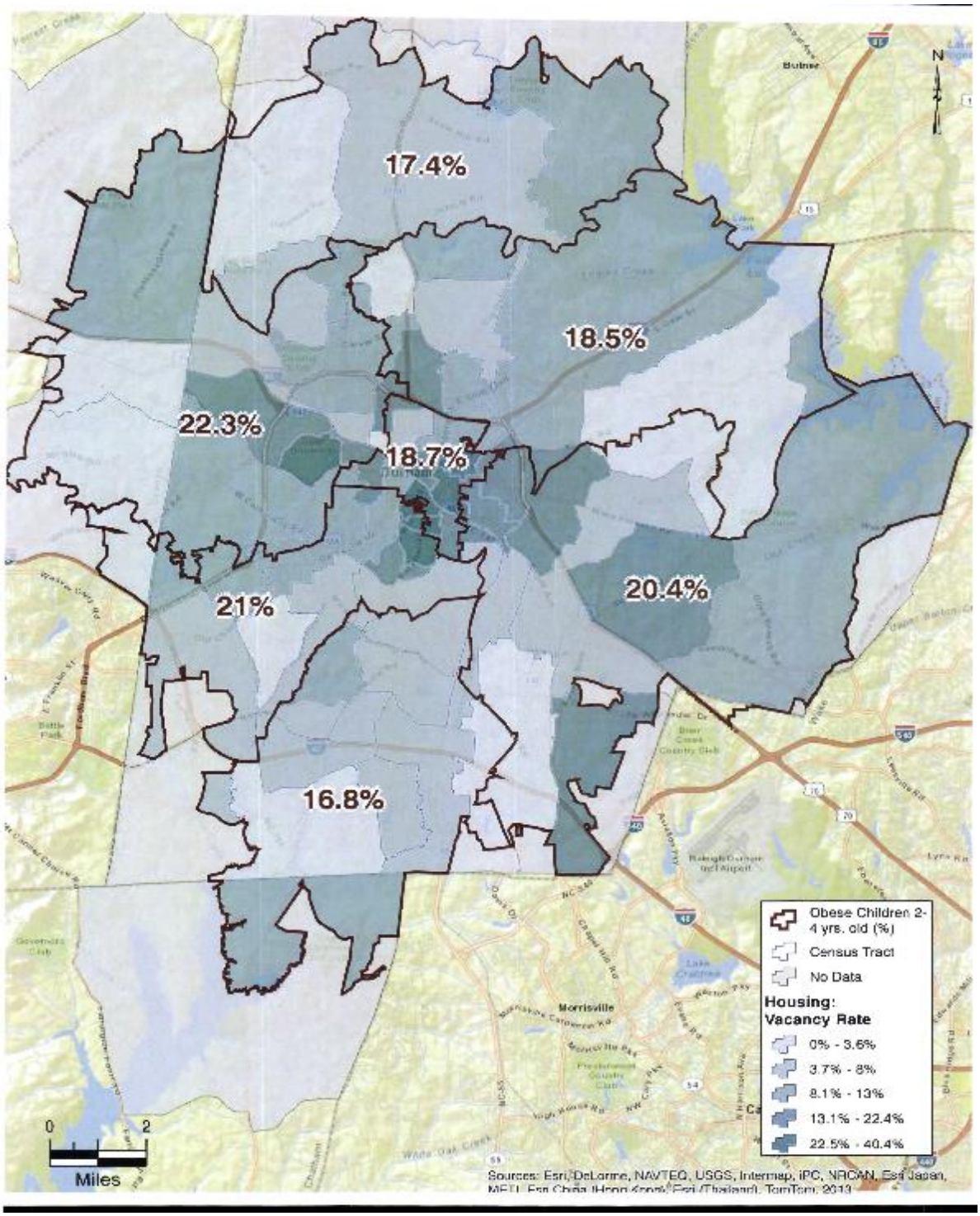
Appendix A15

Occupancy Rate



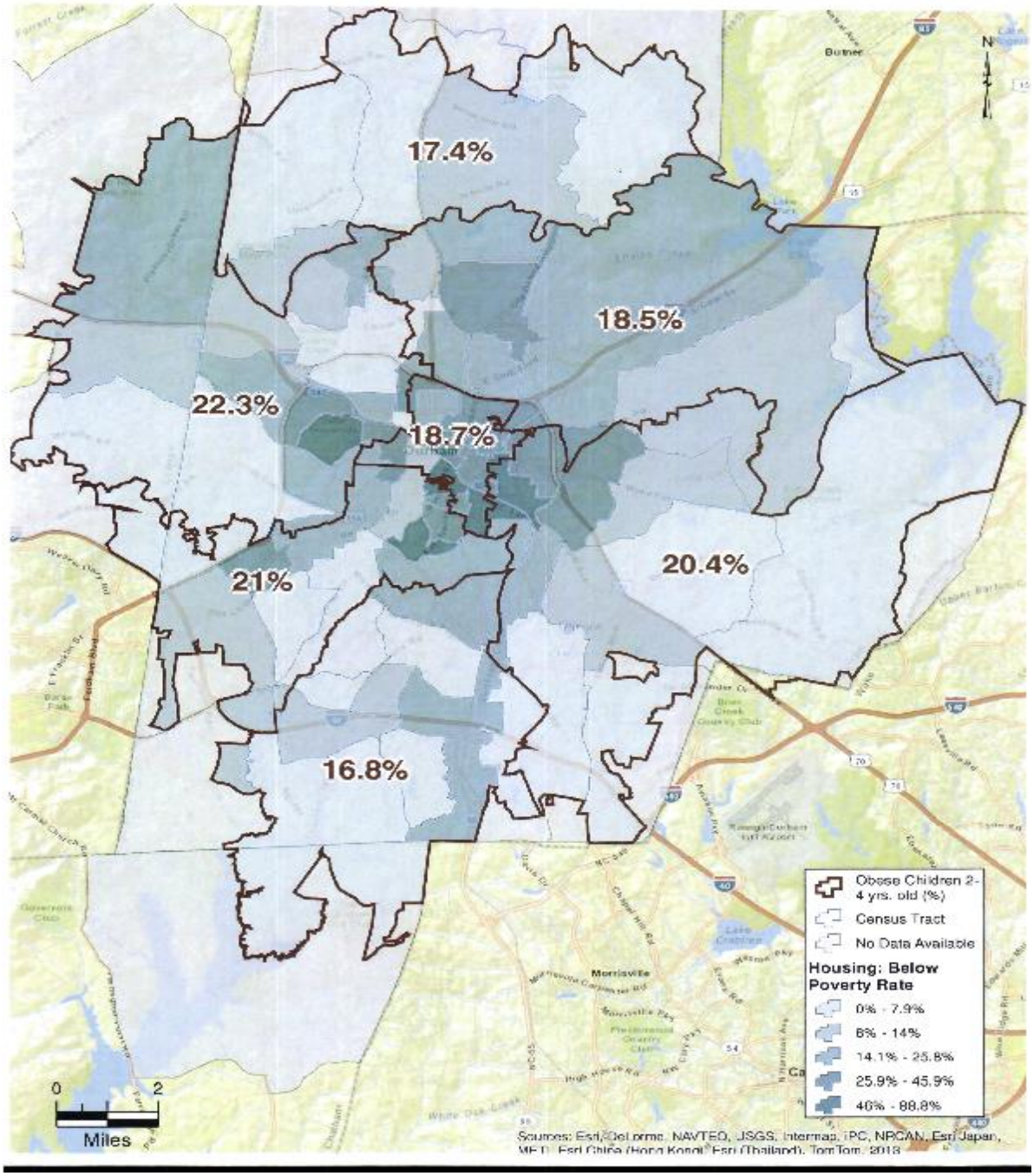
Appendix A16

Housing: Vacancy Rate



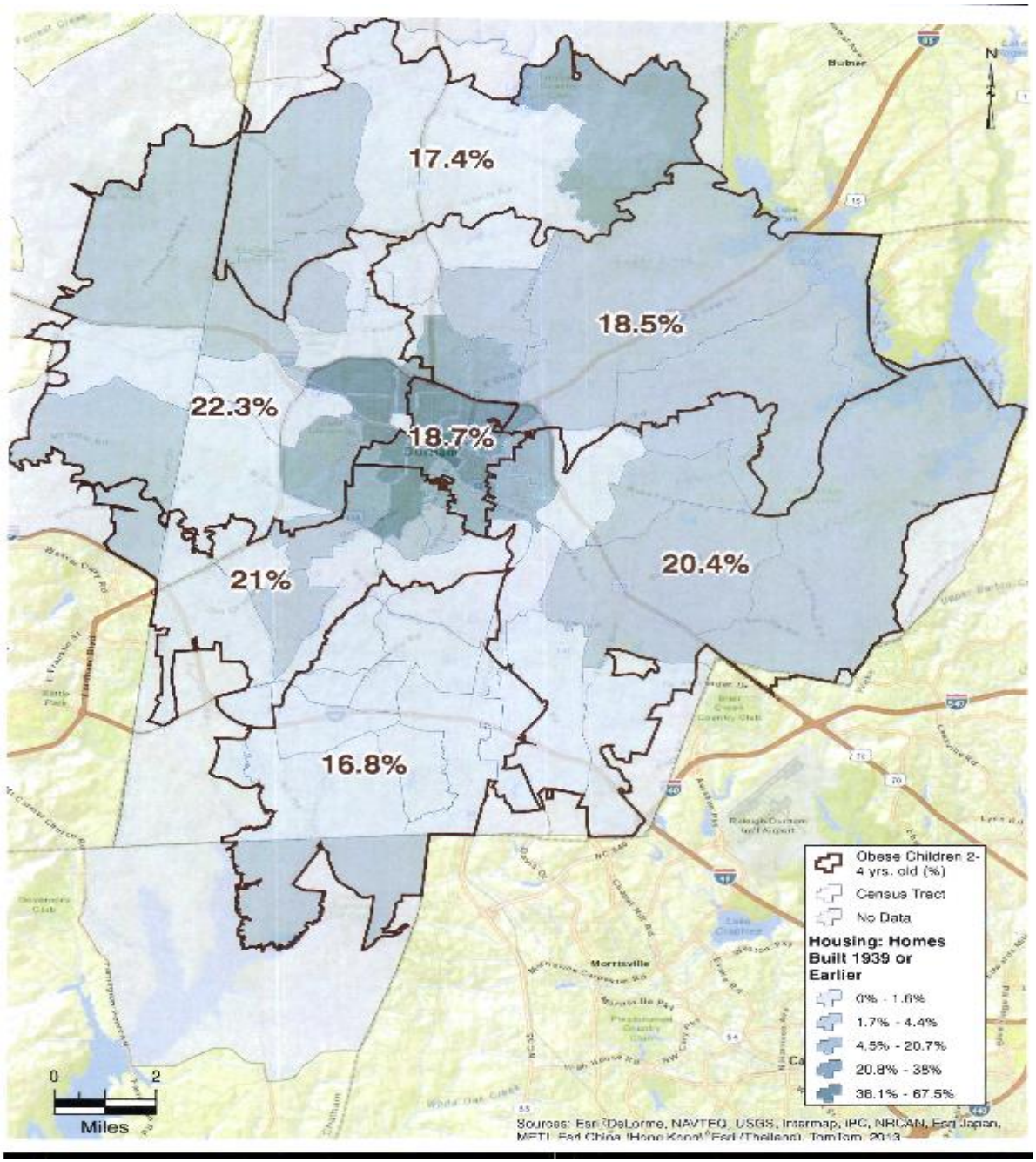
Appendix A17

Housing: Below Poverty Level



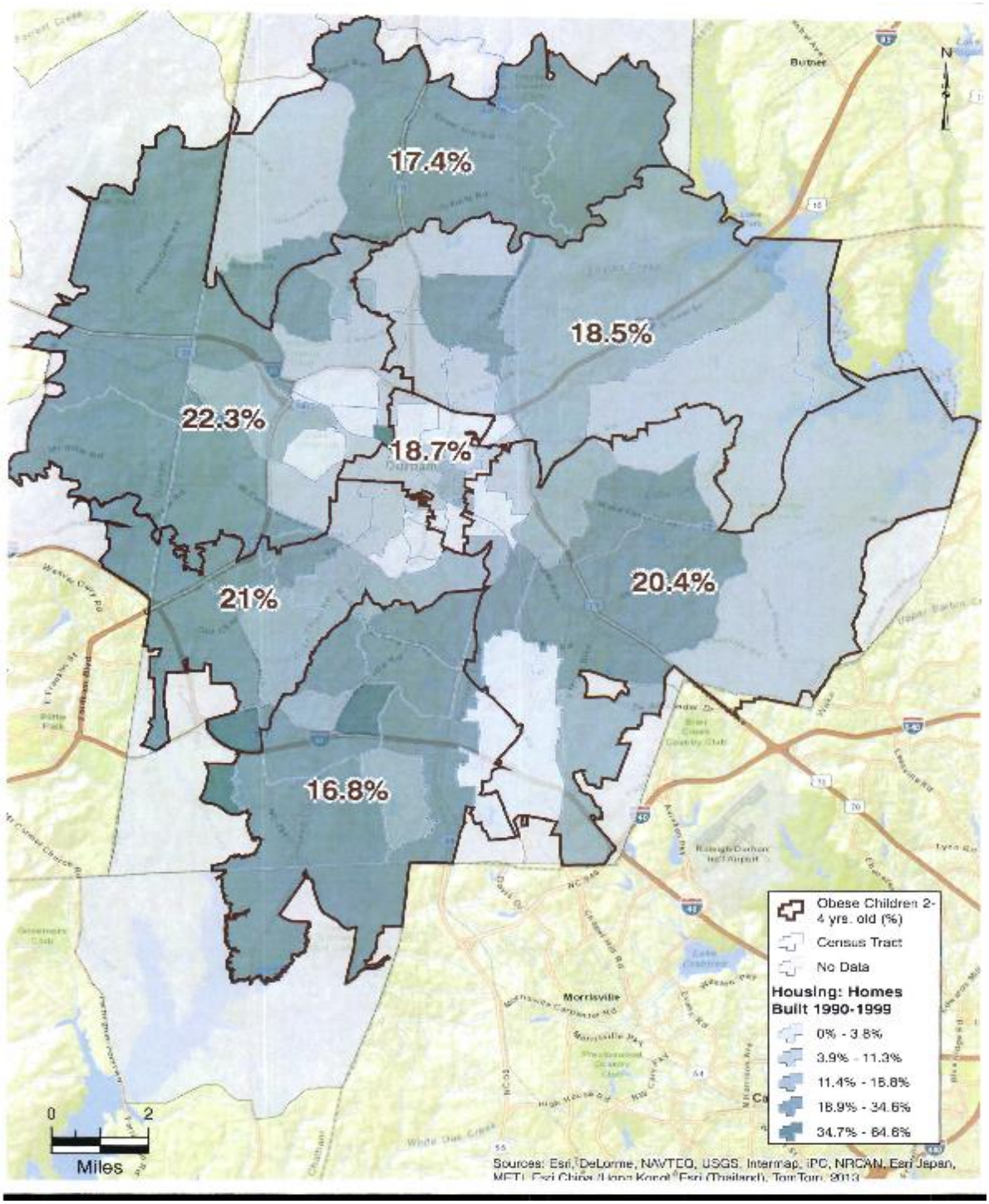
Appendix A18

Homes Built 1939 or Earlier



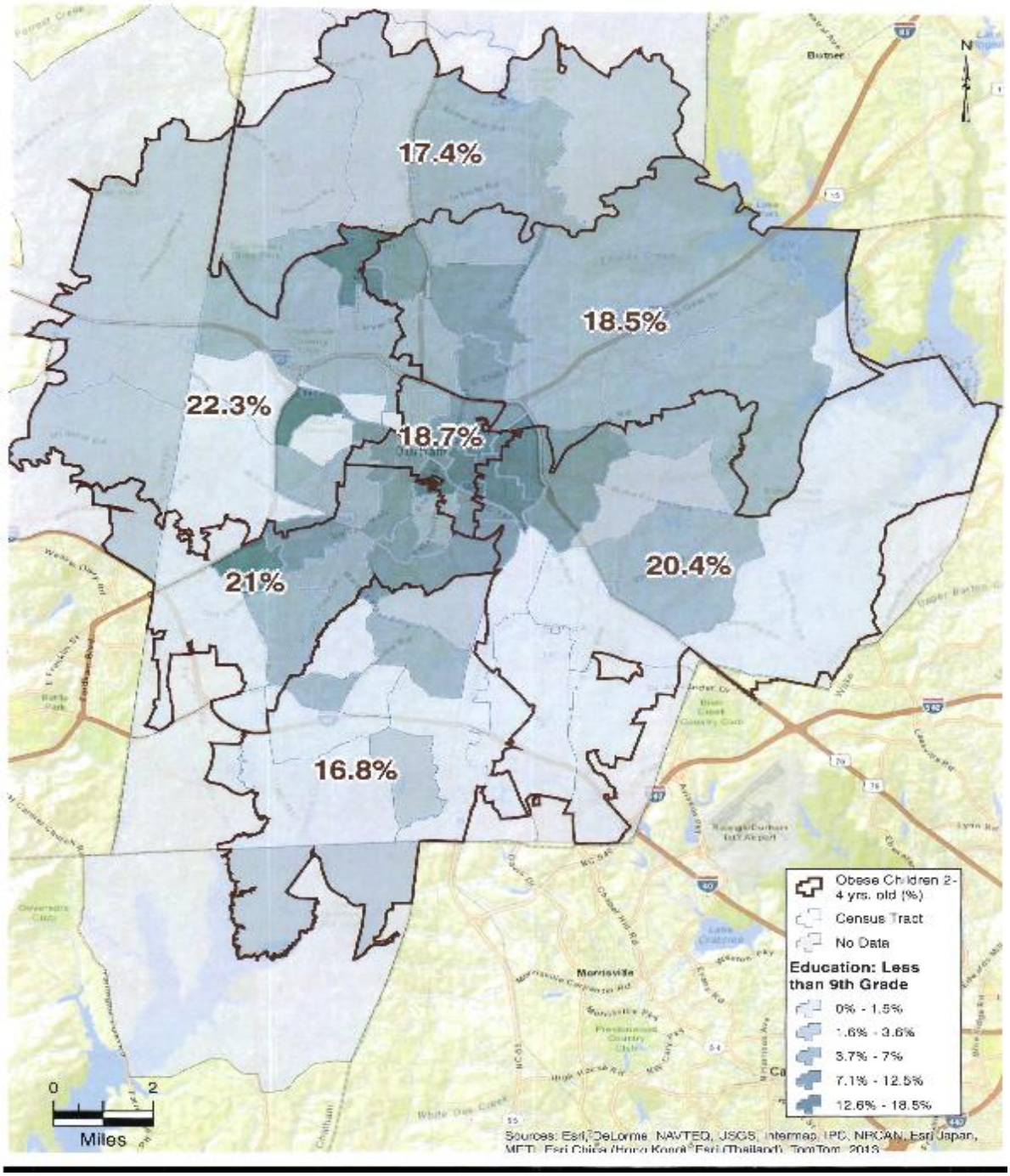
Appendix A19

Homes Built 1990 - 1999



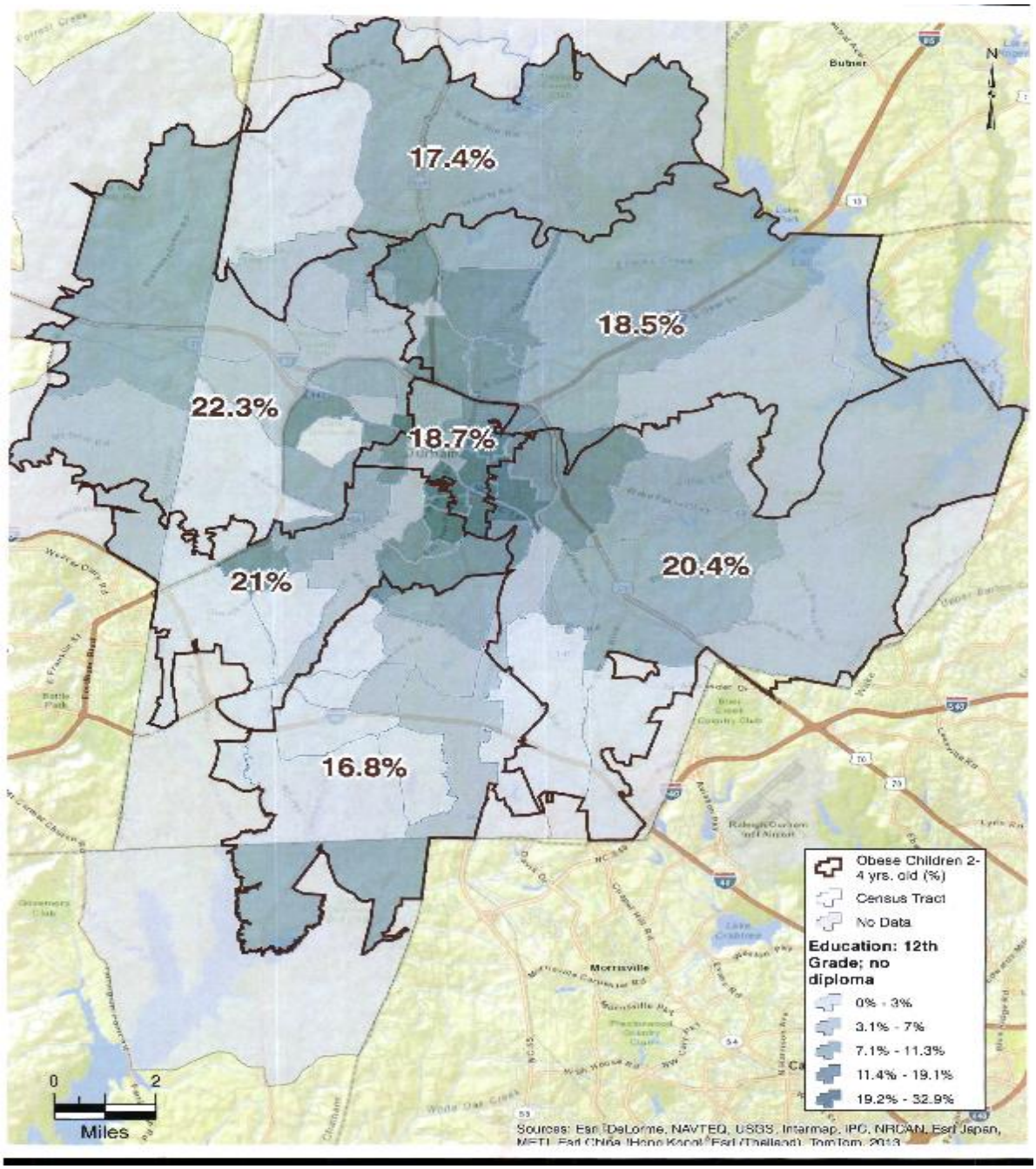
Appendix A20

Education: Less than 9th Grade



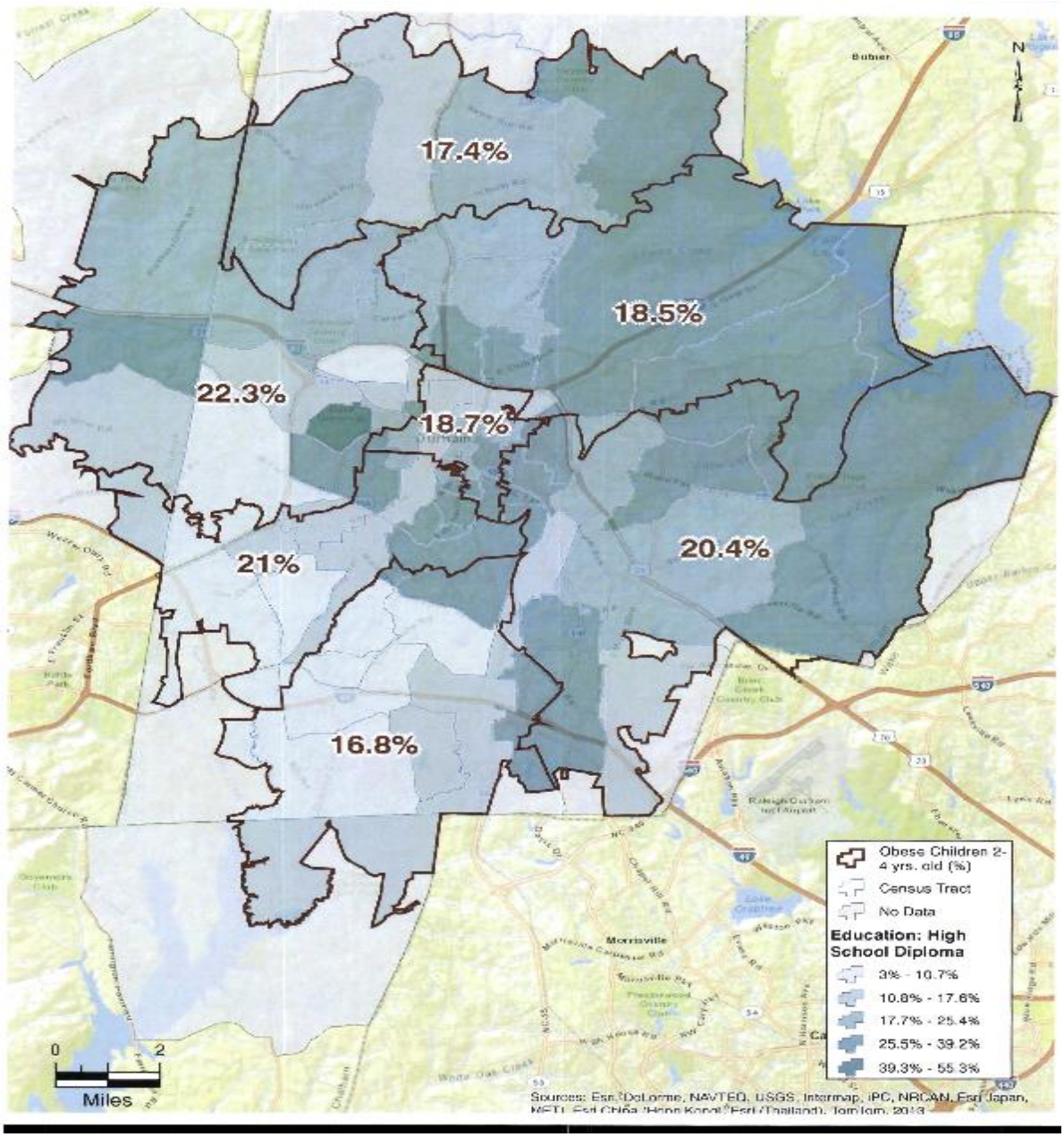
Appendix A21

Education: 12th Grade No Diploma



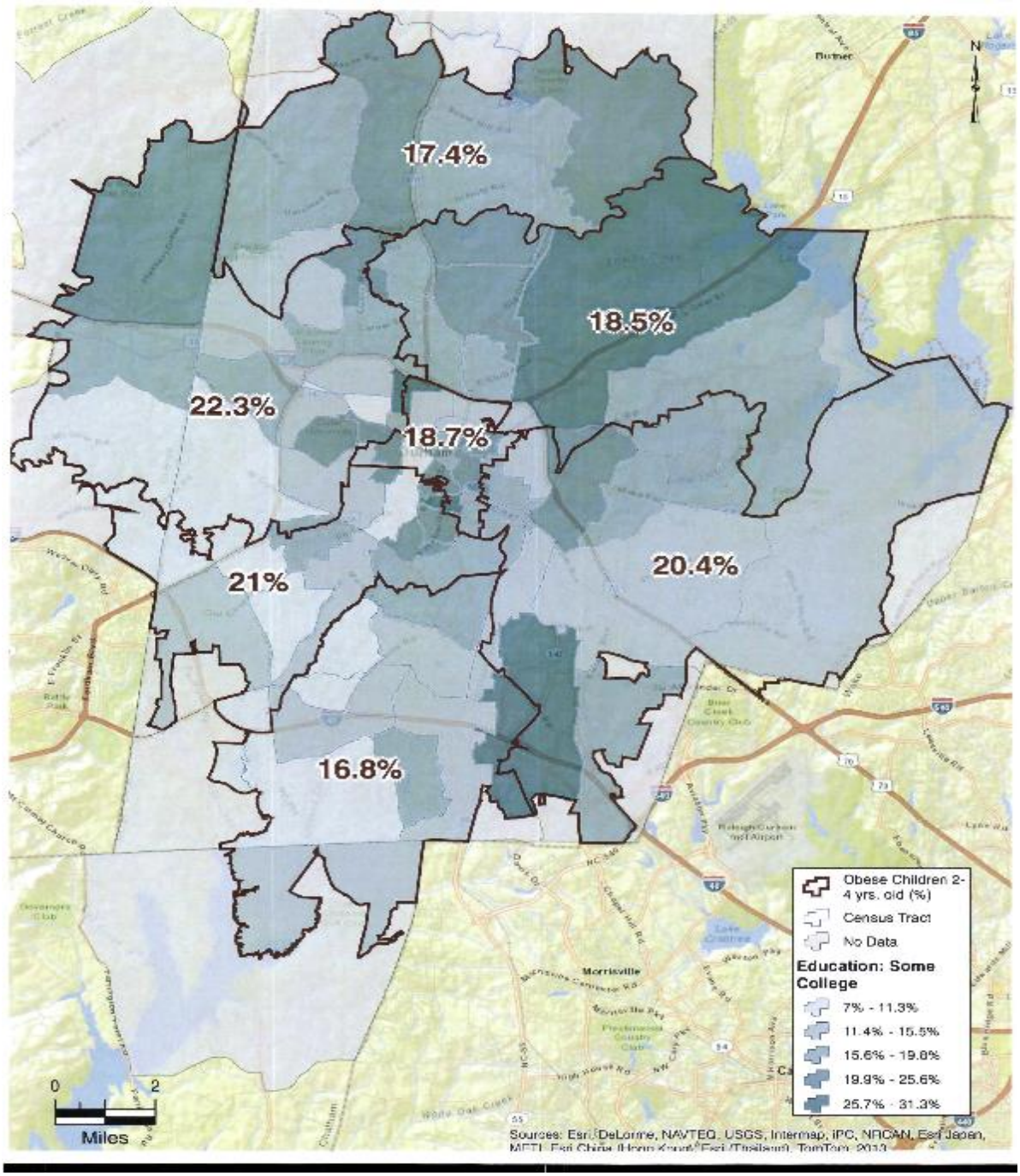
Appendix A22

Education: High School Diploma



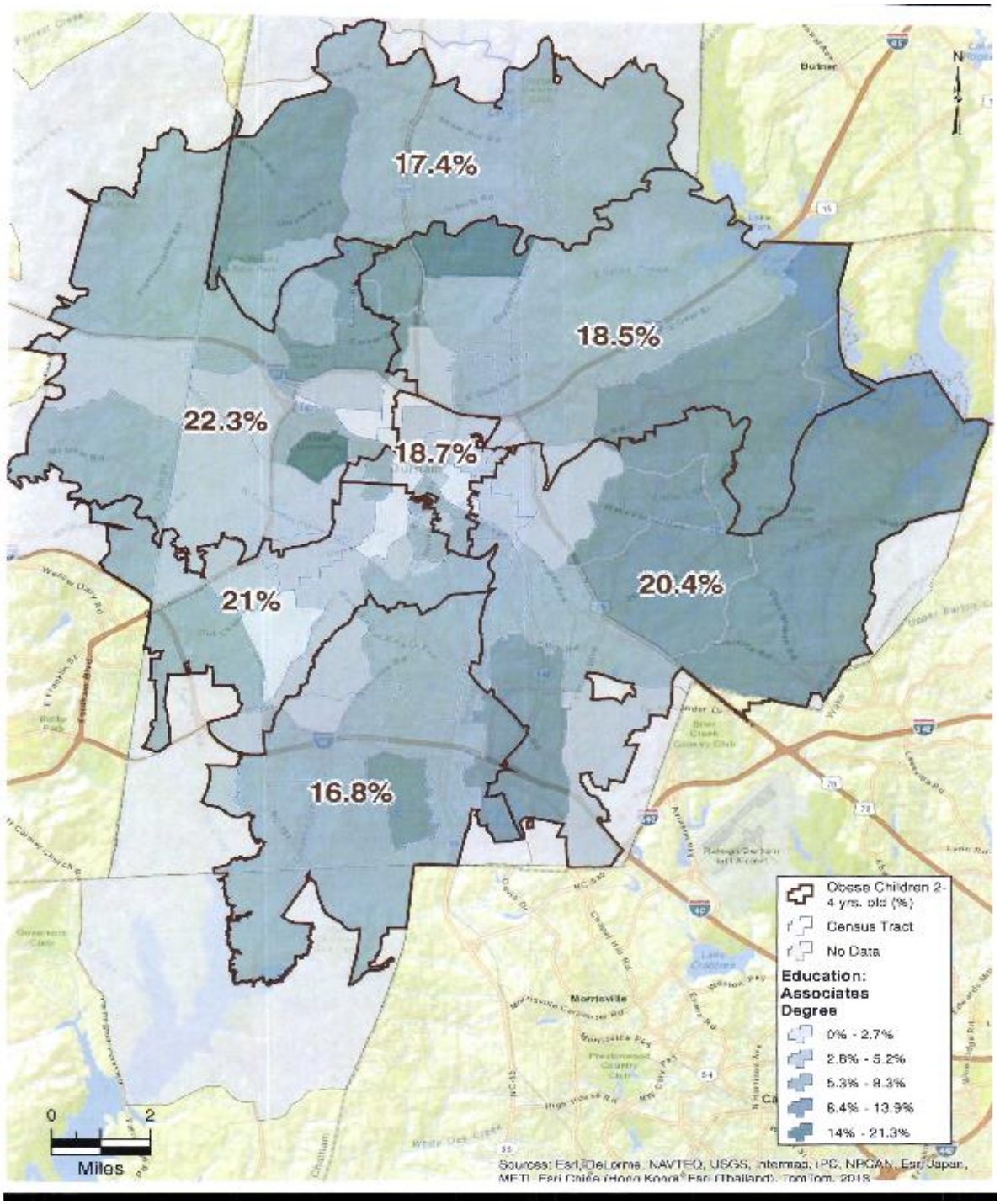
Appendix A23

Education: Some College



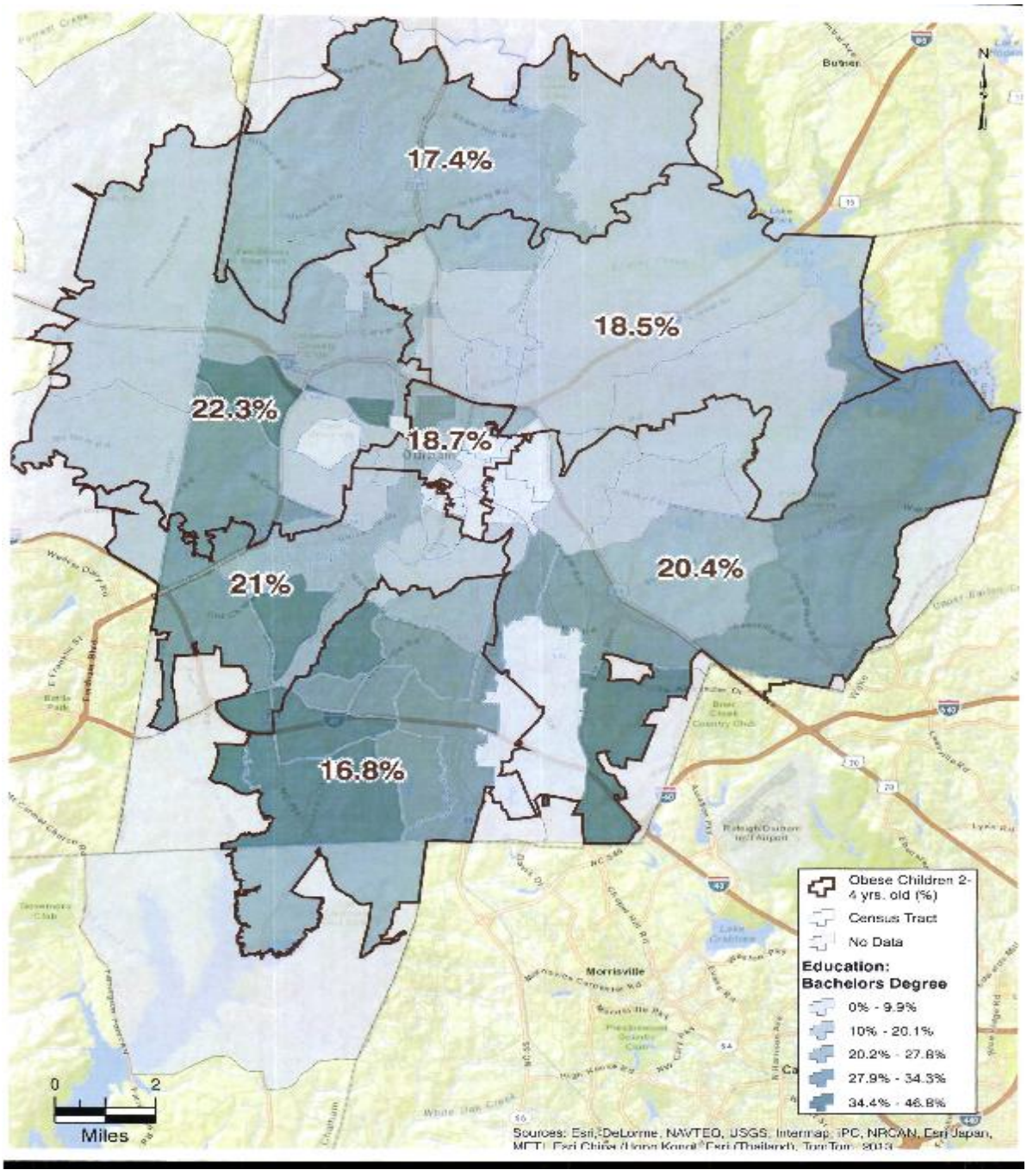
Appendix A24

Associates Degree



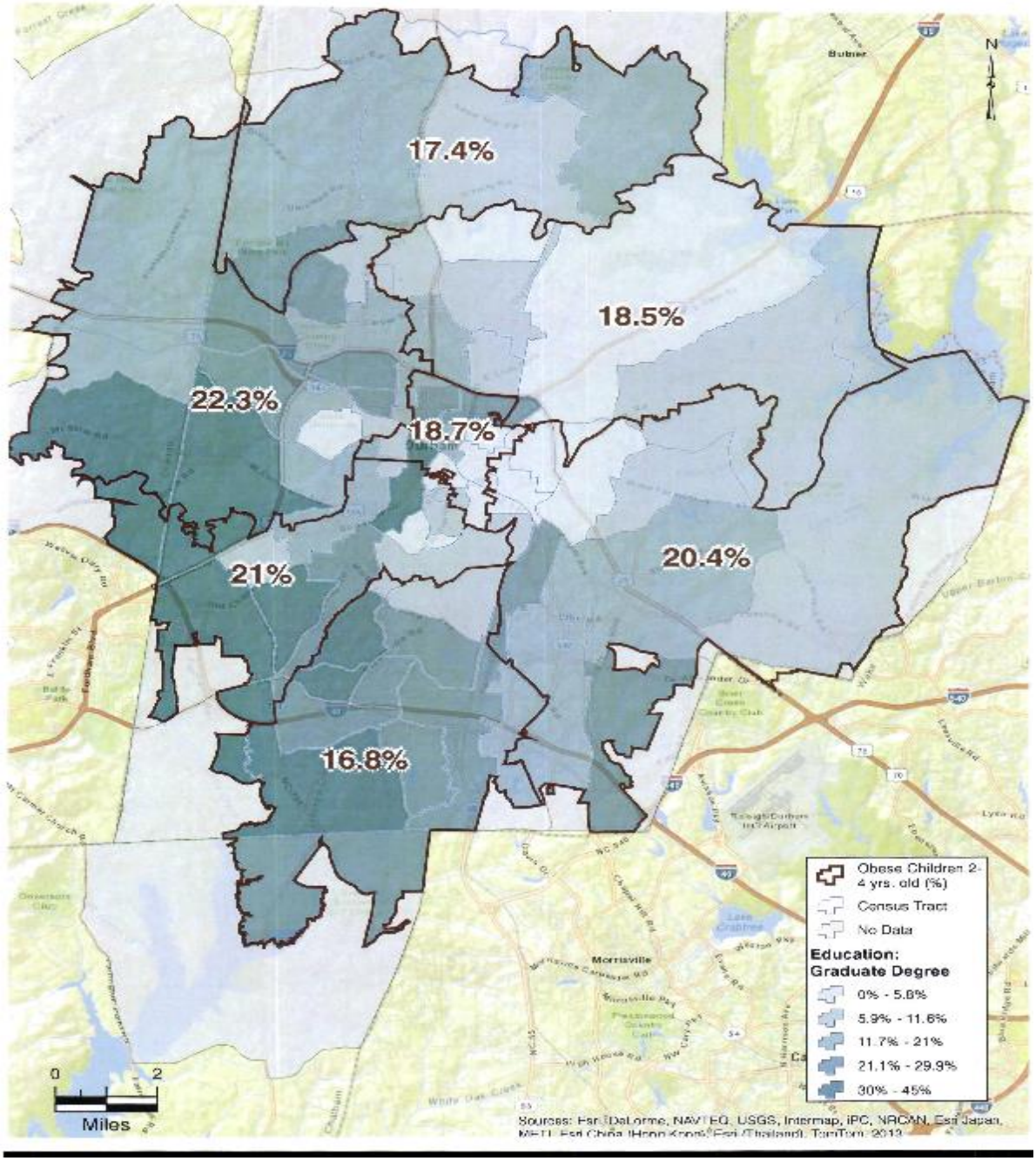
Appendix A25

Bachelor's Degree



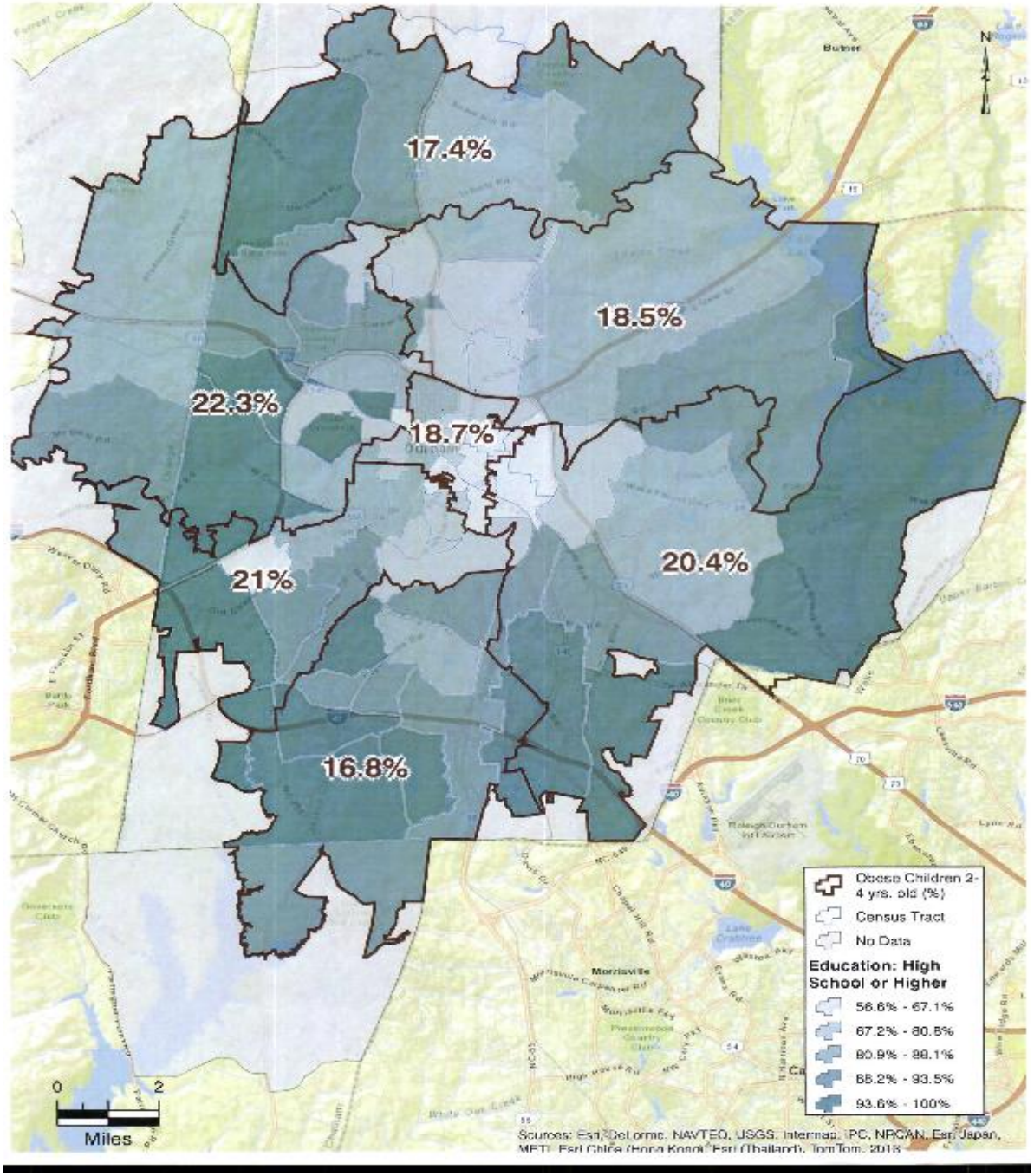
Appendix A26

Graduate Degree



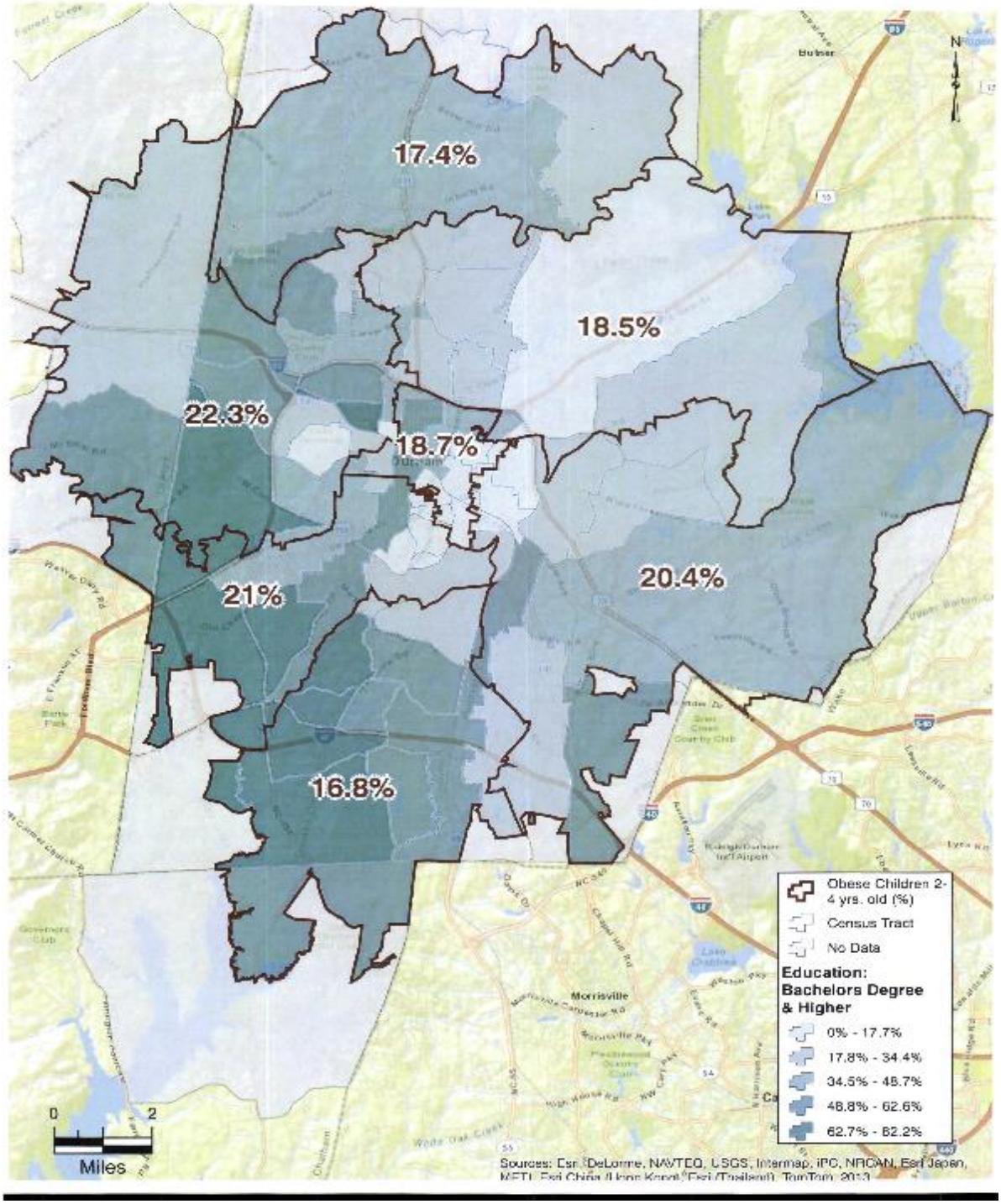
Appendix A27

High School of Higher



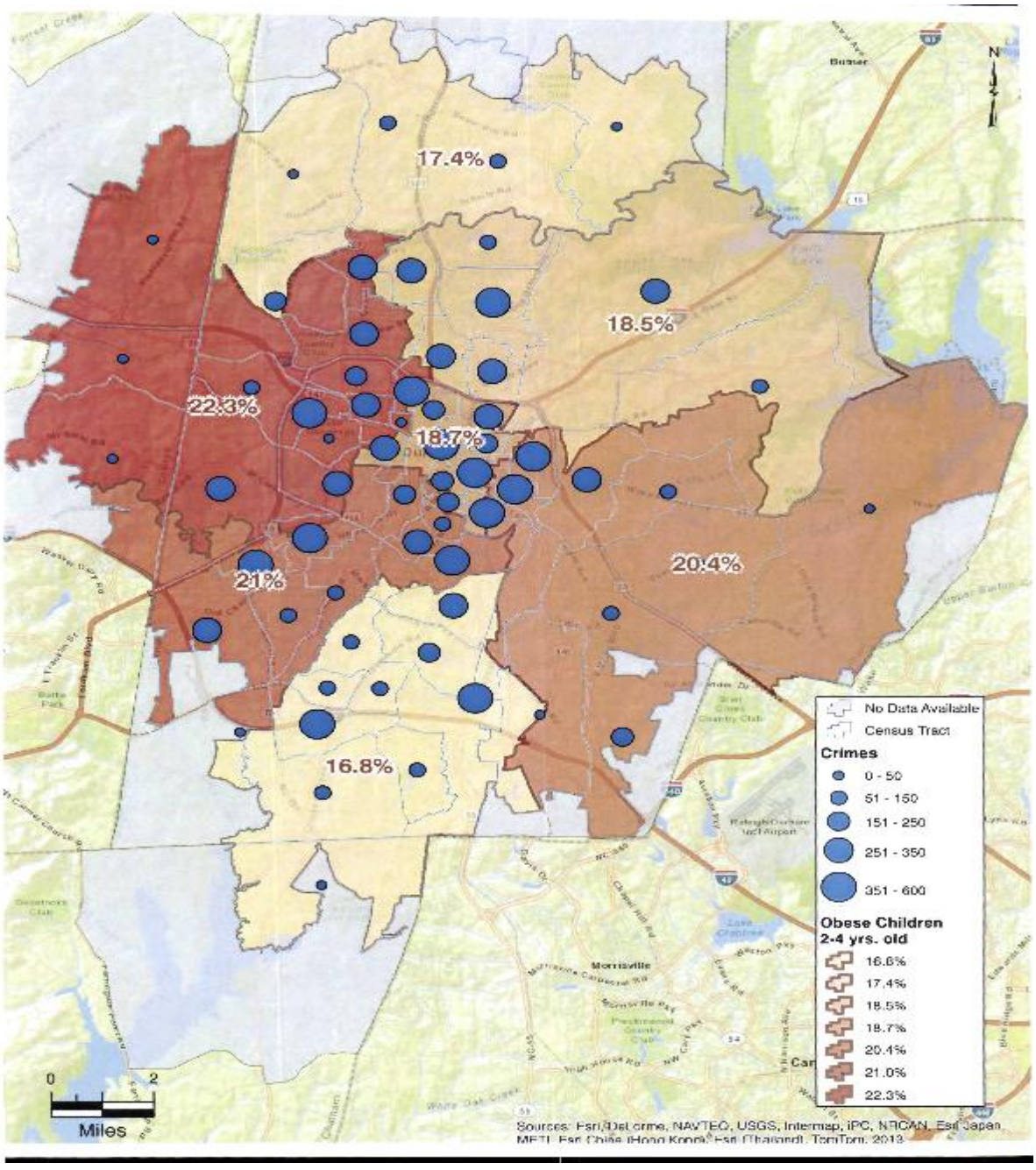
Appendix A28

Bachelor's Degree of Higher



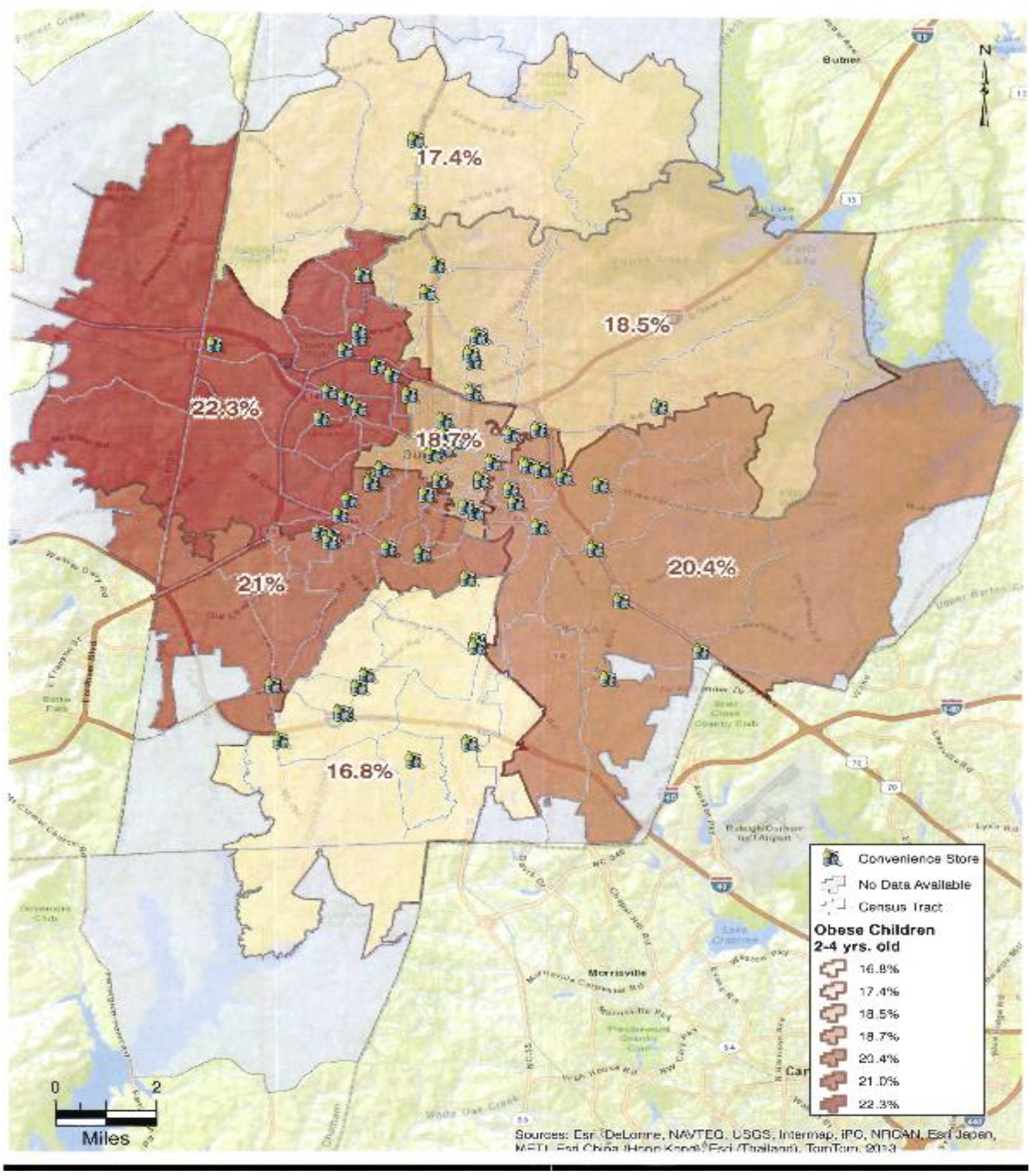
Appendix A29

Crimes



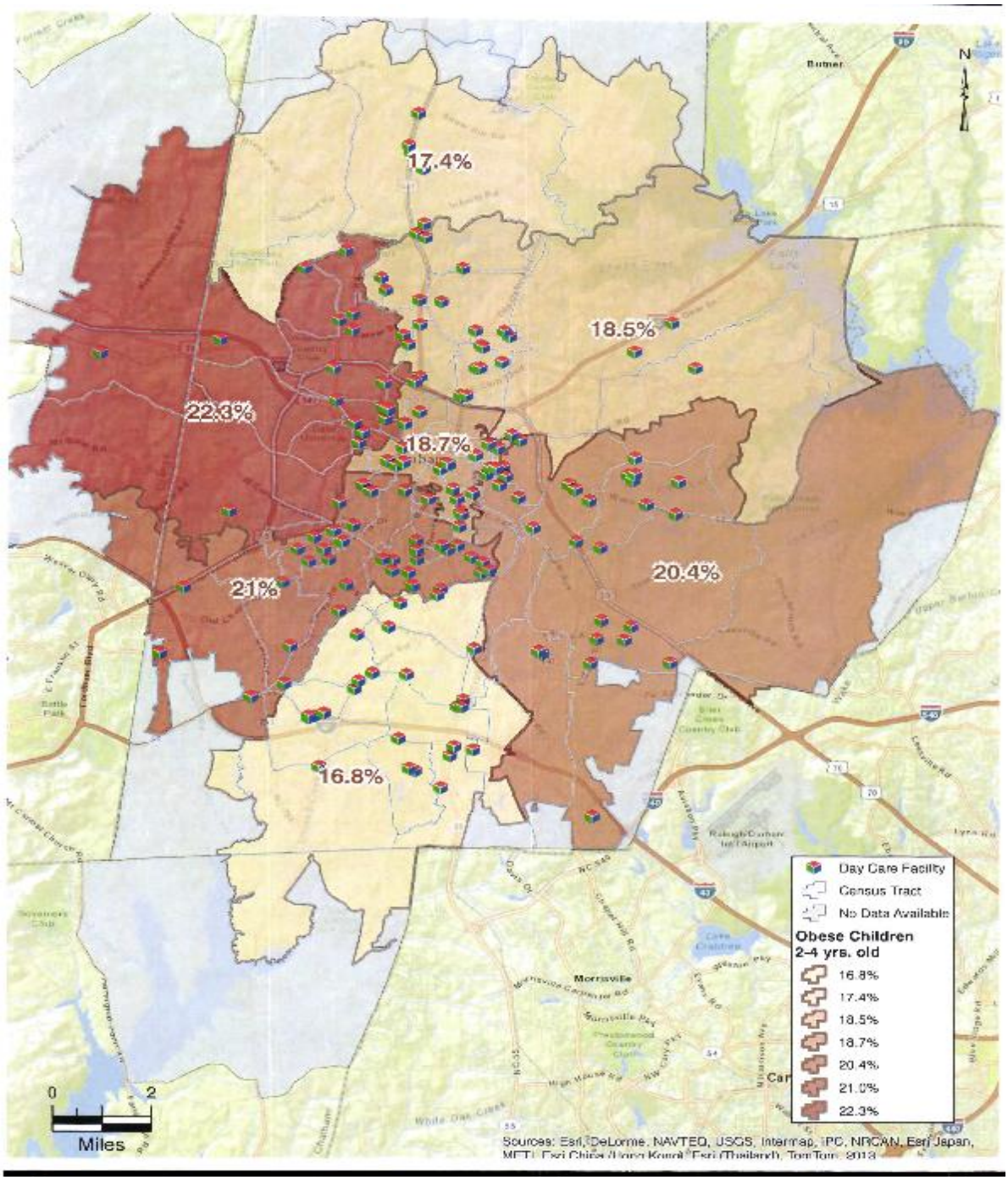
Appendix A30

Convenience Stores



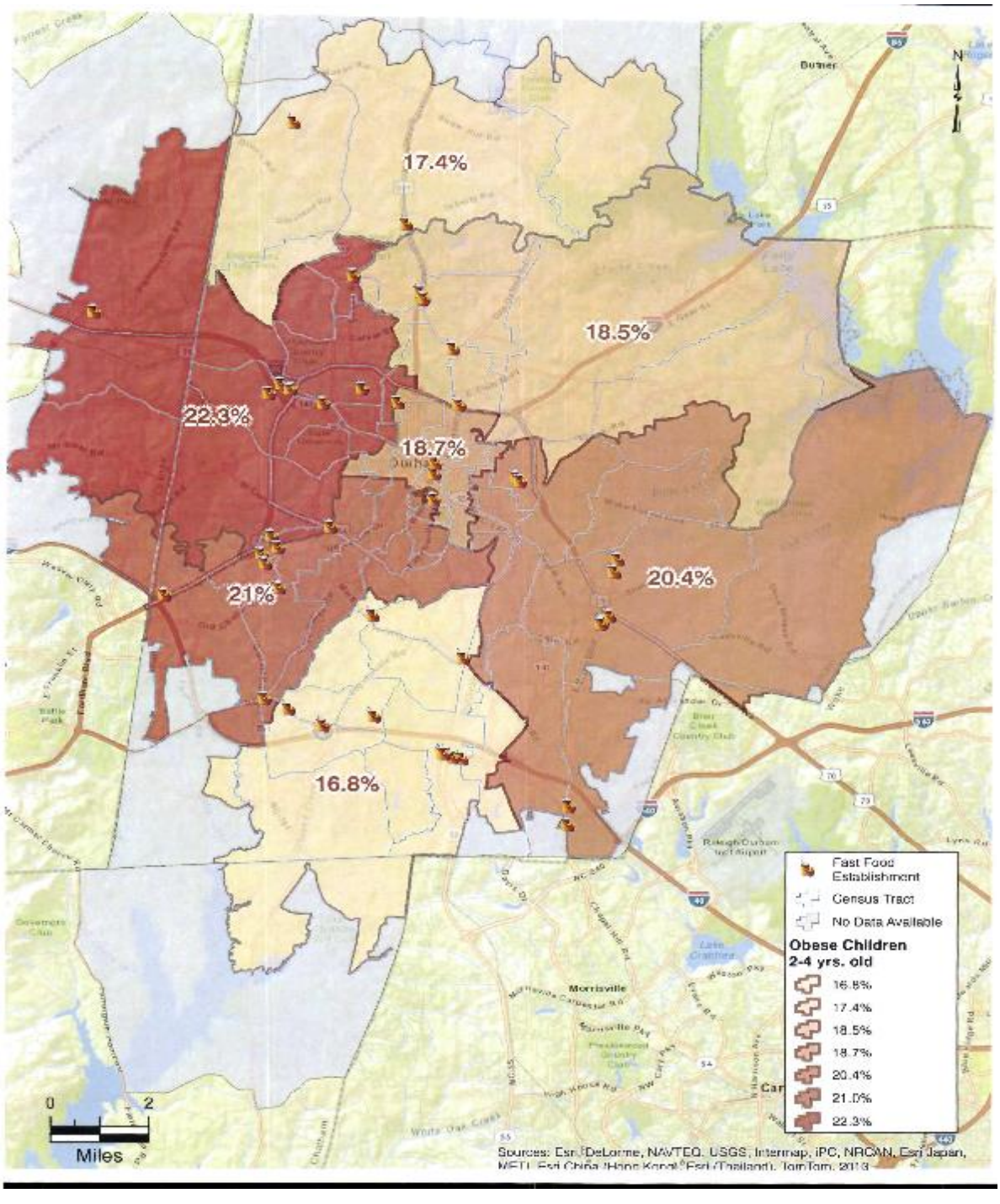
Appendix A31

Day Care Facilities



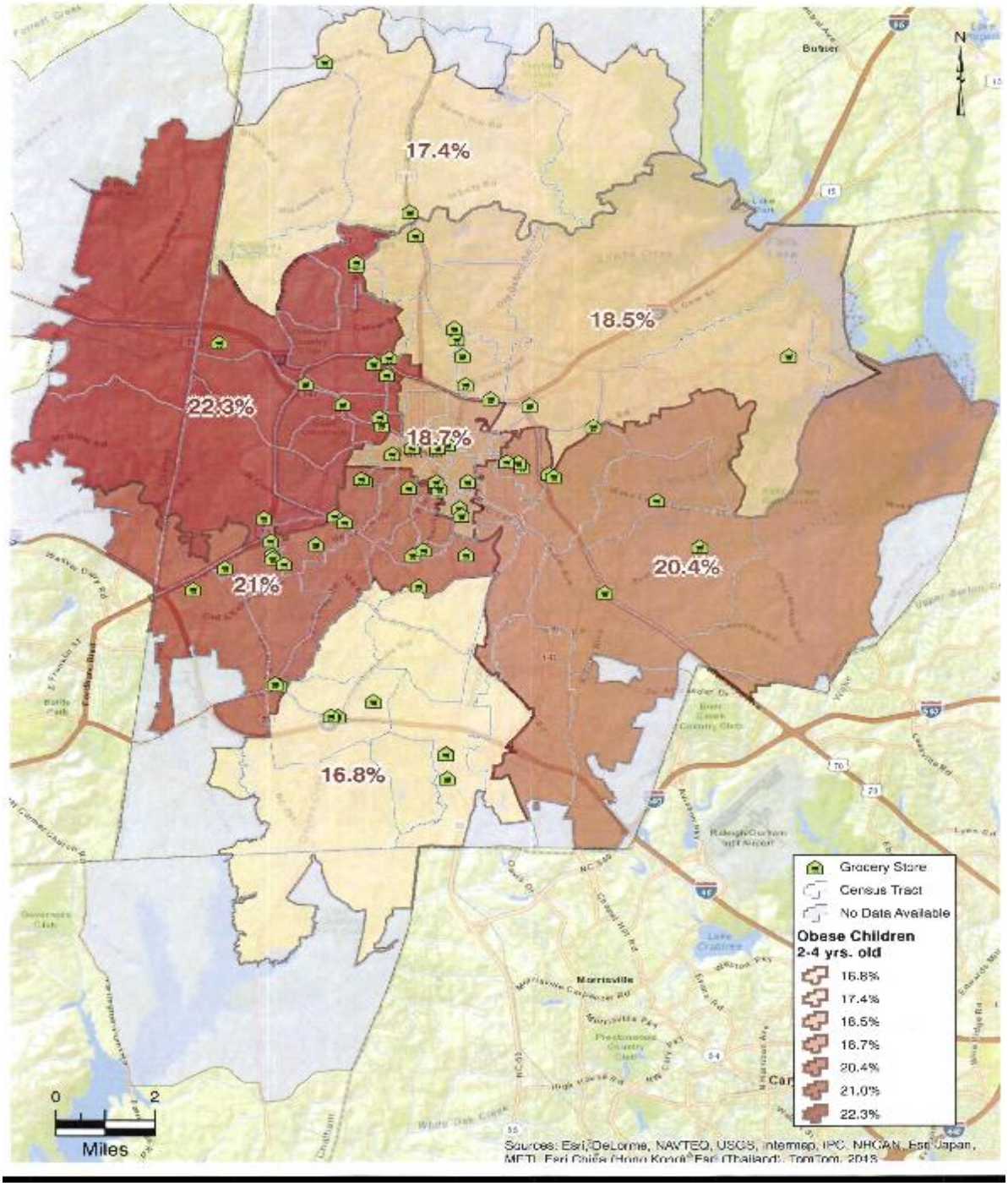
Appendix A32

Fast Food Establishments



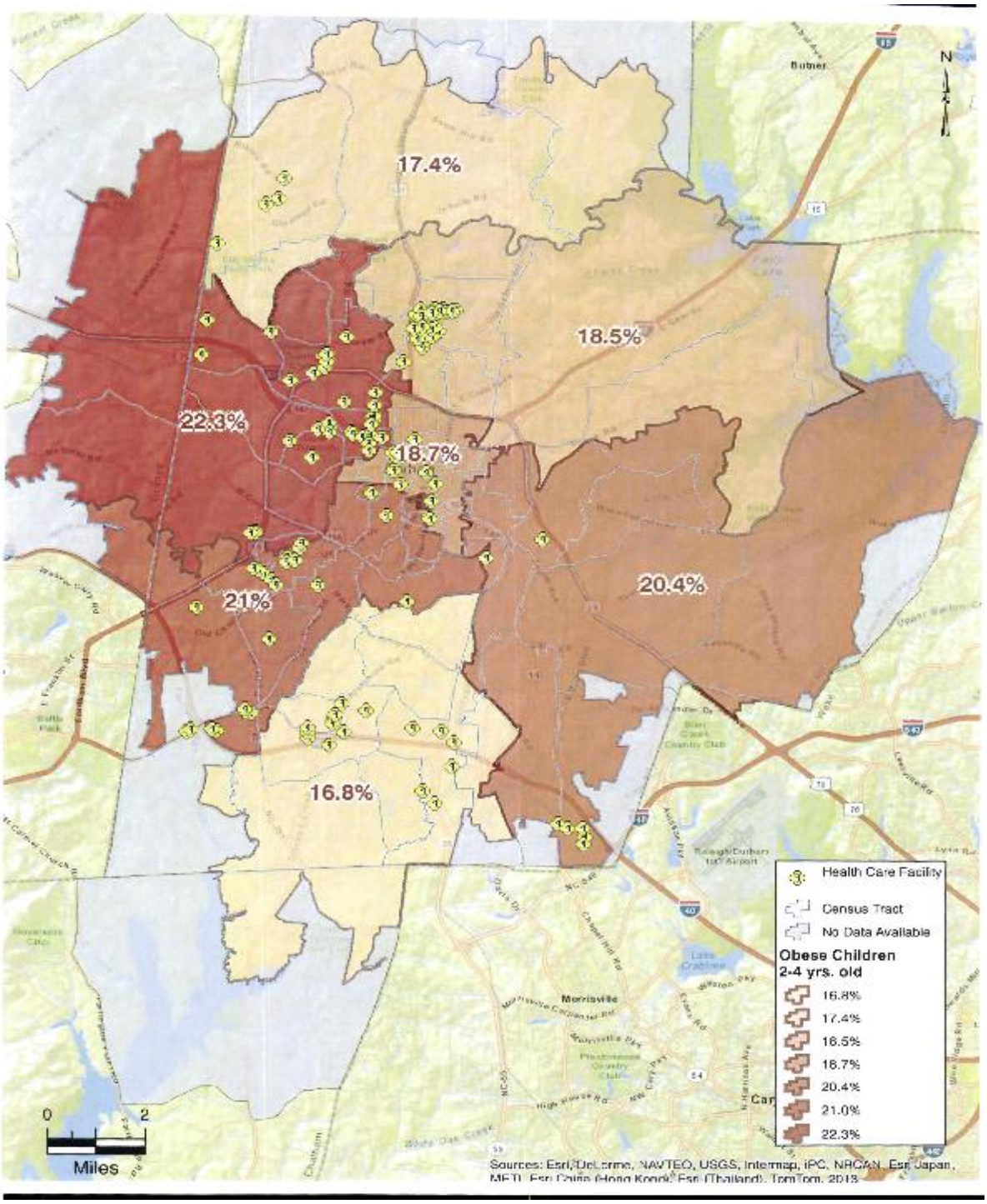
Appendix A33

Grocery Stores



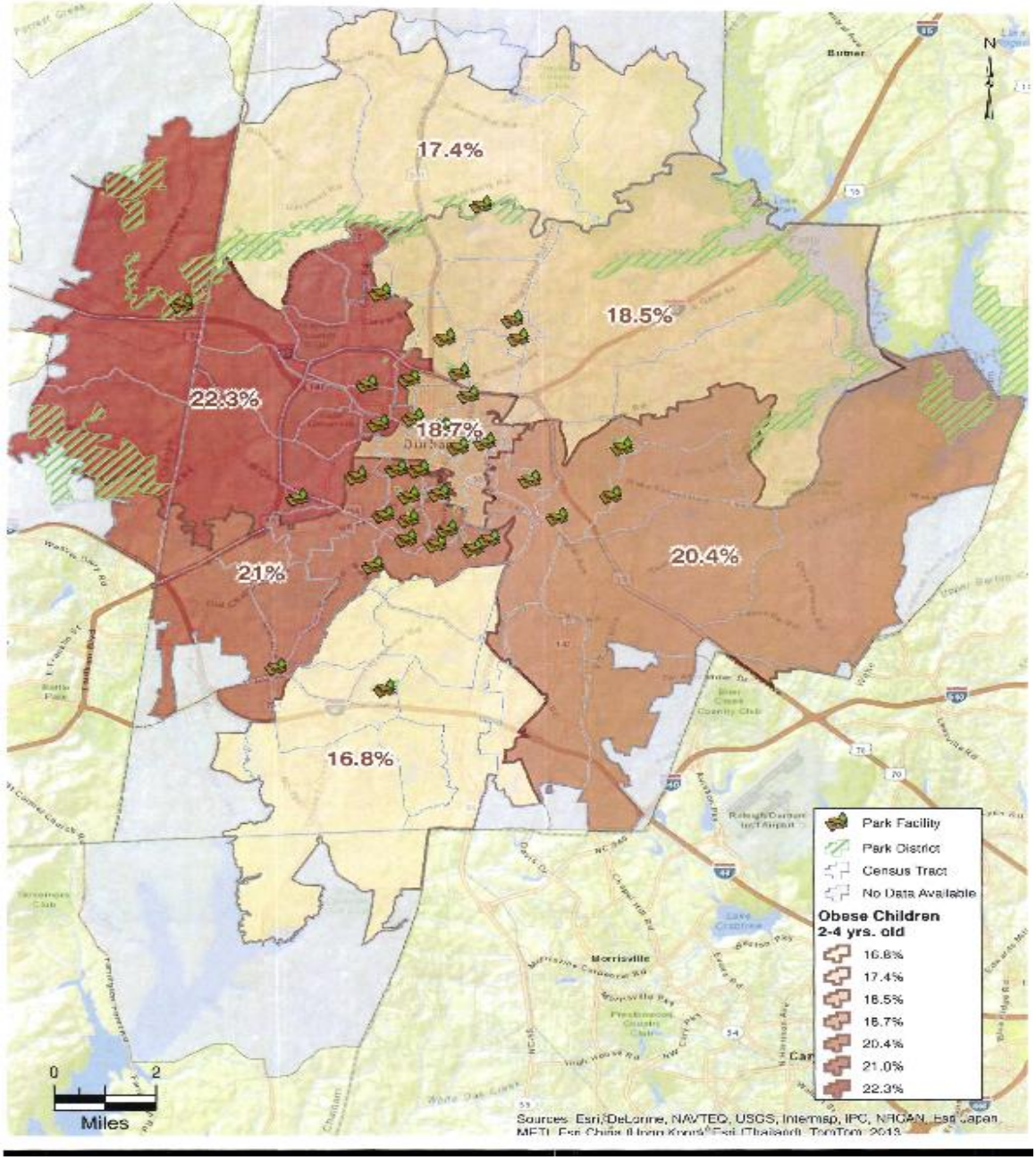
Appendix A34

Health Care Facilities



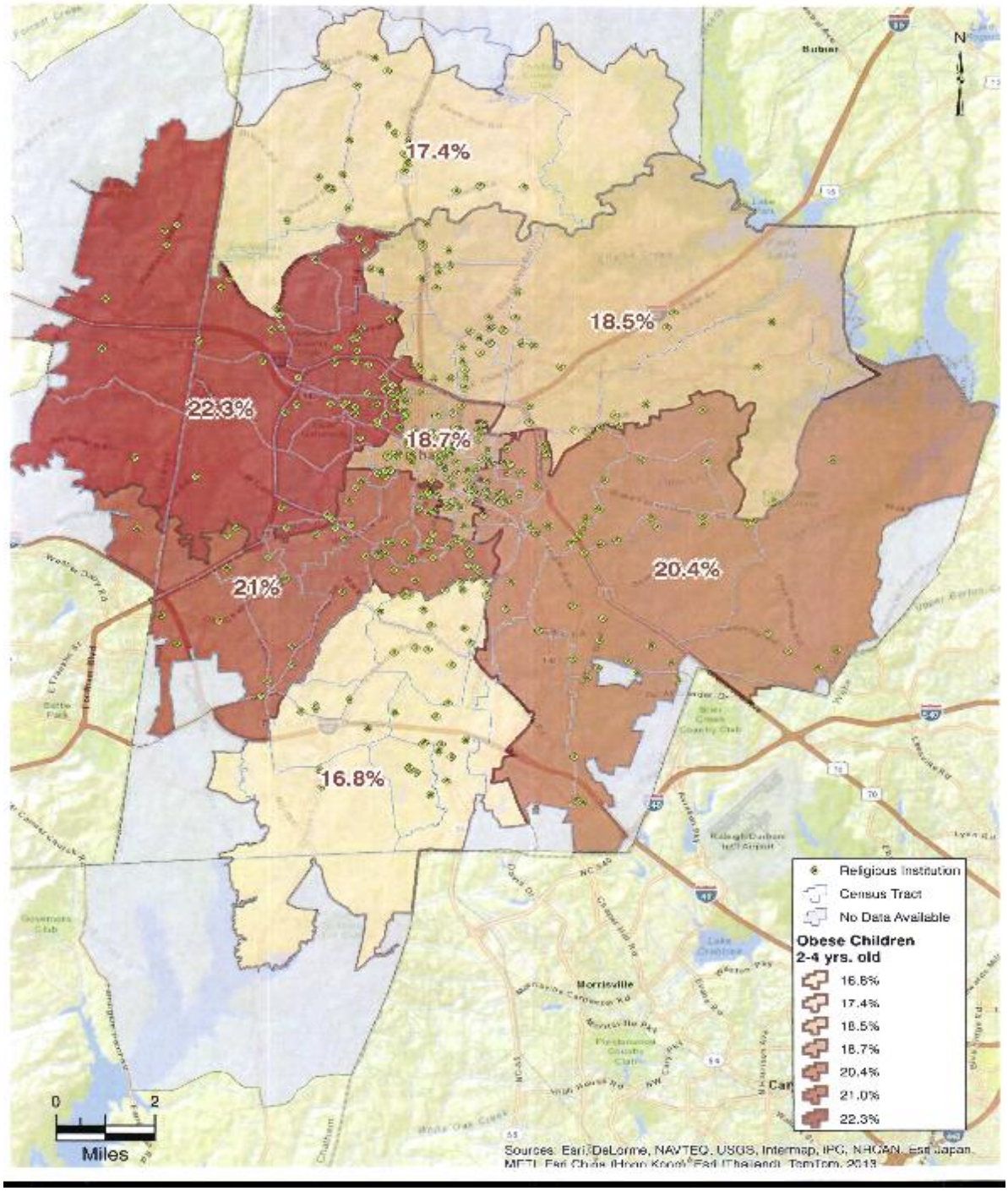
Appendix A35

Park Facilities



Appendix A36

Religious Institutions



Appendix A37

Schools K – 12

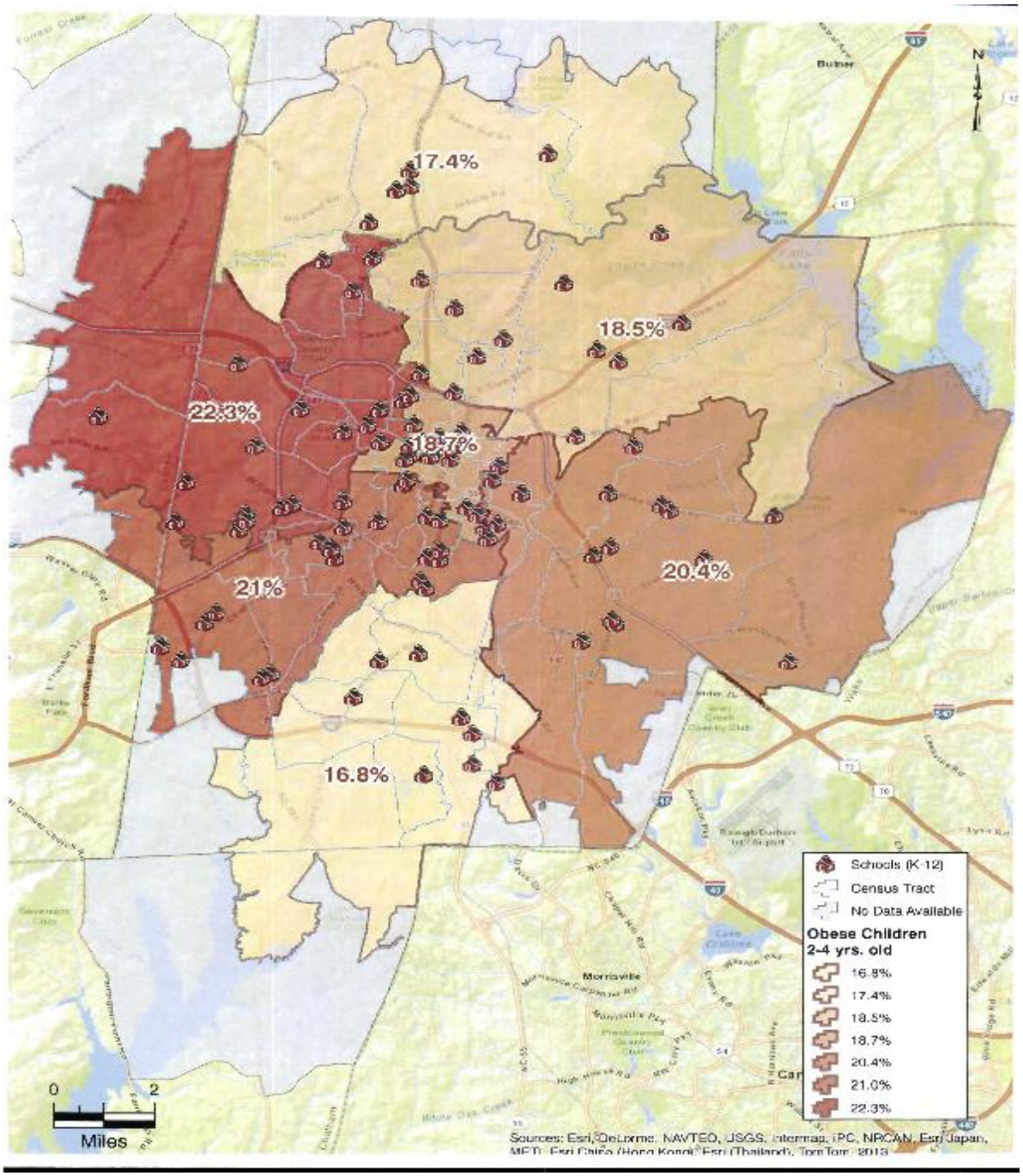


Table B1

Independent Samples Tests

Independent Samples Test										
		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
EdLess9th	Equal variances assumed	1.035	.313	-2.747	62	.008	-3.4842	1.2686	-6.0200	-.9484
	Equal variances not assumed			-2.762	60.950	.008	-3.4842	1.2613	-6.0064	-.9620
Ed912ND	Equal variances assumed	.896	.348	-2.515	62	.015	-4.4177	1.7567	-7.9293	-.9062
	Equal variances not assumed			-2.534	61.237	.014	-4.4177	1.7434	-7.9037	-.9318
EdHighSh	Equal variances assumed	2.220	.141	-.878	62	.383	-2.1194	2.4138	-6.9445	2.7057
	Equal variances not assumed			-.852	49.585	.398	-2.1194	2.4884	-7.1186	2.8798
EdSomCl	Equal variances assumed	.356	.553	-1.773	62	.081	-2.3916	1.3486	-5.0874	.3042
	Equal variances not assumed			-1.787	61.230	.079	-2.3916	1.3385	-5.0679	.2846
EdAssoc	Equal variances assumed	3.090	.084	.652	62	.517	.6064	.9305	-1.2536	2.4664
	Equal variances not assumed			.629	47.896	.532	.6064	.9634	-1.3308	2.5436
EdBachlrs	Equal variances assumed	.645	.425	2.182	62	.033	6.0708	2.7822	.5093	11.6324
	Equal variances not assumed			2.147	54.979	.036	6.0708	2.8275	.4044	11.7372
EdGradgr	Equal variances assumed	.277	.600	2.037	62	.046	5.7736	2.8346	.1074	11.4398
	Equal variances not assumed			2.044	60.591	.045	5.7736	2.8241	.1258	11.4214
EdPctHr	Equal variances assumed	1.232	.271	2.918	62	.005	7.8928	2.7044	2.4868	13.2989
	Equal variances not assumed			2.964	61.951	.004	7.8928	2.6631	2.5692	13.2164
EdPctBHi	Equal variances assumed	.007	.935	2.272	62	.027	11.8266	5.2055	1.4209	22.2323
	Equal variances not assumed			2.276	60.229	.026	11.8266	5.1956	1.4346	22.2186
PctVac	Equal variances assumed	1.380	.245	-2.102	62	.040	-4.791387073	2.279216138	-9.347475214	-.235298932
	Equal variances not assumed			-2.133	61.922	.037	-4.791387073	2.245940495	-9.281069990	-.301704156
PctOcc	Equal variances assumed	1.380	.245	2.102	62	.040	4.791387073	2.279216138	.235298932	9.347475214
	Equal variances not assumed			2.133	61.922	.037	4.791387073	2.245940495	.301704156	9.281069990
PctOwnc	Equal variances assumed	3.739	.058	.781	62	.438	5.5008	7.0422	-8.5764	19.5779
	Equal variances not assumed			.764	52.661	.448	5.5008	7.2022	-8.9472	19.9488
PctRentc	Equal variances assumed	3.739	.058	-.781	62	.438	-5.5008	7.0422	-19.5779	8.5764
	Equal variances not assumed			-.764	52.661	.448	-5.5008	7.2022	-19.9488	8.9472

VALUEN	Equal variances assumed	.392	.533	1.918	62	.060	37716.847	19661.768	-1586.466	77020.161
	Equal variances not assumed			1.873	52.106	.067	37716.847	20138.086	-2691.230	78124.924
PctYr39ar	Equal variances assumed	8.190	.006	-2.210	62	.031	-7.1370	3.2290	-13.5916	-.6825
	Equal variances not assumed			-2.311	56.989	.024	-7.1370	3.0880	-13.3208	-.9533
PctYr9099	Equal variances assumed	.002	.962	1.662	62	.102	5.3340	3.2090	-1.0808	11.7488
	Equal variances not assumed			1.665	60.213	.101	5.3340	3.2032	-1.0729	11.7408
CTRelig	Equal variances assumed	1.833	.181	-1.898	62	.062	-1.972	1.039	-4.050	.105
	Equal variances not assumed			-1.930	61.983	.058	-1.972	1.022	-4.016	.071
CTFFood	Equal variances assumed	1.090	.301	1.334	62	.187	.412	.309	-.205	1.029
	Equal variances not assumed			1.293	49.424	.202	.412	.318	-.228	1.052
CTConvSt	Equal variances assumed	1.030	.314	-1.091	62	.280	-.423	.388	-1.197	.352
	Equal variances not assumed			-1.117	61.766	.268	-.423	.378	-1.179	.334
CTDayCe	Equal variances assumed	.476	.493	-.752	62	.455	-.478	.636	-1.748	.793
	Equal variances not assumed			-.752	59.866	.455	-.478	.635	-1.749	.793
CTGroc	Equal variances assumed	.283	.597	.457	62	.649	.138	.302	-.465	.741
	Equal variances not assumed			.456	59.261	.650	.138	.302	-.467	.743
CTHealtC	Equal variances assumed	.652	.422	.112	62	.911	.311	2.772	-5.231	5.853
	Equal variances not assumed			.117	58.689	.907	.311	2.665	-5.023	5.645
CTHosp	Equal variances assumed	.009	.923	.006	62	.995	.001	.166	-.331	.333
	Equal variances not assumed			.006	60.940	.995	.001	.161	-.321	.323
CTSchoos	Equal variances assumed	.004	.951	-.190	62	.850	-.100	.525	-1.148	.949
	Equal variances not assumed			-.191	60.847	.849	-.100	.522	-1.143	.944
CTRecAa	Equal variances assumed	.530	.469	-1.512	62	.136	-.306	.203	-.711	.099
	Equal variances not assumed			-1.508	59.115	.137	-.306	.203	-.713	.100
CTCrime	Equal variances assumed	.459	.501	-2.011	62	.049	-74.844	37.212	-149.230	-.458
	Equal variances not assumed			-2.006	59.221	.049	-74.844	37.302	-149.480	-.208

Table B2

Summary of Variable Significance

Summary of Variable Significance			
Variable	% Significant	% Negative	% Positive
PCTBELOWPO	0.77	98.46	1.54
CTSCHOOLS1	0.77	63.08	36.92
EDHIGHSCH	0.00	1.54	98.46
PCTRENTOCC	0.00	83.08	16.92
VALUEMEDN	0.00	68.46	31.54
PCTYR39EAR	0.00	3.85	96.15
CTRELIG1	0.00	90.00	10.00
CTFFOOD1	0.00	80.00	20.00
CTHEALTHC1	0.00	43.08	56.92
CTCRIME1	0.00	66.15	33.85

CURRICULUM VITAE

EUREKA C. DAYE
HEALTHCARE EXECUTIVE
CAREER PROGRESSION

- Regional Healthcare Executive & Director of Women's Health California Correctional Health Care Services January 2014 – Present

- Chief Executive Officer, California Correctional Healthcare Services - December 2011– January 2014

- Healthcare Consultant & Practice Group (CornuCopia), Owner / CEO 2005 - current

- Chief Executive Officer / Chief Operating Officer& Administrator, Psychiatric Solutions, Inc. - April 2007– August 2010

- Statewide Vice President, Operations & Clinical Services, Behavioral Link, Inc. - March 2003 – March 2007

- Duke University Fuqua School of Business, Director Health Sector Management - August 2001 – February 2003

- North Carolina State University, Assistant Vice Provost for Institutional Equity & Diversity - May 2000 – August 2001

- University Nebraska Lincoln, Director Organizational Change Management/ EAP Counselor - January 1996 – October 1999

- Vendell Psychiatric Hospitals, Executive Director Clinical Operations - March 1993 – December 1995

- Oak Creek Hospital, Executive Director Pediatric and Ancillary Services - May 1989 – December 1992

EDUCATION

PhD Public Health, Walden University, Minneapolis, MI

MPH – 2006 - Public Health, Walden University, Minneapolis, MI

MA – 1985 - Counseling Psychology, University San Francisco, San Francisco, CA

BA – 1981 - Psychology, Pepperdine University, Malibu, CA

CREDENTIALS

- Licensed Professional Counselor (LPC), State of North Carolina # 4566, NPI 1346420262 / NC Medicaid Provider – 6102625
- Licensed Marriage Family Therapist (LMFT), State of North Carolina # 932, NPI 1346420262 / NC Medicaid Provider - 6102625
- Certified Correctional Health Professional (CCHP) #77979
- Licensed Mental Health Practitioner, State of Nebraska # 1128 (Inactive)
- Licensed Professional Counselor, State of Nebraska # 822 (Inactive)
- Licensed Marriage Family Therapist, State of Nebraska # 54 (Inactive)

  *California Licensure in Progress

PROFESSIONAL AFFILIATIONS

- American Public Health Association
- Academic Consortium on Criminal Justice Health
- American Corrections Association (ACA)
- The American Society for Quality (ASQ)
- Mediation and Dispute Resolution
- Critical Incident and Stress Management
- LPC Association / American Association MFT
- Continental Who's Who Registry of National Business Leaders
- National Association of Professional Women
- Delta Sigma Theta Sorority