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Exploration of the Role of Neighborhood Residential Segregation by Race and Ethnicity in Obesity Risk Among School-Aged

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EXPLORATION OF THE ROLE OF NEIGHBORHOOD RESIDENTIAL SEGREGATION BY RACE AND ETHNICITY IN OBESITY RISK AMONG SCHOOL-AGED YOUTH

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DEDICATION

This dissertation is dedicated to my grandparents, Cecil, Sarah, Earl, and Ruby who helped raise me and shape me into the person I am today. I was and am incredibly lucky to have known all four of my grandparents both as a child and an adult. While Earl and Sarah are no longer living, their impact will continue to be felt by our family for generations to come. My Dad's parents Earl and Ruby, also known as Mama and Papa, showered us with love and affection. Papa, even if he pretended to be a grumpy old man, showed up at almost every soccer game I played in high school yelling at the bad calls the "umpire" kept making. Growing up Mama always made the best potato soup and would deliver it when I didn't feel good. She even tried to teach me how to make her famous biscuits, which unfortunately are still too lumpy to this day when I attempt to make them. My Mom's parents, Cecil and Sarah, also known as Grandmother and Grandaddy, showed us their affection through teaching us things that were important to them. From Grandaddy, I have learned how to shuck corn, shell peas, and how satisfying a long day of yardwork can be. From Grandmother, I learned how cross stitch and make strawberry jam. Her drive and love for learning and education for herself, her children, and her grandchildren were instrumental in where I am today.

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ABSTRACT

Youth obesity prevalence remains high, despite decades of intervention. Grounded in the social ecological model, neighborhoods and schools are important settings in addressing the complex systems that influence obesity. Contributing to disparities, by race/ethnicity, Black and Hispanic youth are more likely to live in segregated neighborhoods and attend segregated schools, which are also more likely to have high rates of poverty and are less likely to have high quality nutrition and built environments. Leveraging data from local school district (district and state department of education) and national datasets (ACS, Childhood Opportunity Index 2.0), this study examined the associations between school and neighborhood environments with obesity risk by race/ethnicity among a large sample of school-aged youth from. Exploring two specific aims, this study 1) used a series of cross-classified multilevel models to explore the associations between school and residential segregation and youth obesity, and 2) used parallel mediation analysis to determine if social/economic, health/built, and education environments mediated the association between residential segregation and youth obesity. Results of the CCMMs, indicated that school and neighborhood environments had a small, but unique influence on the variability in youth BMI z-score. After adjusting for relevant individual, school and neighborhood covariates, school segregation was negatively associated with BMI z-score among Black and Hispanic youth, compared to White youth, while residential segregation was positively associated with youth obesity among Black and Hispanic youth. The addition of an interaction term between Hispanic school segregation and youth race/ethnicity (Black and Hispanic, compared to White) indicated individual race/ethnicity may moderate the association between segregation and youth BMI z-score after controlling for other covariates in the model. Specifically, increased Hispanic school segregation may act as a protective factor for youth BMI z-score among Black and Hispanic youth. Results of the mediation analyses indicated that social/economic, health/built, and education environments may mediate the association between residential segregation and youth obesity. The significance and strength of associations, however, varied by type of residential segregation (Black or Hispanic) and youth race/ethnicity (Black or Hispanic). These findings highlight the important role of federal, state, and local governments and systems in creating equitable investment in infrastructure and resources within schools and neighborhoods and how structural racism can influence investment and access by race/ethnicity and socioeconomic status.

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LIST OF ABBREVIATIONS

ACS.......... American Community Survey

BMI.......... Body Mass Index

CCMM.......... Cross-classified Multi-level Models

CDC.......... Centers for Disease Control and Prevention

COI.......... Childhood Opportunity Index

GIS.......... Geographical Information Systems

ID.......... Index of Dissimilarity

LQ.......... Location Quotient

LWG.......... LiveWell Greenville

MSA.......... Metropolitan Statistical Area

ML.......... Maximum Likelihood

MLM.......... Multi-level Models

NHANES.......... National Health and Nutrition Survey

NHGIS.......... National Historic Geographic Information Systems

PSE.......... Policy, Systems, and Environment Change

SC.......... South Carolina

SES.......... Socioeconomic Status

SPI.......... School Poverty Index

US.......... United States

CHAPTER ONE

INTRODUCTION

Obesity Prevalence and Risk Factors

Youth obesity remains a significant public health problem in the United Sates (US), with significant disparities by income, education, and race/ethnicity that contribute to subsequent inequities in chronic disease and morbidity for certain groups.[1-4] Obesity has significant short- and long-term consequences on youth health outcomes, and youth who have obesity are more likely to become obese adults.[3] In the short-term, youth obesity contributes to increased risk of musculoskeletal problems, hypertension, increased glucose intolerance, and behavioral health issues.[3,5] In the long-term, youth obesity has been associated with increased chronic disease, including diabetes, heart disease, and some cancers over the life course.[3,6]

At the individual and interpersonal level, food and physical activity behaviors directly influence youth obesity risk. Specifically, when there is an energy imbalance, where the number of calories consumed in food exceed the number of calories expended, youth obesity risk increases.[7] To achieve a healthy weight status, national guidelines recommend a number of daily healthy eating and active living behaviors, including consuming five servings of fresh fruits and vegetables, limiting sugar sweetened beverages and foods, engaging in 60 minutes of moderate to vigorous activity or play, and limiting screen time to two hours or less. Despite this guidance, relatively few youths, nationally, achieve these recommendations.[8,9]

Social Ecological Model of Obesity: Schools and Neighborhoods

While individual risk factors such as genetics and food and physical activity behaviors are important, they do not fully explain youth obesity risk.[10] Significant amounts of research point toward the multi-factorial and multi-level influences on youth obesity.[11,12] Once such model that conceptualizes how individuals and systems interact to influence youth obesity risk is the social ecological model. The social ecological model emphasizes that individuals are reciprocally influenced by multiple settings and levels, including the individual, interpersonal, organizational, community, and policy levels. These multiple levels and settings converge to influence healthy eating and active living behaviors, social norms, and environments that contribute to youth obesity.[12] Given the complex systems that shape and sustain youth obesity, intervention efforts must extend beyond individual determinants to those that address policy, systems, and environments (PSE) to have both sustainable and population-level impact.[13,14]

Schools are important settings for youth obesity prevention, given the amount of waking hours spent and calories consumed by youth in school-based environments.[15] Previous school-based youth obesity intervention efforts have shown promise in increasing healthy food and physical activity behaviors, as well as positively changing school level policy, practices, and environments.[16-18] School-based efforts alone, however, are insufficient to address population-level youth obesity.[15,19,20]

Neighborhoods have emerged as important settings where health behaviors, outcomes, and inequities are shaped. They are represented not only by physical features and geographical boundaries, but also impart social and cultural meaning for the residents who reside within them.[21] Neighborhood research has connected both built and social environment characteristics to a broad range of health outcomes, including obesity and chronic disease.[22,23] Neighborhoods have the potential to support behaviors in the form of opportunities or exposures that reduce obesity risk, such as healthy eating and active living opportuntities. These opportunities and exposures comprise the "obesogenic environment," which include social and built environment features.[24,25]

The built environment is the human-made or modified features of a person's external environment that have the potential to affect energy balance through access and opportunities that can shape individual, family, and neighborhood-level food and physical activity behaviors. Previous research has shown that several neighborhood physical activity features are associated with reduced youth obesity risk, including walkability, access to and quality of parks and green space, and neighborhood safety.[26] The nutrition built environment also has implications for youth obesity risk through features such as density of fast food restaurants and access to affordable and high quality produce.[24,27-29] Similarly, neighborhood socioeconomic environment characteristics including poverty, education, social cohesion, crime, and disorder have been linked to obesity outcomes.[25,30,31]

Despite decades of research illuminating the important role of neighborhoods in health and obesity risk, there remain significant limitations in neighborhood obesity research, including study design and other methodological concerns.[23,32] While there is significant research on which neighborhood environment features influence youth obesity, less research has explored the mechanisms by which disparities in neighborhood environments emerge and are sustained.[33] One such potential mechanism is residential segregation.[34]

Residential Segregation, Access to Opportunity, and Obesity Risk

Residential segregation is defined as the spatial separation of two or more social groups within a specified geographic area, such as a municipality, a county, or a metropolitan area.[35] Residential segregation can take many forms, such as segregation by religion, nativity, or income, but one of the most prevalent and persistent forms of segregation across centuries and cultures is segregation by race or ethnicity.[21,36] Rates of residential segregation in the US began to rise in the first half of the 20th century, increased through the 1960s, and have remained relatively stable since.[37] While residential segregation does not emerge nor is it sustained through one mechanism, the residential segregation increases of the 20th century were in large part due to a series of housing policies and practices that were sanctioned and supported by the federal government and led to limited housing quality and locations for racial and ethnic minority families.[34,38,39]

While some research has found that rates of residential segregation have decreased somewhat since the 1980s, Black Americans still live in areas of relatively high residential segregation.[38,40,41] In fact, one recent study found that residential segregation increased in the last two decades, with 81% of American cities and metropolitan areas more segregated today when compared to the 1990s.[39] Further complicating rates of residential segregation by race or ethnicity is the intersectionality of economic and racial residential segregation. For example, a 2021 study found that poverty rates among highly segregated Black communities in the US were much higher (21%) when compared to highly segregated White communities (7%).[41]

Racial and economic residential segregation converge to impact access to opportunities, including quality of housing, education, and employment that have implications for health and wellbeing across the life course.[38,42] Neighborhoods that are residentially segregated may also historically and contemporarily lack investment and access to opportunities and infrastructure that are health-promoting, such as access to parks, safe sidewalks, and lighting.[34,42,43] Additionally, youth who live in residentially segregated neighborhoods are more likely to attend residentially segregated schools, which previous research demonstrates may be of lower quality or have less financial investment, compared to majority White schools.[41]

Health inequities by race and ethnicity in the US remain high despite being a major part of the national health agenda in recent decades.[40,44] While the root causes of health inequities are complex, structural racism is one salient mechanism by which health inequities emerge and are sustained.[45,46] Structural racism is the macrolevel forces, such as policy, law, and culture that produce differential opportunities and exposures for some groups compared to others and is a primary mechanism by which residential segregation and corresponding inequities in neighborhood opportunities for health arise.[21,47] Previous research has found associations between living in highly segregated neighborhoods and a number of health outcomes, including heart disease, asthma, cancer, and birth outcomes.[48-52]

While the evidence is still emerging, residential segregation has been linked to increased obesity risk at the neighborhood level across the US.[40,51-54] Research that indicates living in a highly segregated neighborhood increases obesity risk is strongest for Black women.[40,51] While not as robust, there is some evidence that living in segregation can increase risk of obesity among Black men and Hispanic men and women.[40,55] Conversely, other research suggests living in a residentially segregated neighborhood may be a protective factor for Hispanic residents, shielding them from the effects of acculturation and discrimination.[40] Evidence on the relationship between residential segregation and youth obesity is even more limited, when compared to adult obesity.[56,57]

Only one previous study was found that examined the association between residential segregation and youth obesity as the focal relationship. This study reported that after controlling for individual and family-level covariates, residential segregation explained between 5% and 20% of the difference in obesity and chronic disease risk for Black and Hispanic children.[56] The same study also found that children living in more residentially segregated communities had greater odds of having obesity when compared to children who lived in non-residentially segregated communities, regardless of individual race or ethnicity.

One potential explanation for the association between residential segregation and youth obesity is access to social and built environment opportunities that either support or limit healthy eating and active living.[25] In one study about childhood opportunity at the neighborhood level, McArdle et al. found that 40% of Black youth and 32% of Hispanic youth lived in the bottom quintile of neighborhood opportunity for education, socioeconomic, and built environment opportunities, compared to only 9% of White youth, even after controlling for family poverty levels.[41] Another study by Goodman et al. found that the positive association between residential segregation and youth obesity was partially mediated by dietary behaviors and fully mediated by the food environment at the neighborhood level.[58]

Study Purpose and Significance

To advance the field of residential segregation and youth obesity research, the proposed research has two primary goals and one secondary goal that are part of a larger and ongoing evaluation of youth obesity prevalence, disparities, and contributing community characteristics in Greenville County, South Carolina (SC). This larger evaluation is a partnership with a local healthy eating and active living coalition that is working to make PSE changes to support equitable access to healthy eating and active living opportunities. The first primary goal is to examine the association between residential segregation and youth obesity, while controlling for relevant individual, school, and neighborhood-level characteristics. The second is to examine if, and to what extent, neighborhood-level social and economic environments, health and built environments, and education environments mediate the association between residential segregation and youth obesity. A secondary goal of this study is applied in nature and is to aid the local coalition in identifying contextual PSE determinants of youth obesity and corresponding levers to reduce youth obesity prevalence and its associated disparities, by "making the healthy choice the easy choice."

While research on the relationship between residential segregation and obesity is growing, there remain significant gaps in the literature, especially as it relates to childhood obesity.[56,57,59] Therefore, this study also endeavors to address several gaps in the literature about the relationship between residential segregation and obesity including: 1) variability in results by race/ethnicity and gender, 2) lack of consistent and valid measures of residential segregation and obesity, 3) lack of evidence on the relationship between residential segregation and youth obesity, 4) lack of statistical techniques that control for the effect of clustering or competing environments such as schools, and 5) lack of research on the mechanisms and pathways by which neighborhood residential segregation impacts youth obesity.

Study Overview: Methods, Setting, and Measures

This study draws from two existing frameworks, one by Popescu et al. that describes how structural racism drives residential segregation and resource allocation that creates disparities in health outcomes, and a second by Suglia et al. that details the relationship between neighborhood physical and social environments and obesity risk.[25,42] Both of these frameworks were infused with concepts from the social ecological model in an attempt to integrate the influences of 1) exposures to structural racism and discrimination, 2) socioeconomic environments influences such as neighborhood poverty and education, 3) health and built environments factors such as walkability and access to green space, 4) education environments such as access to high quality early child care and neighborhood level educational attainment and 5) the intersectionality of multiple exposures and environments on the relationship between residential segregation and youth obesity.[12,25,42]

The study setting was Greenville County, SC, which is in the northeastern corner and is the most populous county in the state.[60] There is only one school district in the county, which is the 44th largest in the nation and covers more than 800 square miles. The district contains 51 elementary schools, 20 middle schools, 14 high schools, and 16 special schools or centers, which serve more than 76,000 students.[61,62] Obesity rates in the county are higher than the national average, where 30% of adults have obesity and 18.8% of third through fifth grade youth have obesity.[63]

The individual-level data that were used to explore these aims are part of routine data collection conducted by the school district. Youth demographics were obtained from the district *PowerSchools* database and youth height, weight, and physical fitness data were obtained from the district *FitnessGram* database, as part of a data sharing agreement with Furman University.[64] Objectively collected youth height and weight data were used to create Body Mass Index (BMI) z-scores, the main outcome of interest, using the Centers for Disease Control and Prevention's (CDC) youth BMI percentiles.[65] Remaining youth-level data (biological sex, grade, race/ethnicity) were used as study covariates.

Additional data sources included school and neighborhood characteristics that were used as study covariates and in some cases potential mediating variables. District and school-level poverty and minority composition were obtained from the SC State Department of Education website.[61,62] School poverty was estimated using the School Poverty Index (SPI), which is calculated by the state department for the district and each school, annually. School minority composition was used to calculate school segregation among Black and Hispanic youth using the Location Quotient (LQ), which is the proportion of one group in the smaller area unit (schools) divided by the proportion of that same group in the larger area unit (district).[21]

Neighborhood data came from two sources, census tract data available from the US Census Bureau American Community Survey (ACS) and the Childhood Opportunity Index (COI) 2.0, developed by Brandeis University.[66] The primary independent variable, neighborhood residential segregation, was also measured using the LQ from ACS racial composition data at the census tract and county level. Residential segregation LQs were calculated separately for Black and Hispanic segregation as the proportion of one group in the smaller area unit (census tract) divided by the proportion of that same group in the larger area unit (county).[21] Additional neighborhood-level measures included the three domain z-scores from the COI 2.0: the social/economic environment, the health/built environment, and the education environment. Each domain z-score is calculated at the census tract level, relative to all other census tracts in the US, using component indicators that are empirically and conceptually linked to the domain.[66] The remaining neighborhood measure, educational attainment, was calculated using ACS data and defined as the percentage of neighborhood residents who have a high school diploma or less. The educational attainment variable was grand mean centered and treated as a continuous measure.

1.1 SPECIFIC AIMS AND HYPOTHESES

This dissertation is part of a larger body of research aimed at better understanding how school and neighborhood healthy eating and active living environments, and disparities within these environments by race/ethnicity and socioeconomic status, converge to impact youth obesity rates. This dissertation will examine the association between neighborhood residential segregation and youth obesity in the context of school (poverty and segregation) and neighborhood environments (social/economic, health/built, education) through the following study aims and hypotheses:

Aim 1a: Examine the relative influences of school and neighborhood environments on youth obesity risk among elementary, middle, and high school youth.

- **Hypothesis 1a**: School and neighborhood environments will have a unique contribution on youth obesity risk.

Aim 1b: Examine the association between neighborhood residential segregation and youth obesity, after controlling for individual (gender, race/ethnicity, grade), school (segregation, poverty), and neighborhood (education) characteristics.

- **Hypothesis 1b**: There will be a statistically significant and positive association between neighborhood residential segregation and youth obesity, after controlling for individual, school, and neighborhood characteristics.

Aim 2a: Determine if neighborhood social and economic environments, health and built environments, and education environments mediate the association between neighborhood residential segregation and youth obesity.

- **Hypothesis 2a**: Neighborhood social and economic environments, health and built environments, and educational environments will mediate the relationship between residential segregation and youth obesity, after controlling for other mediators and covariates in the model.
- **Hypothesis 2b**: The significance of mediating effects across social and economic environments, health and built environments, and education environments will vary by type of segregation (Black versus Hispanic) and individual youth race/ethnicity (Black versus White).

CHAPTER TWO

BACKGROUND AND SIGNIFICANCE

2.1 BACKGROUND

Current Prevalence, Disparities, and Complications of Youth Obesity

Obesity is arguably one of the most important- and at the same time- most complex public health problems in the US and many other developing nations, with obesity prevalence more than tripling in the US over the last three decades.[1,2] In particular, obesity prevalence among school-aged youth has increased more rapidly when compared to other segments of the population.[1,67] While increasing prevalence of obesity among youth 2 to 19 years of age began showing signs of plateauing in 2005-2006, overall prevalence remains high.[68] In 2016, an estimated 18.4% of youth aged 6 to 11 years of age and 20.6% of youth 12 to19 years of age had obesity, with significant disparities by race/ethnicity, income, and education.[4,68,69]

Data from the 2015-2016 National Health and Nutrition Survey (NHANES) indicated non-Hispanic Black youth (22.0%) and Hispanic youth (25.8%) 2 to 19 years of age were more likely to have obesity when compared to non-Hispanic White youth (14.1%).[1] There were also significant disparities in severe obesity by race and ethnicity, with much higher percentages of Black (9%) and Hispanic (9.1%) youth meeting the criteria for severe obesity when compared to White youth (3.9%).[70,71] NHANES data from 2011 to 2014 also showed that obesity prevalence decreased with increasing levels of education for the head of household: 21.6% for high school graduate or less, 18.3% for some college, and 9.6% for college graduates.[69] While the relationship between obesity and SES is not linear, youth living in low and middle-income households are much more likely to have obesity when compared to youth in high-income households. Specifically, obesity prevalence was 18.9% among children and adolescents 2 to 19 years of age in the lowest income group, 19.9% among those in the middle-income group, and 10.9% among those in the highest income group.[69]

Geographic setting is another factor that is associated with youth obesity, with higher prevalence in the southeast and in rural settings.[14,72] This is exemplified by the rates of youth obesity in the state of SC, which exceed national averages.[73] Furthermore, disparities by income and race/ethnicity are magnified in SC when compared to the nation.[74] According to a 2019 report from the State of Obesity, SC ranked 3rd in the nation for rates of youth obesity among youth 10 to 17 years of age at 22.1% compared to only 15.5% in the nation.[71] A 2018-2019 statewide study of public school fitness and weight status found that among elementary and high school-aged youth, Hispanic and Latino youth had the highest percentage in the overweight or obese category (47%), followed by Black (41%) and White (32%) youth.[73]

Obesity results in not only short- and long-term complications on physical health for youth, but also affects their psychosocial health and development.[75,76] In the short-term, youth who have obesity experience increased rates of fatty liver disease, dyslipidemia, blood glucose dysregulation, elevated blood pressure, asthma, and musculoskeletal issues.[77,78] Youth who have obesity are more likely to be bullied and socially isolated when compared to their normal weight peers, and alternatively youth who are obese are more likely to participate in bullying.[5,79] They also report higher levels of anxiety, depression, behavioral problems, and sleep dysregulation when compared to their normal weight peers.[5] Moreover, childhood obesity often persists into adulthood.

Youth who have obesity are five times more likely to become obese adults when compared to youth in the normal weight category.[80,81] A 2018 longitudinal study (n=51,505), found that 90% of children who had obesity at the age of three were also overweight or obese as adolescents and among adolescents with obesity, the greatest acceleration of weight gain occurred from ages 2 to 6.[81] Another study found that if a child had an increased risk of obesity, their risk factors for obesity and chronic disease in adulthood were more severe.[71]

Adult obesity is associated with increased risk of chronic disease (diabetes, heart disease, and some cancers), morbidity, and mortality, as well as decreased quality of life and psychosocial function.[14,67,82] Therefore, disparities in youth obesity rates by race and ethnicity, gender, and SES, examined through the particular lens of the life course perspective, also lead to disparities in prevalence and age of onset of obesity-related comorbidities as children of color enter adulthood.[67,83] Given these trends in overall prevalence rates, the demonstrated disparities by demographic characteristics, and the adverse health consequences associated with obesity, there is a need to address obesity at the population level during the critical period of early childhood.[3,12,84]

Role of Food and Physical Activity Behaviors

Most proximally, the cause of childhood obesity is an energy imbalance, where caloric intake exceeds energy expenditure, resulting in weight gain over time as the imbalance persists.[7] Obesity intervention on this level has focused on restoring the energy balance by increasing energy expenditure through increased physical activity and reducing caloric intake through decreasing consumption of calorically dense foods, such as processed sweets and snacks, while also increasing the consumption of healthy foods, such as fruits and vegetables.[7,85] Despite this focus on promoting healthy eating and active living behaviors in youth, relatively few youth currently meet national recommendations for nutrition and physical activity.[8,86,87]

The Physical Activity Guidelines for Americans recommends that youth 6 to 17 years of age participate in at least 60 minutes of physical activity seven days per week; however, the National Physical Activity Alliance found that less than 25% of youth met the guidelines for daily physical activity, with significant disparities by demographic characteristics.[8,83,85] An estimated 35% of high school boys met the guidelines for physical activity, but only 18% of girls reported an hour or more of physical activity each day and the proportion of female youth meeting recommendations for physical activity declined with age.[86] Differences in physical activity by race and ethnicity are not well understood, but when examining sedentary behaviors (i.e., screen time) that correspond with decreased physical activity, there are clear patterns of disparities by race and ethnicity.[86] Black youth (25%), Hispanic youth (32%), and Asian (30%) youth 6 to 19 years of age are less likely to meet the guidelines of two hours or less of screen time per day, compared to White youth (35%).[83,86] Girls 6 to 19 years of age are more likely (38%) than boys (28%) to watch two hours or less of screen time per day.[86]

The Dietary Guidelines for Americans has four overarching guidelines: follow a healthy pattern across the lifespan; focus on variety, nutrient density, and amount; limit calories from added sugars and reduced fats and reduce sodium intake; and shift to healthier food and beverage choices.[9] As part of the 2015-2020 guidelines, it is recommended that all youth consume five or more servings of fresh fruits and vegetables per day, yet the 2013 Youth Risk Behavior Surveillance System survey estimated that only 8.5% of high school age youth met guidelines for fruit consumption and only 2.1% met recommendations for vegetable consumption.[9,88,89] Fruit and vegetable consumption is lower among families living in poverty, with 7% of adults below the poverty line meeting fruit and vegetable recommendations, compared to 11.4% among the highest household incomes.[87]

Sugar-sweetened beverages, and in particular sodas, have been identified as a major contributor to the obesity epidemic.[20,90] The 2015-2020 Dietary Guidelines for Americans includes the recommendation of consuming no more than 10% of daily calories from added sugars and to choose beverages with no added sugars.[9] A 2017 NHANES report indicated that almost two-thirds of youth 2 to 19 years of age consumed at least one sugar-sweetened beverage each day, with disparities by gender, age, and race/ethnicity.[91] Boys were more likely to consume more calories from sugar-sweetened beverages compared to girls, and sugar sweetened beverage consumption increased by age.[91] At the intersection of gender and race/ethnicity, non-Hispanic White boys (176 kcal) consumed a higher average number of calories from sugar-sweetened beverages, followed by non-Hispanic Black (167 kcal), Hispanic (156 kcal), and non-Hispanic Asian boys (73 kcal). [91] Non-Hispanic Black girls (156 kcal) consumed a higher average number of calories from sugar-sweetened beverages, followed by non-Hispanic White (124 kcal), Hispanic (115 kcal), and non-Hispanic Asian girls (58 kcal).[91]

Social Ecological Model of Obesity: Familial, School, and Neighborhood Context

Individual and family-level factors such as genetics and health behaviors do not fully explain youth obesity and chronic disease risk.[12,67,92] While increased caloric intake and insufficient energy expenditure are drivers of increased youth obesity rates, addressing these behaviors alone does not account for the complex systems and upstream factors that influence obesity risk, such as education, income, and access to opportunities.[12,14] Individual and system-level influences, including school and neighborhood settings where children and their families live, learn, work, and play, should be considered simultaneously to better understand youth obesity.[12,13,93] Despite numerous calls from national and global health organizations, including the World Health Organization and the Centers for Disease Control and Prevention (CDC), for interventions that address the multifactorial nature of obesity, to date, few have comprehensively and successfully addressed the multiple risk factors for obesity across individual and system-level determinants.[12-14,94]

Long used in biological and other sciences, public health research now commonly employs the social ecological model to emphasize the interplay of individuals with their environment and that behaviors are influenced and reinforced by factors at the individual, household, organizational, and community levels.[12,95,96] Specifically, youth obesity has been linked to system and community factors, including neighborhood SES, safety and quality of housing, availability of public transit, and access to healthy eating and active living opportunities.[97-99] Addressing these upstream determinants at the population-level requires consideration of government and organizational-level policies and systems that shape school and neighborhood health environments and opportunities, such as land use zoning, housing, and government investment in supplemental nutrition programs.[100,101]

School Environments and Obesity

Schools are important settings for the prevention and reduction of youth obesity, as children spend a significant number of their waking hours in school-based environments and schools provide a significant portion of children's daily intake via the National School Lunch Program or competitive foods.[20,102,103] The majority of youth obesity prevention efforts have been concentrated in school settings and while many of these intervention efforts have shown promise, they have not been significant enough alone in preventing increases in population-level youth obesity rates in recent decades.[104,105] Common PSE driven approaches to improving school-based nutrition include strategies such as limiting food used as rewards for good behavior and academic performance, limiting unhealthy food items used as fundraisers, and increasing availability of healthy snacks and school lunches.[15,85] For example, the Children's Lifestyle and School Performance Study found that after for controlling for school and individual covariates, youth at schools with a CDC school health intervention had lower levels of obesity, healthier diets, and more participation in physical activities when compared to students at schools who offered healthy menu options, but did not have a school health program.[85]

Similarly, school-based physical activity interventions efforts have encouraged policy and practice changes, such as active transit to school, increased recess time, and incorporating physical activity into curricula.[106] These efforts have shown promise in increasing physical activity and other outcomes, such as improved classroom behavior and cognition, but less success in reducing individual youth body mass index BMI and overall obesity prevalence.[18,107,108] For example, a randomized control trial to increase school based physical activity among economically disadvantaged youth in New South Wales found that a multi-component intervention that included education, practice, and environment changes successfully increased moderate to vigorous physical activity and had a positive, but small effect, on adiposity and BMI *z*-score.[109]

While schools provide a captive environment where youth make food choices and engage in physical activity, population-level obesity rates remain high despite decades of successful school-based intervention.[10] Growing evidence suggests that youth experience accelerated weight gain during the summer months that is linked to increased sedentary and screen time, decreased physical activity, and increased consumption of sugar sweetened beverages and foods.[19] During the academic year, school-based settings are not the only context in which food and physical activity behaviors occur. A nationwide study of more than 3,000 youth found that only 32% of empty calories were consumed in school settings, with the rest coming from stores and fast-food restaurants outside of school hours.[20] Therefore, while schools remain important settings for addressing childhood obesity, continued research and intervention efforts should aim to address the full context in which the food and physical activity choices of children and families are shaped, including neighborhood settings.[16]

Neighborhoods, Health, and Obesity

Another important context and rapidly growing area of research in which health behaviors, outcomes, and inequities emerge is neighborhoods.[22,23,25,110] There are numerous definitions of neighborhoods in the field of health research that attempt to explain how geographic boundaries, physical characteristics, social context, and history converge to create neighborhoods and influence health outcomes.[110,111] Hallman defines neighborhood as *"a limited territory within a larger urban area where people inhabit dwellings and interact socially."*[110] Building upon the concept of connection and social interaction, Duncan and Kawachi similarly define neighborhoods as *"geographical places that have social and cultural meaning to residents and nonresidents alike and are subdivisions of large places."*[111] While there remains significant variability in how neighborhoods and the separate, but interrelated, concept of "communities" are defined, there is consensus that neighborhoods contain both social and physical features that exert influence over health-related behaviors and conditions, including obesity.[23,25,112]

Grounded in epidemiological theory, neighborhood research attempts to understand the interplay between individuals, space, and time.[23] Previous studies have found significant associations between a broad range of health outcomes and neighborhood environment features, including depression and mental health, cancer risk, and birth outcomes, while others have found mixed findings for behaviors and health outcomes such as physical activity and obesity.[40,113-116] A 2016 review of multi-level neighborhood and health studies by Arcaya et al. identified 259 papers published about US populations between 1995 and 2014.[22] Indicators of SES, such as unemployment rates, single-headed households, and educational attainment were the most common neighborhood-level measures utilized (28.2%), followed by the built environment (9.3%) and poverty (6.95%). BMI was the most common health outcome of interest (19.7%), followed by mental health (13.5%), pregnancy and birth outcomes (7.7%), cancer prevention and outcomes (7.7%), and self-rated health (7.3%).[22]

As it relates to obesity and chronic disease risk, one important concept of the neighborhood is the "obesogenic environment," which is conceptualized as neighborhood opportunities and exposures, or lack thereof, that contribute to obesity risk.[10,63] Two important neighborhood components of the obesogenic environment are socioeconomic and built environments, which encompass features such as lack of parks and green space, high density of fast food restaurants, low walkability, crime, poverty, and low social cohesion.[23,25,63] The built environment can be defined as all that is external to the individual and encompasses aspects of a person's surroundings, which are human-made or modified, as compared with naturally occurring aspects of the environment.[13,117] The built environment has potential to affect energy balance through access- or lack of access- to physical activity and healthy food consumption resources.[24,117] Built environment interventions have shown promise for population-scale and sustainable impact on healthy eating and active living behaviors and subsequently obesity and chronic disease, but the evidence remains mixed.[23,117] Built environment features supportive of physical activity that have been positively associated with obesity include, but are not limited to, park availability, proximity, and quality; green space; sidewalk and street connectivity to promote walkability and active transit; and perceived and objective neighborhood safety.[13,25,26,118,119] For example, a 2015 observational study of physical activity features associated with physical activity found that compared to home environments, youth were more likely to engage in moderate to vigorous physical activity in outdoor built environments including parks, playgrounds, and while using active transit features, such as streets and sidewalks.[26]

The built environment may also exert influence over obesity risk through the nutrition environment that includes features such as proximity and density of healthy food outlets (grocery stores, farmers markets, community gardens) and unhealthy food outlets (fast food, convenience stores).[29,120] There is more variability in findings from nutrition built environment research when compared to physical activity built environment research, with some researchers citing a lack of consistency in definitions and measurement and others the over or underestimation of relationships because of lack of variables to elucidate causal pathways or serve as adequate controls.[27,28,121] Using longitudinal data from the Framingham Heart Study Offspring Cohort Study, Block et al. explored the relationship between proximity to food establishments and BMI.[29] The study found a significant and negative association between proximity to fast food outlets among women, with each kilometer increase in distance to a fast-food outlet resulting in a 0.11 unit decrease in BMI. In contrast to other studies, the Framingham Study found no significant relationships between BMI and any type of food outlet among men or any other type of food outlets such as grocery stores, full-service restaurants, or convenience stores among women.[29]

In a 2010 systematic review, Feng et al. identified 22 articles exploring the association of food and physical activity built environment factors with obesity using place-based units like census tracts and block groups.[117] Collectively, these 22 papers examined 80 different built environment relations with obesity and found that only 40 were significant in the hypothesized direction, two were significant in the opposite direction, and 38 did not reach significance.[117] In a 2007 systematic review, Papas et al. identified 20 articles on the relationship between built environment features and obesity, with 84% reporting a statistically significant positive association between some aspect of the built environment and obesity.[13] Similar to Feng et al., Papas et al. cited measurement and built environment definition issues, methodological inconsistencies, lack of multiple measures and controls, and lack of longitudinal studies as barriers to definitively explaining the contribution of built environment factors in the relationship with obesity and obesity related behaviors.[13,117] Therefore, despite the rapid proliferation of built environment literature on its role in the prevention and reduction of obesity, significant research is still needed to determine which built factors are consistently related, the strength of their contribution, and their respective pathways that influence obesity risk.

Socioeconomic environment features such as neighborhood poverty and education, social cohesion, neighborhood crime, and neighborhood conditions such as vacant or damaged housing also play an important role in youth obesity risk.[25,30,122] While individual SES has been clearly linked to increased obesity risk, there is a growing body of evidence on the relationship between neighborhood poverty and obesity risk, net of family level SES.[31] For example, a longitudinal study of neighborhood SES and youth obesity risk found that male and female youth who moved into a higher poverty neighborhood over the study period had less favorable weight status outcomes and that the effects were strongest for younger children and adolescents.[31] In another study Chang et al. found that neighborhood disorder (i.e. housing vacancies, disrepair, code violations) was associated with increased obesity risk among Black women.[30]

Intersectionality of Individual, School, and Neighborhood Environments

To date, the majority of neighborhood built environment research and intervention efforts have focused on where and how to mitigate inequitable access to healthy eating and active living opportunities, such as identifying and promoting interventions to increase access to healthy foods in retail outlets, or physical activity opportunities, such as walkability and park access, rather than examining the root causes of why inequitable environments emerge or how individuals interact with and perceive those environments.[23,32] To expand understanding of complex health problems like obesity, in addition to examining more proximal neighborhood features, neighborhood health research must consider these factors in the context of complex social and economic dynamics, such as poverty, crime, and segregation.[32] For example, a 2020 study of neighborhood-level influences on youth obesity found that youth whose parents perceived their neighborhoods to be unsafe were 2.23 times more likely to be obese compared to youth whose parents perceived their neighborhood to be safe, after adjusting for neighborhood food and physical activity opportunities and individual demographics.[123]

Youth who live in residentially segregated neighborhoods are also more likely to attend residentially segregated schools that have higher levels of school poverty, with previous research linking attendance at predominantly minority, low-income schools to lower educational and employment attainment. segregation and poverty on youth educational and employment outcomes.[41,124] These same schools are more likely to have lower school quality ratings and fewer resources, therefore, youth who live in residentially segregated neighborhood that also attend segregated schools are doubly impacted by the effects of segregation on environments and opportunities.[41] Limited research, however, has explored the relationship between school segregation and health outcomes, including youth obesity, and how it intersects with residential segregation at the neighborhood level.[57,59]

Neighborhoods are couched within broader geographical settings that exert policy and system influences over them, such as investment in schools or availability of high paying jobs.[23] Through social stratification and hierarchy, these macrolevel forces result in differential access of certain groups to resources and opportunities, which subsequently create inequalities across a broad range of determinants, including health, education, housing, employment, and transportation.[21,37,38] One of the most enduring representations of how these macrolevel forces impact the relationship between neighborhood and health is the inextricable relationship between race and class in the US, and in particular within the Deep South. This relationship has driven patterns by which minority individuals and families have been historically and contemporarily limited in housing options and neighborhood of residence, giving rise to pervasive residential segregation and concentrated poverty in communities of color.[34,38,43]

Defining and Measuring Residential Segregation

Residential segregation, where elite groups have been isolated through the exclusion of other groups, has been present throughout many societies across the globe and history.[36,38,125] One historical example is the neighborhoods of 15th and 16th century Venice, where Jewish residents were required to reside separate from their fellow residents. These segregated, Jewish communities are where the term “ghetto”, commonplace to modern America, was created.[37] Residential segregation is defined as the spatial separation of two or more social groups within a specified geographic area, such as a municipality, a county, or a metropolitan area.[36] The most common form of residential segregation in the US is racial or ethnic segregation, but it may take other forms such as religious, economic, gender, and nativity.[21,39]

Residential segregation is measured in numerous ways, using a number of constructs within its broader definition.[36,126,127] In efforts to create consensus in the field of residential segregation and health research, Massey and Denton evaluated 20 indices of residential segregation in the 1980s using principal axis factor analysis.[36] Based on these analyses, they proposed five conceptually and empirically distinct dimensions of residential segregation: evenness, exposure, concentration, centralization, and clustering (Table 2.1).[36]

These conceptual and empirical definitions dominated the field for over two decades, until Reardon and O'Sullivan and Johnston et al. separately proposed only two dimensions that were composites of the original five.[21,126,127] Johnston et al. used 1990 and 2000 US Census Bureau data to revisit Massey and Denton's original analyses.[126] Using principal components factor analysis, they identified two dimensions they called separation and location, where separation comprised the original dimensions of unevenness, isolation, and clustering, while location comprised concentration and centralization. Reardon and O'Sullivan similarly identified two dimensions of residential segregation using the same combinations of the original five dimensions as Johnston et al., but they labeled their dimensions as spatial exposure and spatial evenness.[127] For the purposes of this study, the two-dimension framework proposed by Johnston et al. and Reardon and O'Sullivan will be adopted.

Rates of Racial and Ethnic Residential Segregation in the US

As industrialization drove the demand for workers at the turn of the 20th century, more Black Americans than ever began moving to northeast cities for employment.[38,125] At the same time, there was a rapid increase of European immigrants seeking new opportunity in the US. As the number of Black and immigrant residents in northeast cities rose, so did racial tensions and gradually lines began to form by employment, education, and housing.[38] This, combined with federally sanctioned housing policies and lending practices post WWII, led to pervasive residential segregation that continued to rise until the 1960s.[34,38,125] While some research suggests residential segregation in the US declined following the passage of the Fair Housing Act of 1968, the majority of Black Americans still live in neighborhoods that are residentially segregated.[34,37,51]

From 1980 to 2010, there was a decrease in residential segregation, as measured by the Index of Dissimilarity (ID), which is the most common measure of residential segregation.[37] The ID ranges from 0 to 1.0, with 0 indicating complete integration, 1.0 indicating complete segregation, and .60 considered high segregation.[36] The ID decreased significantly (.731 vs. .594) from 1980 to 2010, while Hispanic-White segregation decreased slightly (.518 vs. .496), and Asian-White segregation remained relatively stable (.412 vs .415).[37] These data indicate that while residential segregation decreased the most for Black-White segregation, Black Americans still lived in high levels of residential segregation in 2010 and were more segregated than Hispanic or Asian residents.

Emerging evidence, however, suggests racial residential segregation in recent decades may have been higher than previously accepted in research and that American cities have actually become more segregated in the last two decades.[39] Using an alternative measure of residential segregation, Menendian et al. found that 81% of American cities and metropolitan areas are more segregated today compared to rates of segregation in 1990.[39] Cities with high rates of residential segregation were more likely to span the rustbelt, or the industrial Midwest to the mid-Atlantic, compared to other areas of the US. Only two of the 113 largest US cities met the criteria for residential integration, and perhaps most notable, cities with higher levels of residential segregation were also more likely to be politically polarized.[39] Similarly, a recent analysis of 2015-2019 ACS data found that although residents of color represented the vast majority of population growth in the US (53% among residents of color vs. 1% among White residents), White residents still live in largely White neighborhoods and Black and Hispanic residents still live in disproportionately Black neighborhoods.[33] Another recent study, using geotagged locations of more than 130 million tweets, found that residential segregation occurs not only within the neighborhoods in which individuals reside, but also in their mobility across neighborhoods.[128] In cities with a larger proportion of Black residents and a greater level of historical racial conflict, individuals were less likely to travel to neighborhoods that differed in composition from that of the overall city composition (i.e., minority threat), indicating segregation extends to where people shop, dine, and socialize.

According to 2020 US Census Bureau data, White residents still make up the majority of the US adult population, despite the rapid growth of other racial and ethnic groups in recent decades.[129] This is not the case among youth under the age of 15, with 2018 census data indicating that White youth were no longer the majority youth population.[130] While these statistics are not problematic themselves, what is problematic are the disparities experienced by youth of color. For every racial or ethnic group, children are more likely to live in residential segregation compared to adults.[41]

Segregation is also linked to segregated schools, subsequently impacting school quality and educational outcomes.[41] One study using 2000 decennial census tract data from 318 US Metropolitan Statistical Areas (MSAs) found that while overall Black-White segregation (ID=0.600) among all households was high, segregation was even higher when considering only Black and White married households with children (ID=0.657).[131] This number was even higher when considering only Black and White married households with children that were also living in poverty (ID=0.796). Therefore, while income exacerbates rates of residential segregation among households with children, it does not fully explain why families with children live in higher rates of residential segregation compared to those that do not have children.[41]

Intersection of Economic and Racial/Ethnic Residential Segregation

Given the intersectionality of race and SES in the US, one cannot examine racial and ethnic residential segregation without considering economic segregation and the complex relationship between the two.[132] Despite poverty reaching an all-time low in 2019 since it was first measured in 1959, 34 million Americans still live in poverty (10.5%).[133] While overall poverty rates have declined in the US since 1970, economic residential segregation at the census tract level has increased.[134] Children and youth 18 years of age and younger are more likely to live in poverty (14.4%) when compared to adults. Furthermore, children 18 years of age or younger represent 30.8% of individuals living in poverty, yet they comprise only 22.4% of the US population.[133]

A 2012 Study by the Pew Research Center for Social and Demographic Trends, examined trends in economic residential segregation for the nation's largest 30 MSAs and found that in the last three decades, economic residential segregation has increased for 27 of these 30 major MSAs.[135] Specifically, in 2010, 28% of lower-income households lived in majority lower-income neighborhoods, compared to 23% in 1980. During the same time, the number of high-income households living in majority high-income neighborhoods doubled (9% to 18%) from 1980 to 2010. These shifts may be at least partially attributable to the increase in income inequality over the same period, with a marked decrease in America's middle class.[135] In 1970, 62% of Americans were middle class, compared to 43% in 2015. During this same time, there was only a 1% decrease in the number of individuals in the lower-income category, indicating the economic gap between high income and middle-income households grew larger, on average.[136]

In recent decades, families with children have been most impacted by increases in income segregation, with rates of income segregation two times higher than among families without children.[41] Further, disparities exist by race and ethnicity among poor children, with Black and Hispanic children more likely to live in economically segregated neighborhoods, compared to poor White children.[41] Children of color are also more likely to live in single, female-headed households, which are most likely to live at the highest levels of poverty.[137] These findings are especially problematic given the strong link between neighborhood-level income, school quality, and educational attainment.[134]

While there have been promising shifts in the racial and ethnic diversity of the middle class in recent decades, there remains stark inequality in income by race and ethnicity in the distribution of wealth and burden of poverty.[136,138] A 2019 study conducted by the Brookings Institute found that from 1979 to 2019, the percentage of Black, Hispanic, and individuals from other race categories in the middle class rose from 16% to 40%.[138] While this distribution is similar to the nation's racial and ethnic composition, there is variability within the middle class (the middle 60%), with White Americans overrepresented in the higher levels of income within the middle class. Semega et al. reported that the median household income for non-Hispanic White Americans was $76,057, compared to $45,438 among Black and $56,113 among Hispanic (White and non-White) households.[133]

Looking at the intersection of SES and racial segregation in the US, a 2021 study found that the poverty rates among highly segregated Black communities was much higher (21%) compared to highly segregated White communities (7%).[39] The study also found that living in highly segregated communities is associated with decreased economic mobility. Black youth who grew up in highly segregated neighborhoods earned $1000 less on average annually as adults and Hispanic youth earned $844 less on average, when compared to their peers who grew up in integrated neighborhoods. Another study, examining the relationship between racial/ethnic and income segregation, found that 76% of Black children and 69% of Hispanic children lived in higher poverty neighborhoods, compared to the "worst-off" (defined as the 25% who live in the highest poverty neighborhoods in each of the 100 largest US MSAs) White youth.[41]

Relationship between Racial Residential Segregation and Health

Health inequities, defined by the World Health Organization, are systematic differences in health status, or in the distribution of opportunities to support optimal health that lead to unfair and avoidable differences in health outcomes.[44] Persistent inequities by race and ethnicity have been part of the US health gradient for centuries and remain high, despite being part of the national health agenda for organizations, such as Healthy People and the National Institutes of Health.[43,45] While the driving factors of health inequities by race and ethnicity are not fully understood, racism- and in particular structural racism- has been identified as one mechanism by which health inequities emerge and are sustained.[34,45,46] Structural racism is defined as the macrolevel forces, including policy, law, and culture that produce differential opportunities and exposures for some groups.[45,139] Residential segregation is not solely a product of self-selection into neighborhoods; it acts as a system-level determinant of racial and ethnic health disparities, in which minority-segregated neighborhoods lack public and private investment and resources as a result of institutional forces and social stratification that is further compounded by concentrated neighborhood-level poverty.[21,140]

The impact of residential segregation on health and well-being, while well documented in the literature for a number of decades, is continuing to emerge as a relevant public health and societal issue.[40,141] Residential segregation can have both short and long-term consequences on life opportunities and health outcomes and acts through multiple pathways.[42] Over time, residential segregation can create cumulative disadvantage that encompasses both time (historically rooted and constantly evolving) and space (where we live and work).[21] Previous research on the relationship between racial residential segregation and health has found associations between living in highly segregated neighborhoods with a number of health and health outcomes, including, heart disease, obesity, asthma, birth outcomes, and most recently, Covid-19 infections and mortality.[48-53]

In 2015, one study using data from the Multi-Ethnic Study of Atherosclerosis found that with each standard deviation unit increase in Black-White residential segregation, there was 12% increase in the hazard of developing cardiovascular disease among Black individuals, after controlling for individual and neighborhood-level SES and individual cardiovascular disease risk.[52] The same study found that increased Black-White segregation was a protective factor against cardiovascular disease risk among White residents, but that the association was no longer significant after controlling for neighborhood-level factors.[52] Popescu et al. examined the association between residential segregation and the Black-White survival gap using a nationwide sample of 2009 to 2013 CDC mortality data. Residential segregation was negatively associated with Black survival from age 35 to 75, but there was no association with White survival and the Black-White survival gap was only partially explained by income inequality.[42] A 2017 systematic review on the relationship between residential segregation and birth outcomes consistently found that among Black women living in the most residentially segregated neighborhoods, the odds of pre-term birth were 17% higher and the odds of low birth weight was 15% higher when compared to Black women living in the least residentially segregated neighborhoods.[49] The same review found decreased, or no risk, for White mothers who lived in highly segregated neighborhoods.

Using electronic medical record data for 446,152 children in New Jersey, Alexander and Currie examined the relationship between low birth weight and asthma by race/ethnicity and neighborhood minority composition.[50] After controlling for mother-level effects, such as smoking status, education, and marital status, the relationship between low birth weight and asthma was the same for children of all races and ethnicities who lived in Black zip codes and children living in Black zip codes were at higher risk for asthma compared to children living in White zip codes. These findings suggest that neighborhood-level characteristics of Black zip codes contribute to higher rates of asthma for all children living in these neighborhoods. Kotecki et al. explored the relationship between residential segregation and four child health outcomes: child mortality, teen births, children in poverty, and disconnected youth. As Black-White segregation at the MSA level (measured by the ID) increased, Black youth had poorer outcomes across all four health indicators, across MSAs of all size and minority composition.[142] They also found that in small MSAs, with a high population of only Black residents, White youth had higher rates of teen births and disconnected youth.

Racial and Ethnic Residential Segregation and Adult Obesity

Previous research also links living in a residentially segregated neighborhood to increased risk of obesity, even after adjusting for individual level SES.[51,54,143,144] While the link between living in a residentially segregated neighborhood and obesity risk is especially clear among Black women, the relationship is less clear among Black men and Hispanic men and women.[40,51,55] In fact, there is some evidence that residential segregation can act as a protective factor against obesity risk for Hispanics and individuals born outside of the US by buffering negative exposures, such as experiences of personal discrimination and the negative effects of acculturation.[21]

A 2018 longitudinal study of Black and White Americans, ranging from 18 to 30 years of age, (n=5,115) from four US cities (Birmingham, Chicago, Minneapolis, and Oakland) examined the relationship between residential segregation and risk of adult obesity. High residential segregation was associated with increased hazard of obesity for Black women across 25 years (from early adulthood to middle age) and was strongest when examining cumulative exposure.[51] Among Black men, there was no consistent pattern in the association between residential segregation and risk of obesity. The same study found that regardless of race, living in a neighborhood that has more than 25% Black residents increases the odds of having obesity. These findings suggest that neighborhood opportunity and built environment may play an important role in individual obesity incidence.

In 2015, Bower et al. conducted a cross-sectional study on the association between Black-White segregation (measured by the isolation index) and obesity among Black and White women.[144] Using multi-level logistic regression, the researchers found that for every one-point increase in Black isolation, there was a 1.06 (95% CI= 1.01, 1.11) times higher odds of obesity risk among Black women, after controlling for individual and neighborhood-level covariates, including education, age, marital status, household poverty, neighborhood-level poverty, and region. Conversely, for every one-point increase in Black isolation, there was a 6% decrease in the risk of obesity among White women, after controlling for individual and neighborhood-level covariates.

In a 2018 study, Do et al. used longitudinal data from the Multi-Ethnic Study of Atherosclerosis to examine the relationship between residential segregation and obesity among Black, White, and Hispanic adults.[143] Cross-sectionally, increased segregation was negatively associated with obesity risk among White women and positively associated with obesity risk among Hispanic women, after controlling for neighborhood-level poverty, but no other associations were found by race/ethnicity or gender. Conversely, when examining the longitudinal models, the same study found no association between segregation and obesity among White women, but they did find a positive association for Black women and a negative, but small association, for Hispanic men and women.

Racial and Ethnic Residential Segregation and Youth Obesity

While the relationship between residential segregation and adult obesity is still emerging, even less research has explored the relationship between residential segregation and youth obesity.[56] Given that obesity trajectories are shaped and solidified during early childhood, a better understanding of the relationship between residential segregation and its influences on neighborhood resources and youth obesity is critical to the reduction and prevention of the US youth obesity epidemic.

Using a nationally representative sample (n=1910) of US youth, Ryabov found that after controlling for individual and family-level covariates, racial/ethnic segregation explained between 5% and 20% of the difference in obesity and obesity-related chronic disease risk (diabetes, asthma, hypertension) for Black and Hispanic children compared to their non-Hispanic White peers.[56] The study also found that regardless of race or ethnicity, all children living in more residentially segregated communities had greater odds of having obesity or an obesity-related chronic disease compared to children who lived in less residentially segregated communities.

Another cross-sectional study conducted by Piontak et al. examined the relationship between neighborhood and school contexts with youth obesity, including neighborhood and school-level poverty and segregation using multilevel logistic models in a sample of third and fifth grade elementary school youth in North Carolina.[57] The researchers found a significant and positive association between school poverty and youth obesity risk, but after controlling for school-level poverty, the positive association between school-level segregation and youth obesity was not statistically significant. When county-level residential segregation and other county level characteristics, including urban versus rural classification, percent receiving SNAP benefits, percent uninsured, food desert status, and poverty were added to the model containing individual and school-level characteristics, neighborhood residential segregation was also not statistically significant.

While they did not specifically use residential segregation as their independent variable, Kimbro and Denney looked at neighborhood characteristics, including the percentage of black residents, and the relationship between early childhood obesity.[145] They found that while neighborhood characteristics did not explain a large proportion of the variance in youth obesity, youth living in neighborhoods with a higher proportion of black residents, regardless of individual race or ethnicity, had 17% higher odds of having obesity, and youth living in neighborhoods with a higher proportion of foreign-born residents were at lower risk for having obesity.

No other studies were identified that specifically explored the relationship between residential segregation and youth obesity. One natural experiment examined the association between change in residential segregation and cardiovascular health outcomes and BMI among youth (n=2250) attending a multisite park-based afterschool fitness program.[146] The study results indicated that after controlling for demographic characteristics and park-area poverty, youth who attended a program in a less segregated area, compared with their home area, had greater improvements in cardiovascular fitness and BMI, compared to youth who attended the program in a neighborhood with similar levels of segregation to their home area.

Relationship between Racial Residential Segregation, Opportunity, and Obesity Risk

Future studies on the relationship between residential segregation and obesity remain a crucial area of exploration, given that the causal pathways between residential segregation and adult obesity are not clear and there is a paucity of evidence on the relationship between residential segregation and youth obesity.[56] Under the premise that residential segregation serves to concentrate disadvantage, some previous neighborhood research has explored the mediating pathways of inequitable access to the built or social environment resources in the association between residential segregation and health outcomes, including obesity.[30,58,147,148]

This link between neighborhood resources and health is especially important in the context of racial and ethnic health inequities because regardless of family income, Black and Hispanic youth are more likely to live in low income neighborhoods that are also more likely to have limited social and physical environment features, when compared to White families.[41] Acevedo-Garcia et al. found that while there is variability in childhood opportunity across communities and regions across the US, as measured by a composite index of environmental, educational, and SES indicators such as school quality or access to parks and greenspace, the majority of variability in childhood opportunity occurred within MSAs (>90%).[149] The same study found that 40% of Black youth and 32% of Hispanic youth lived in the bottom quintile of neighborhoods with the lowest childhood opportunity scores compared to only 9% of White youth, and that these disparities persisted after controlling for family poverty levels.[40]

A 2018 study, focusing on quality of food environments, examined the association between residential segregation and adult obesity while considering the mediating influence of the neighborhood-level food environments across the St. Louis and Kansas City metro areas.[58] The study findings suggested that the positive association found between residential segregation and BMI was partially mediated by dietary behaviors and fully mediated by the food environment. Chang et al. focused on neighborhood disorder when examining the relationship between residential segregation and youth obesity, finding a significant and positive association among women regardless of individual race, income, and education, but no association among men.[30] Further, the study found that neighborhood disorder, as measured by an index of property vacancies, percent of residential units that are public housing, property fires, and code violations, partially mediated the relationship between residential segregation and obesity.

Another study, examining the association between neighborhood minority composition and obesity risk by race and gender, explored the mediating effect of neighborhood features, including SES, social cohesion, street connectivity, and parks availability.[147] The study found no association between neighborhoods with a high proportion of Black residents and increased obesity risk for Black women, Black men, or White men. However, there was a significant and positive association between living in a neighborhood with a high proportion of Black residents and obesity risk among White women and that the association was partially mediated by lower neighborhood social cohesion and SES. Conversely, Kershaw et al. found that while there were significant associations between residential segregation and obesity risk for Black (positive) and Hispanic women (negative), these associations were not mediated by neighborhood-level poverty or income incongruity with neighborhood of residence.[55] The studies, detailed above, provide preliminary evidence on the potential mediating factors in the association between residential segregation and obesity, but there remains limited empirical evidence illuminating the causal mechanisms between the two.[55,58]

Structural Racism and the Origins of Residential Segregation in the US

Residential segregation does not emerge, nor is it sustained, through one single mechanism, but, the majority of modern day residential segregation can be attributed to systemic structural racism, both de facto (by common practice) and de jure (by the law).[38]

Two major explanations of how residential segregation arises include spatial assimilation theory and the more nefarious spatial stratification theory.[21,38] Spatial assimilation theory is conceptualized as the self-selection, often tied with economic limitation, of immigrants into communities of familiarity that create communities of shared social capital and culture to help navigate issues such as language barriers or employment.[21] While offering supports or benefits to the residents, these neighborhoods are often located in less desirable areas with greater economic constraints when compared to the broader community. This type of segregation is most salient in the Hispanic/Latino community, which is experiencing high rates of growth across the nation, but particularly in the American south.[33,150]

This study will focus on spatial stratification theory, which is the ordering of groups based on some perceived hierarchy that creates spatial separation by group membership and where the majority group uses their economic, social, and political influence to separate themselves into locations that are more desirable.[21] Residential segregation that rises through this mechanism is most salient to the empirical exploration of the relationship between residential segregation and health inequities because, as Kramer highlights, it is not the race or ethnicity of one's neighbors that influence health outcomes, but rather the processes that give rise to residential segregation and their interrelation with health related factors.[21]

A prime example of spatial stratification in the US is the federal housing policies and practices, highlighted by Rothstein in his book *The Color of Law,* that emerged in the 1930s because of the New Deal.[38] These policies and practices corresponded with rising rates of residential segregation that persisted into the 1960s and created a modern day precedent of acceptance, or even the pursuit of housing separation by race/ethnicity, where Whites are willing to pay premiums to live in segregation from residents of color.[38] These policies were created and sanctioned by Federal Housing Authority to address a national housing shortage and included the practice of redlining neighborhoods in and proximal to Black neighborhoods, thus allowing lenders to refuse to insure mortgages in these areas.[151] Redlining created barriers to home ownership and generational wealth building opportunities for Black families, helping cement the race and economic gradient, as well as limiting choice and quality of housing and neighborhood.[38,152]

Residential Segregation and Racism as Fundamental Causes of Health Disparities

In 1995, Link and Phelan defined fundamental causes of health inequities as those that involve access to resources that enable or prevent disease, affect multiple health conditions through multiple pathways, and that are either sustained or continue to emerge over time despite innovations in health interventions.[153] In the following decades, significant research was dedicated to understanding differences in health outcomes by SES and race, but less attention was paid to how inequities in SES by race emerge.[34] Building upon Link and Phelan's seminal work identifying SES as a fundamental cause of health inequities by race and ethnicity, in 2001, Williams and Collins proposed residential segregation as an additional fundamental cause of health inequities by race and ethnicity.[34] Therefore, if residential segregation is a fundamental cause of health inequities by race and ethnicity, research must examine its health implications beyond just that of access to resources and opportunities and extend consideration to how residential segregation, through the mechanism of structural and interpersonal racism, results in increased burden of behavioral health risk factors and toxic stress.

While SES in the US closely tracks with race and ethnicity, there is an independent pathway by which race and ethnicity impact individual health inequities among people of color. These race and health inequities are largely driven by experiences of direct discrimination and systemic, structuralized racism that create inequities in opportunity for people and communities of color.[154] In 2015, Phelan and Link proposed systemic racism as another fundamental cause of enduring health inequities, meaning that we cannot reduce the enduring health inequities by race and ethnicity without addressing the upstream causes of residential segregation.[47] The authors concluded that racism is a fundamental cause of inequalities in SES by race ethnicity, which subsequently drive health inequities through lack of education and knowledge about disease risk factors and adequate resources to prevent and treat disease. They also found credible evidence that experiences of racism have a direct pathway to health inequities by race and ethnicity through differential power, neighborhood context, and health care.

Supporting this theory of systemic racism as a fundamental cause is a wealth of evidence that as the Black-White educational attainment gap has decreased, the SES gap has not decreased at the same rate.[45] Further, the socioeconomic-health gradient is not equal by race and ethnicity. Black Americans are less likely to experience decreases in morbidity and mortality and improvement in disease outcomes with increasing educational and SES compared to their White peers.[47,140,155,156] Bell et al. examined indicators of structural racism, including residential segregation and racial income equality, and their association with obesogenic environments and obesity risk using

Robert Wood John Foundation County Health Rankings and County Business Patterns.[156] The authors found preliminary evidence to suggest that structural inequality, by race/ethnicity, was associated with higher obesity risk. Specifically, after controlling for county-level SES and other covariates, racial inequality by poverty, unemployment, and home ownership were associated with higher obesity rates.

In addition to experiences of structural racism, interpersonal experiences of racism, defined as differential actions toward or assumptions about the abilities of people according to their race, have the potential to have deleterious impacts on health outcomes.[46,139] Cozier et al. examined the association between individual experiences of racism and obesity risk among Black women.[157] They found that both everyday racism and lifetime racism were associated with an increased risk of obesity, where Black women with the highest reported burden of racism were 1.69 times more likely to have obesity, compared to those that reported the lowest burden of racism, but they did not find that residential segregation modified association between racism and obesity. A study from the United Kingdom examined the association between maternal experiences of racism and childhood obesity risk and found differences between high and low experiences of racism, with higher experiences of racism corresponding to increased obesity risk.[158]

Significance and Innovation

Despite decades of intervention, childhood obesity remains a significant issue in the US that has both short and long-term health consequences at the individual level.[1,4,68] These individual-level consequences also extend to the societal level, contributing to significant levels of morbidity and mortality as well as large amounts of health-care spending related to chronic disease treatment.[10,159] Neighborhood research has been – and continues to evolve as – a crucial area of research to understand how obesogenic environments contribute to increased rates of youth obesity and corresponding inequities by race and ethnicity, income level, education, gender, etc.[23,52,149] Residential segregation and its relationship with opportunity and neighborhood physical and social environments is one such line of inquiry that remains under-explored in the efforts to better understand the relationship between neighborhoods and youth obesity.[21,141]

There is a growing body of evidence on the relationship between residential segregation and obesity risk, but the evidence remains mixed with some studies reporting significant and positive associations, while others have reported null and, in some cases, even negative associations. Consistency of these associations has also varied significantly by race/ethnicity, nativity, and gender.[51,54,144] By gender, the most consistent results have indicated positive associations among women, and in particular Black women, while associations between residential segregation and obesity among men have remained markedly mixed.[40,53] Similarly, reported associations between residential segregation and obesity risk among Hispanic residents and Hispanic immigrants are inconsistent, with some studies pointing towards immigrant enclaves acting as protective and others indicating they may increase obesity risk.[21,33,150]

One strong explanation for these inconsistencies in findings in the relationship between residential segregation and obesity risk is that the majority of previous studies have not used consistent or precise measures of residential segregation and weight status.[40,143] For example, previous studies commonly-used less valid measures, such as the percent of residents who identify as Black, as a proxy for residential segregation as well as self-reported height and weight measures to calculate weight status.[51] In a systematic review of the evidence on the relationship between residential segregation and youth obesity, Corral et al. identified 11 empirical studies; eight (78%) found a positive association between residential segregation and obesity, however, only half of the eight used valid measures of residential segregation and obesity.[40] The present study will expand upon this gap in the literature by using validated measures of residential segregation and objectively collected height and weight measures to calculate youth weight status using CDC guidelines.

While there is a growing evidence base on the relationship between adult obesity and residential segregation, fewer studies have focused on youth obesity.[56,57,59] Only three studies were identified where the primary outcome measure was youth obesity and the model included some neighborhood-level indicator of residential segregation; and of these three, only one used residential segregation as the independent variable of interest. Most studies examining segregation and youth obesity have looked at school-level segregation (i.e., school minority composition).[56] While school-level segregation is related to neighborhood-level segregation, the two are not always well matched because of school choice or district school zoning that does not align well with neighborhood of residence. Further, the majority of studies examining school segregation have used minority composition, rather than a validated measure of residential segregation.[160]

Ryabov was the one study identified as part of this literature review that used neighborhood-level residential segregation as the independent variable of interest and youth obesity as the dependent variable of interest.[56] While the study included a valid measure of residential segregation (Index of Dissimilarity), it included no other neighborhood-level characteristics as covariates, including neighborhood-level SES, which is a potential confounder. Additionally, youth obesity was measured using self-report. Ryabov's study focused on the impact of family-level covariates, such as household marital status, income, and education. Another missing component of this study that is important to consider is school of attendance, which the proposed study will incorporate, thus allowing for the examination of the relative contribution of neighborhood versus school environments in youth obesity risk.

Despite the now decades of research that emphasizes the multifactorial influences on youth obesity, much of the extant research on the contribution of settings, such as schools and neighborhoods, has examined these contexts in isolation.[22,93,161,162] This is exemplified by a review conducted by Arcaya et al. in which only 259 of the 7140 research articles about neighborhoods and health outcomes published between 1995 and 2014 used multi-level models (MLMs) to parse out the relative contribution between individual and neighborhood-level effects on health outcomes.[22] The present study presents not only the opportunity to account for the clustering effect of multiple levels of influence (i.e. individual and neighborhood) on youth obesity risk, but also to introduce a second level-2 unit (schools) using a cross-classified, multi-level model (CCMM). CCMMs are an extension of traditional MLMs that will allow this study to understand the relative contribution of school versus neighborhood environments while also allowing school of attendance and neighborhood to vary by child simultaneously (i.e., not hierarchical).[93] Therefore, while CCMMs still do not indicate causality, they do represent a significant advancement in methodology.[162] Dunn et al. used CCMMs to examine the relative influence of neighborhood versus school environments on adolescent smoking behaviors in the US.[93] After the application of a CCMM statistical approach incorporating schools as a cross-classified level-2 unit, the contribution of neighborhood environment on youth smoking behaviors decreased from 5.2% to only 0.4%.

The majority of studies to date have focused on large urban areas such as Detroit, Chicago, or Los Angeles, or have focused on larger neighborhood units such as zip codes, cities, or counties.[58] The relationship between neighborhood and health outcomes is nuanced by history and geography and the pathways through which regional residential segregation emerges within and across communities varies.[23,141] In an examination of childhood opportunity by race and ethnicity, Acevedo-Garcia found that while opportunity varied by region, with the South having the lowest average and the Northeast having the highest average opportunity, only 9% of the variability was across MSAs compared to 91% within the MSA.[149] Krieger et al. examined the association between racial and income segregation with fatal and non-fatal assaults at both the citywide and census tract levels and found there was a stronger association between residential segregation and assaults at the census-tract level, compared to the city level.[163] The present study presents an important opportunity to examine the relationship between residential segregation and youth obesity using smaller neighborhood-level units (census tracts) in a large southeastern county that is predominantly suburban, with small pockets of urban and rural areas.

While there is growing and significant evidence that neighborhoods influence youth obesity, there remain significant gaps in the literature on the mechanisms by which neighborhood characteristics influence youth obesity risk. This study also extends residential segregation and obesity research by examining if select built and social environment characteristics mediate the association between residential segregation and youth obesity. For example, Sharifi et al. found that while neighborhood SES and food and physical activity environments attenuated the relationship between neighborhood of residence and youth obesity, they did not completely explain the racial/ethnic disparities observed, even after controlling for individual characteristics.[164] This finding could be the result of several unmeasured neighborhood factors driving racial/ethnic disparities in youth obesity, such as residential segregation.

To advance understanding of the association between residential segregation and youth obesity, this study attempted to address several gaps in the literature about the association between residential segregation and obesity, outlined above. These gaps included: 1) variability in results by race/ethnicity and gender, 2) lack of consistent and valid measures of residential segregation and obesity, 3) lack of evidence on the relationship between residential segregation and youth obesity, 4) lack of statistical techniques that control for the effect of clustering or competing environments such as schools, and 5) lack or research on the mechanisms and pathways by which neighborhood residential segregation impacts youth obesity.

Table 2.1. Constructs and definitions of residential segregation

Massey/Denton Constructs	Massey and Denton Definitions		Johnston et al. Composite Constructs	Johnston et al. Definitions
Evenness	How evenly or unevenly a group is spread across a region	→	**Separateness**	Composite definition involving evenness, exposure, and clustering; defined as the degree to which members of a racial/ethnic group live apart from the remainder of the population in defined area such as county or MSA
Exposure	Likelihood of neighborhood-level interaction between members of two groups	→		
Clustering	How much neighborhoods predominantly made up of the same group are located near each other	→		
Centralization	How much a group is concentrated at central or urban core	→	**Location**	Composite definition involving centralization and concentration; defined as the degree to which members of a racial/ethnic group are congregated, regardless of their degree of separation, into high-density, inner-city areas
Concentration	Relative density per area unit a group occupies compared to other groups	→		

METHODOLOGY

CHAPTER THREE

This dissertation is part of a larger body of research aimed at better understanding how school and neighborhood healthy eating and active living environments, and disparities within these environments by race/ethnicity and SES impact youth obesity rates. The following methods chapter describes the conceptual framework, study setting, data collection procedures, study measures, and analytical approach used in this study.

3.1 CONCEPTUAL FRAMEWORKS

The conceptual model for this study drew from two different frameworks and is infused with concepts from the social ecological model, described in the background section. The first framework that guided the development of the conceptual model for the current study was proposed by Popescu et al. and describes the pathways by which residential segregation impacts health (Figure 3.1).[42] The framework suggests three primary pathways by which residential segregation produces health inequalities by race and ethnicity, each of which are rooted in exposures to institutional and interpersonal racism. The first pathway is through the relationship between residential segregation and income inequality, where minority residentially segregated neighborhoods are more likely to have high rates of poverty when compared to predominantly White neighborhoods. This income inequality is linked to limited educational and employment opportunities in minority-segregated communities. The second pathway is through differential access to neighborhood amenities and services, such as parks, safe sidewalks, streetlights, and social services, where increased access and quality can enhance health and lack thereof can limit health. The third pathway is through differences in health-care delivery, where residents of minority neighborhoods are less likely to have insurance (linked to job and income inequality) and access to high quality health services and providers.[41]

The second framework that this study drew from was proposed by Suglia et al. and details the relationship between neighborhood physical and socioeconomic environments and obesity risk (Figure 3.2).[25] Suglia et al. proposed neighborhood-level SES as the primary driver in neighborhood built and social environment disparities, which may be protective of or, conversely, may increase risk for obesity. Important social environment features include social norms, social cohesion, and community crime and safety. These factors have a direct influence on energy balance as they affect individual food and physical activity behaviors. They also have an indirect influence on food and physical activity behaviors through individual mental health status. Built environment features include walkability, park access, and access to healthy food, and they have a direct pathway to food and physical activity behaviors. Suglia et al. also proposed that the neighborhood social environment moderates the effect of the built environment on obesity and vice versa. For example, if a resident perceives their neighborhood as being unsafe, they may be less likely to engage with the built environment features in their neighborhood that are supportive of physical activity.

Building upon the aforementioned conceptual models, the conceptual model for this study illustrates the hypothesized relationship between residential segregation and youth obesity risk (Figure 3.3). On the left side of the model, structural racism is the primary driver of residential segregation and inequalities in the distribution of resources at the neighborhood level. There is a bidirectional relationship between these two factors in that predominantly Black or brown neighborhoods receive less economic investment and resources and Black and brown families' choice of residence may be limited to minority residentially segregated neighborhoods that are under-resourced. The model then shows that residential segregation and inequality of resources influence quality of neighborhood physical and socioeconomic environments, including features such as access to parks, walkability, crime, poverty, and social cohesion. These neighborhood-level features affect healthy eating and active living behaviors and, in turn, obesity and chronic disease risk. In this model, it is hypothesized that the relationship between residential segregation and youth obesity is partially mediated through the pathway of neighborhood social and built environment disadvantage.

While not directly measured in this study, mental health does have an important place in the model. Structural racism sustains disparities in neighborhood environments. It also facilitates experiences of interpersonal racism or discrimination by influencing individual perceptions of race and class, which can result in negative mental and behavioral health outcomes. Neighborhood environment features, including perceptions of safety and neighborhood cohesion and capital, also can negatively influence mental health and behavioral health outcomes. Negative mental health and behavioral health outcomes then influence obesity and chronic disease risk in two ways. The first is through decreased healthy eating and active living behaviors that also increase obesity and chronic disease risk. The second mechanism is through the cumulative burden of chronic stress (i.e., allostatic load) that has a direct impact on chronic disease risk, regardless of obesity.

3.2 STUDY SETTING

Located in the northwest corner of SC, Greenville County is the most populous county in the state with approximately 523,542 residents as of 2019.[60] The geography is predominantly suburban with smaller pockets of rural and urban areas. Greenville County is experiencing higher rates of growth (16% since 2010), when compared to both the state and the nation. Persons under the age of 18 represent 22.9% of the population and children under the age of five account for 6.2% of the population. The county is predominantly non-Hispanic White (76.3%), followed by non-Hispanic Black (18.4%) and Hispanic (9.5%) residents. Approximately 87.3% of residents over the age of 25 have their high school diploma and 33.3% have a bachelor's degree or higher.

The median household income is $60,351 and approximately 10.6% of individuals live in poverty, which is fewer than the 12.3% across the nation and 13.9% in SC. Children under the age of 18 are more likely to live in poverty (15.0%) and children under five are at greatest risk (21.3%) of living in poverty. Children of color are also more likely to live in concentrated poverty, with Hispanic youth the most likely live in concentrated poverty (26.7%), followed by Black youth (19.1%), and White youth (3.9%).[165] The local school district is a unified district and is the 44th largest in the nation. The district covers more than 800 square miles with 51 elementary schools, 20 middle schools, 14 high schools, and 16 special schools or centers that serve more than 76,000 students.[61] Approximately 30% of adults have obesity and 18.8% of third through fifth grade youth have obesity.[63,166]

3.3 DATA COLLECTION

The following section describes the data collection procedures for each of the sources used in this study, including individual youth, school, and neighborhood data.

Study Sample

The study sample includes second, fifth, eighth, and ninth grade youth from 80 elementary, middle, and high schools across Greenville County, SC. Youth included in the sample are those who participated in physical education classes and had complete height, weight, demographic, and address data for the spring data collection time point during the 2016-2017 academic year. Additional inclusion criteria were identifying as belonging to the White, Black, or Hispanic racial/ethnic categories. Students who identified as belonging to the "Other" race/ethnicity category were not included in the final study sample because of the small sample size of the category as well as the heterogeneity within the category.

Youth Weight Status and Demographic Data

All youth level data were obtained from the local school district through a data sharing agreement with Furman University, in partnership with a countywide healthy eating and active living coalition. Trained physical education teachers collected height, weight, and physical fitness data twice annually, once at the beginning of the school year and once at the end during the 2016-2017 academic year. Physical education teachers entered the data into the *FitnessGram* database, a comprehensive fitness assessment battery for youth that includes a number of health- related physical fitness tests designed to assess cardiovascular fitness, muscle strength, muscular endurance, flexibility and body composition.[64] This study utilized height and weight measurements collected using a SECA standardized stadiometer (specific measures derived using these data are described in the following section).

Youth demographic data, including a unique student identifier, age in months, biological sex, race/ethnicity, grade, school of attendance, and home address were obtained from the district's *PowerSchools* database. *PowerSchools* is a student information software system that warehouses student socio-demographic and school-related data.[167] Parents and/or guardians verify the *PowerSchools* data each year, as part of school enrollment, through an online portal linked to individual student profiles. District employees mined and compiled all youth-level data into a single database and transferred it to the study team via an encrypted online portal.

School-level Characteristics

The SC State Department of Education, as part of state, district, and school performance monitoring, collects a wealth of school-level data as part of their annual district and school report cards.[62] Additionally, the local school district publishes individual school enrollment, poverty, and other data on their website each year. Relevant school-level measures, including minority composition and poverty status, were mined from the state and district websites for the 2016-2017 academic school year. The data were then computed into study variables and merged with the youth-level database by the study researcher.

Neighborhood-level Characteristics

Youth address was also obtained from the local school district as part of the data sharing agreement to identify youth neighborhood of residence (census tract). Youth address was geocoded to the census tract level using Arc GIS technology. Address was then deleted from the database to protect student anonymity. Geocoding involves converting address data to corresponding latitude and longitude, which can then be assigned to areal units including census tracts.[59] Relevant neighborhood characteristics from the ACS 2014 to 2018 five-year estimates, including demographics and education, were downloaded from the National Historic Geographic Information System (NHGIS) website, and merged to the census tract associated with student home address.[168]

Neighborhood social/economic, health/built, and education environment data from 2015, as measured by the COI 2.0, were obtained from the Institute for Child, Youth and Family Policy at the Heller School for Social Policy and Management at Brandeis University.[66] The COI is a measure of relative opportunity across neighborhoods (census tracts) in a metropolitan area, where each neighborhood is assessed relative to the other neighborhoods in the metropolitan area across three domains: social and economic environments, health and built environments, and education environments.

The final step of the data collection process was to merge each dataset into a comprehensive database that joined individual, school, and neighborhood characteristics, enumerated above, and described further below, at the individual-youth level. Youth demographic data and the neighborhood-level census data were joined using a unique identifier that was provided by the district and the US census bureau assigned census tract number.

3.4 MEASURES

The following section describes the study variables derived from the aforementioned data sources, including individual youth, neighborhood, and school characteristics. Individual youth variables included weight status (dependent variable), and demographic characteristics (gender, race/ethnicity, grade level, school attended, address). Neighborhood-level characteristics included residential segregation (independent variable), the COI social and economic z-score, the health and built environment z-score, the education environment z-score, and other relevant neighborhood characteristics. School-level characteristics include Black and Hispanic school segregation along with school poverty.

Individual Youth Measures

Youth Weight Status. Youth weight status was the dependent variable of interest in this study and was operationalized in analyses as BMI z-score, which was treated as a continuous variable. Youth height, weight, and test date from the *FitnessGram* database were used along with biological sex and age in months from the *PowerSchools* database, to calculate youth BMI z-scores, using the standardized protocols developed by the CDC.[169] Specifically, youth weight status was estimated using body mass index (BMI) percentiles based on sex and age specific height-for-weight. These percentiles are then used to create weight status categories: underweight (<5th percentile), normal weight (5th percentile to <85th percentile), overweight (85th percentile to <95th percentile), and obese (>95th percentile).[170] BMI z-score is the number of standard deviation units above or below the mean BMI percentile of the sample distribution. BMI z-score is widely used in youth obesity research as it accounts for uneven distributions and prevents extreme values from skewing the distribution.[171]

Youth Demographic Characteristics. Youth demographic characteristics were used as covariates to account for their influence on youth weight status (BMI z-score).

Demographic variables from the *PowerSchools* database include biological sex (male=0, female=1), grade (2nd=0, 5th=1, 8th=2, 9th=3), and race/ethnicity (White=0, Black=1, Hispanic=2). The "Other" race category was not included in this study because of the small sample sizes for certain race/ethnicity categories, which limits its use in analysis and interpretation.

Neighborhood-level Measures

Residential Segregation. The independent variable of interest in the study was neighborhood residential segregation by race/ethnicity. This study calculated Black and Hispanic neighborhood-level residential segregation at the census tract level using the Location Quotient (LQ). The LQ is a localized measure of separateness that is calculated by taking the quotient of the proportion of one group at the local level (census tract) divided by the proportion of that group in the broader area (county).[21,172] If the proportion of one group in a local areal unit is the same as that for the overall area, the LQ will equal one, if the local proportion is greater than regional proportion LQ will be greater than one, and if the proportion in a local area is less than the overall area the LQ will be less than one. The LQ has a theoretical range of zero to infinity, but for example, a LQ score of 5 indicates that the population of interest at the local level (census tract) is 5 times that of than the broader areal unit (county). Standardized cut offs for the LQ have been established that are approximately one standard deviation above or below a LQ of one, where less than .85 is underrepresentation of a population and greater than 1.2 is overrepresentation or segregation.[172] The LQ has several advantages, including its ease of calculation and interpretation.[172,173] Black and Hispanic residential segregation LQs were calculated, separately, using ACS data of racial composition at the census tract and county level. The residential segregation LQs were then grand mean centered to improve interpretability of study analyses.

Neighborhood Socioeconomic and Built Environments. Three important dimensions of the neighborhood environment were included in this study. The first was the social and economic environment, the second was the health and built environment, and the third was the education environment. All three environment dimensions were operationalized using domain z-scores from the COI 2.0. The COI measures and maps the quality of resources and conditions at the neighborhood level that influence childhood health and development.[66] The three domain z-scores were used as potential mediators in of the association between residential segregation and youth obesity in aim 2.

In 2014, a team of researchers at Brandeis' Heller School for Social Policy and Management released the original COI 1.0. The goal of developing the COI was to provide the field of child health research and practice with a measure of children's neighborhood opportunity, where childhood opportunity is defined as " the context of neighborhood-based conditions and resources such as access to quality early childhood care and school quality, that influence children's healthy development and long-term outcomes".[66,149] In 2020, the COI 2.0 was released with updated methodology based on lessons learned and newer neighborhood and childhood health datasets.[149]

The COI 2.0 calculates scores for approximately 72,000 census tracts in the US at two time points, which are approximately 2010 and 2015. The COI includes both an overall score of neighborhood opportunity that is composed of three domains – health and environment, social and economic, and education. The three domains are constructed from 29 component indicators that are conceptually and empirically linked to the domain score (Table 3.2). To create the COI domain scores, each of the component indicators are standardized using z-scores to create a uniform measurement scale and then weighted based on their association with select health and economic outcomes. A detailed description of the development and methodology, including validation of COI measures, can be found in the COI 2.0's Technical Documentation.[66]

Neighborhood Covariate. The remaining neighborhood-level measure that was included as a study covariate was neighborhood education level. Neighborhood education level was defined as the percentage of individuals who have a high school diploma or less. It has a potential range from zero to 100, where zero indicates all individuals residing in the neighborhood have greater than a high school diploma and 100 indicating none of the individuals residing in the neighborhood have greater than a high school diploma. The educational attainment variable was grand mean centered and treated as a continuous measure.

School-Level Measures

School Level Segregation and Poverty. School segregation served as a secondary indicator of interest in this study. School segregation was also calculated using the LQ, applying it to the context of schools located within the school district instead of census tracts within the county. Like neighborhood residential segregation, Black and Hispanic school segregation were calculated separately, using racial/ethnic composition data from the SC State Department of Education for the school district and each school. School segregation LQs were calculated by taking the quotient of the proportion of one group within a school divided by the proportion of that group in the school district.

School segregation LQs were also grand mean centered to increase interpretability in analyses and treated as continuous variables.

School-level poverty served as a study covariate, when exploring the association between neighborhood residential segregation and youth obesity, using the School Poverty Index (SPI). The SPI is calculated by the SC State Department of Education for each school and district across the state and is publicly available on their website. The SPI is defined as the proportion of students who are 1) living in poverty as specified by the federal poverty guidelines; 2) students participating in TANF, Medicaid, or SNAP; or 3) students who are identified as a foster child or homeless.[62] SPI scores can range from zero to 100, where zero represents the complete absence of students who live in poverty and 100 indicates that all students live in poverty. The SPI was treated as a continuous measure and grand mean centered.

3.5 ANALYTIC APPROACH

The following section details the descriptive and inferential statistical analyses that were used to explore aim 1 and aim 2 of this study.

Data Analyses

Aim 1a: Determine the relative influences of school and neighborhood environments on youth obesity.

Aim 1b: Examine the association between neighborhood-level residential segregation and youth obesity, after controlling for associated individual, school, and neighborhood individual characteristics.

To accomplish aim 1, this study used as series of cross-classified, multi-level models (CCMM), which are an extension of standard multi-level models (MLM) that account for non-hierarchal, cross-classified data structures.[162] Despite recognition of the complex and multiple levels of influence on youth obesity, statistical techniques have commonly only examined these outcomes as individual or siloed contexts.[93,162] Such an approach ignores the fact that individuals are part of, and influenced by, multiple contexts and more likely to be similar to individuals within their shared contexts, which creates dependence among observations that violate the assumptions of ordinary least squares (OLS) linear regression.[174]

In a standard MLM, data are hierarchal, or nested, whereby children (Level-1 units) are nested within schools (Level-2 units), which are nested within school districts (level-3 units). CCMMs extend the standard MLM by allowing us to examine multiple settings that are not nested within each other (e.g., non-hierarchical), such as neighborhoods and schools, simultaneously. In contrast, a MLM can only incorporate both settings if schools were nested within neighborhoods and all students at the same school always resided in that same neighborhood.[162,175] Using standard regression and MLM techniques that do not account for individuals who are cross classified across settings, can result in significant violations of assumptions and potential sources of error, including but not limited to, independence of data, reduced power to detect treatment or covariant effects, less accurate estimates of variances, inflated Type I errors, and errors in interpreting tests for statistical significance.[175]

Figure 3.4 depicts the cross-classified nature of youth school of attendance and neighborhood of residence.[176] CCMMs account for instances where children living in the same neighborhood, defined as census tracts, do not attend the same school, or vice versa (i.e., not all children who attend the same school live in the same neighborhood).[162] This natural occurrence related to school district attendance zones can be further exacerbated by situations such as school choice, where children attend school well outside of their home neighborhood for academic, extracurricular, or parental preference reasons.[41] This simultaneous examination of the effects of school and neighborhood environment on youth obesity is important to decompose the influence of each of the respective environments and inform effective multi-setting interventions.[93,162]

Model Building Process and Equations

This analysis used two levels, with individual youth as the Level-1 units who are cross-classified between two Level-2 units, neighborhoods and schools. Level-1 predictors included youth weight status (BMI *z*-score) and demographic characteristics, where weight status is the dependent variable of interest. Youth demographic characteristics were used as individual-level covariates and were treated as fixed effects. The demographic characteristics that were used as Level-1 covariates were biological sex (male=0, female=1), grade (second=0, fifth=1, eighth=2, ninth=3), and race/ethnicity (White=0, Black=1, Hispanic=2).

The Level-2 units in this analysis were school and neighborhood characteristics, which were treated as random effects. Treating school of attendance and neighborhood as random effects allowed the influence of these two environments to vary, rather than remaining equal across all schools and all neighborhoods. School-level characteristics included school-level poverty (continuous) and residential segregation (continuous). The primary neighborhood-level characteristic of interest was residential segregation (Black and Hispanic LQs). An additional neighborhood-level characteristic that was included as a study covariate was educational attainment, measured as the percentage of neighborhood residents who have a high school diploma or less. The educational attainment variable was grand mean centered and treated as a continuous measure.

The first step in the analytic approach for aim was to obtain descriptive statistics for each study variable. Bivariate analyses were then used to assess the association between the main outcome variable, youth obesity (BMI *z*-score), and each of the individual, school, and neighborhood characteristics. All analyses were conducted using SAS version 9.4.[177] Next, CCMMs were used to examine the association between residential segregation and youth obesity in the context of school and neighborhood environments, where youth were nested within schools and neighborhoods. Prior to beginning the model building process, the data were examined for violations of assumptions associated with CCMMs, including normality and homogeneity of variance using the techniques recommended by Raudenbush and Bryk (2002).[178,179]

Normality of the Level-1 units was assessed using box and whisker plots of the standardized residuals as well as normality summary statistics including the Shapiro-Wilk test for normality. Homogeneity of variance for the Level-1 units was assessed using scatterplots of the standardized residuals against the predicted values of youth obesity. Normality and homogeneity of variance were assessed for the Level-2 units, schools and neighborhoods, separately. Normality for the Level-2 unites was assessed using histograms of the Level-2 residuals and using multivariate normality diagnostics. Homogeneity of variance for the Level-2 units was assessed using scatterplot of the residuals versus the predicted values of youth obesity and using Levene's test for level-2 random effects by Level-2 predictors.

The next step was to assess whether there was statistical justification for using a multilevel models by calculating the intraclass correlation coefficient (ICC), which measures the proportion of variance in youth obesity that was explained by the Level-2 units.[178] The ICC was calculated using formula 1, below, where $\sigma^2_{u_0}$is an estimate of the Level-2 variance and σ^2_r is an estimate of the Level-1 variance. These variances were obtained by fitting a null-model that includes no Level-1 or Level-2 predictors.

$$P = \frac{\sigma^2_{u_0}}{(\sigma^2_{u_0}+\sigma^2_r)} \quad (1)$$

After calculation of the ICC, the PROC MIXED command was used to build the CCMMs following the model building process elaborated by Raudenbush and Bryk, with maximum likelihood (ML) estimation and Satterthwaite degrees of freedom.[178] ML estimation was chosen over restricted maximum likelihood estimation, given that both methods produce the same fixed-effect estimates and the large number of Level-2 units reduces the potential bias of the random-effect estimates using ML estimation.[179] Predictors that did not have a meaningful interpretation of zero were grand-mean centered, including school and residential segregation LQs and school poverty.

The first step in the model building process (Model 1) was to build the null, or intercept only, model. Formula 2, below, represents the null model at Level-1, where $y_{i(jk)}$ represents the expected BMI z-score for person *i*, at school *j*, who lives in neighborhood *k*. $B_{o(jk)}$ is the average BMI z-score for students at *j* school and who lives in *k* neighborhood and $e_{oi(jk)}$is the random effect of individual *i* who attends *j* school and lives in *k* neighborhood.

Level-1: $y_{i(jk)} = \beta_{0(jk)} + e_{i(jk)}$ (2)

In Level-2 of the null model (formula 3), the Level-1 intercept ($\beta_{0(jk)}$) was modeled as a random effect. γ_{00} is the grand mean BMI z-score across all schools and neighborhoods, u_{0j} is the is the random effect of school *j* averaged across neighborhoods, and u_{0k} is the random effect of neighborhood *k* averaged across schools. Formula 3 and 4 yield the combined null model (formula 4).

Level-2: $\beta_{0(jk)} = \gamma_{00} + u_{0j} + u_{0k}$ (3)

Combined Level-1 and 2: $y_{i(jk)} = \gamma_{00} + u_{0j} + u_{0k} + e_{i(jk)}$ (4)

Model 2, or the control model, included the introduction of Level-1 covariates that were treated as fixed effects, including gender, grade, and race/ethnicity. The inclusion of the Level-1 covariates allowed this study to evaluate to what extent the between-level variance estimates was explained by individual characteristics that vary across schools and neighborhoods. At Level-1 of the control model (formula 5), $\beta_{biosex_{(jk)}}$ is the expected different between male and female youth at school *j* and in neighborhood *k* and $\beta_{grade_{(jk)}}$ is the expected difference between second and fifth graders at school *j* and in neighborhood *k*, after controlling for individual covariates. Race/ethnicity is a categorical variable with more than two levels that requires the creation of a set of dummy variables to be included in the model, where White youth are the referent category. Therefore, in formula 4, $\beta_{Black_{(jk)}}$ is the expected difference between White and Black students at school *j* and in neighborhood *k* and $\beta_{Hispanic_{(jk)}}$ is the expected difference between White and Hispanic students at school *j* and in neighborhood *k*, after controlling for individual covariates. $\beta_{0(jk)}$ now represents the average BMI z-score when all the Level-1 predictors are set at zero, or the average BMI z-score for a male student, in second grade, and who is White.

$$\textbf{Level 1}: y_{i(jk)} = \beta_{0(jk)} + \beta_{biosex(jk)}X_{i(jk)} + \beta_{grade(jk)}X_{i(jk)} + \beta_{Black_{(jk)}}X_{i(jk)} + \beta_{Hispanic_{(jk)}} + e_{i(jk)} \quad (5)$$

At Level-2 (formula 6) of the control model, the Level-1 intercept, $\beta_{0(jk)}$, was modeled as a random effect. γ_{00} now represents the grand mean BMI z-score when all the Level-1 predictors are set at zero, or the average BMI z-score for a male youth, in second grade, and who is White. The fixed effects for each of the Level-1 characteristics across schools and neighborhoods are represented by $\gamma_{10biosex}$, $\gamma_{20grade}$, $\gamma_{30Black}$, and $\gamma_{40Hispanic}$. The interpretation of the random effect parameters (u_{0j} and u_{0k}) is the same as in the previous model, except that it now controls for individual-level fixed effect covariates at Level-2. Formula 7 is the combined Level-1 and Level-2 formulas for the model that includes individual covariates.

$$\textbf{Level 2}: \beta_{0(jk)} = \gamma_{00} + u_{0j} + u_{0k} \quad (6)$$

$$\beta_{biosex(jk)} = \gamma_{10biosex}$$

$$\beta_{grade(jk)} = \gamma_{20grade}$$

$$\beta_{Black_{(jk)}} = \gamma_{30Black}$$

$$\beta_{Black_{(jk)}} = \gamma_{40Hispanic}$$

$$\textbf{Combined Level-1 and Level-2}: y_{i(jk)} = \gamma_{00} + \gamma_{10biosex}X_{i(jk)} + \gamma_{20grade}X_{i(jk)} + \gamma_{30Black}X_{i(jk)} + \gamma_{40Hispanic}X_{i(jk)} + u_{0j} + u_{0k} + e_{i(jk)} \quad (7)$$

Model 3 (school model) included the introduction of the Level-2 school covariates, including school residential segregation and poverty, in addition to the Level-1 covariates from Model 2. The Level-1 formula (8) in Model 3 is the same as Model 2.

Level 1: $y_{i(jk)} = \beta_{0(jk)} + \beta_{biosex(jk)}X_{i(jk)} + \beta_{grade(jk)}X_{i(jk)} + \beta_{Black_{(jk)}}X_{i(jk)} + \beta_{Hispanic_{(jk)}} + e_{i(jk)}$ (8)

At Level-2 (formula 9), the Level-1 intercept, $\beta_{0(jk)}$, was modeled as a random effect and a function of three school level covariates: school level poverty, Black school segregation, and Hispanic school segregation. γ_{00} now represents the grand mean when both the individual and school level predictors are set to zero. In other words, it is the expected BMI z-score for a male, second grade student, who is White, and who attends a school with average Black and Hispanic school segregation and average school poverty. $\gamma_{01schpov}$ is the effect of school level poverty across all schools, while $\gamma_{02schsegblack}$ and $\gamma_{03schseghisp}$ is the effect Black and Hispanic school segregation across all schools. The interpretation of the random effect parameters (u_{0j} and u_{0k}) is the same as in the previous model, except that it now controls for Level-1 and Level-2 school predictors. The interpretation of the gammas representing Level-1 as fixed effects at Level-2 remains the same as in model 2. Formula 10 is the combined Level-1 and Level-2 formulas for the school model.

Level 2: $\beta_{0(jk)} = \gamma_{00} + \gamma_{01schpov}T_j + \gamma_{02schsegblack}T_j + \gamma_{03schseghisp}T_j + u_{0j} + u_{0k}$ (9)

$$\beta_{gender(jk)} = \gamma_{10biosex}$$

$$\beta_{grade(jk)} = \gamma_{20grade}$$

$$\beta_{Black_{(jk)}} = \gamma_{30Black}$$

$$\beta_{Black(jk)} = \gamma_{40Hispanic}$$

Combined Level-1 and Level-2: $y_{i(jk)} = \gamma_{00} + \gamma_{01schpov}T_j + \gamma_{02schsegblack}T_j + \gamma_{04schseghispanic}T_j + \gamma_{10biosex}X_{i(jk)} + \gamma_{20grade}X_{i(jk)} + \gamma_{30Black}X_{i(jk)} + \gamma_{40Hispanic}X_{i(jk)} + +u_{0j} + u_{0k} + e_{i(jk)}$ (10)

Model 4 (neighborhood model) included the introduction of the Level-2 neighborhood predictors, Black and Hispanic residential segregation (LQ), and one covariate, neighborhood educational attainment, in addition to the Level-1 covariates. The Level-1 formula (11) in Model 4 is the same as in Model 2.

Level 1: $y_{i(jk)} = \beta_{0(jk)} + \beta_{biosex(jk)}X_{jk} + \beta_{grade(jk)}X_{i(jk)} + \beta_{Black(jk)}X_{i(jk)} + \beta_{Hispanic(jk)} + e_{i(jk)}$ (11)

At Level-2 (formula 12), the Level-1 intercept, $\beta_{0(jk)}$, was modeled as a random effect and a function of two neighborhood level predictors and one covariate. γ_{00} now represents the grand mean when both the individual and neighborhood-level predictors are set to zero. In other words, it is the expected BMI z-score for a male, second grade student, who is White, and lives in a neighborhood that has an average level of Black residential segregation, an average level of Hispanic residential segregation, and average neighborhood educational attainment. $\gamma_{01residentialblack}$ and $\gamma_{02residentialhisp}$ are the effects of Black and Hispanic residential segregation across all neighborhoods, while $\gamma_{03neighborhoodedu}$ is the effect of educational attainment across all neighborhoods. The interpretation of the random effect parameters (u_{0j} and u_{0k}) is the same as in the previous model, except that it now controls for the Level-1 and the Level-2 neighborhood predictors. The interpretation of the gammas representing Level-1 predictors as fixed effects at Level-2 remains the same as in model 2. Formula 13 is the combined Level-1 and Level-2 formulas for the neighborhood model.

Level 2: $\beta_{0(jk)} = \gamma_{00} + \gamma_{01residentialblack}T_k + \gamma_{02residentialhisp}T_k +$

$\gamma_{03neighborhoodedu}T_k + u_{0j} + u_{0k}$ (12)

$$\beta_{biosex(jk)} = \gamma_{10biosex}$$

$$\beta_{grade(jk)} = \gamma_{20grade}$$

$$\beta_{Black(jk)} = \gamma_{30Black}$$

$$\beta_{Black(jk)} = \gamma_{40Hispanic}$$

Combined Level-1 and 2: $y_{i(jk)} = \gamma_{00} + \gamma_{01residentialblack}T_k + \gamma_{02residentialhisp}T_k +$

$+\gamma_{03neighborhoodedu}T_k + \gamma_{10biosex}X_{i(jk)} + \gamma_{20grade}X_{i(jk)} + \gamma_{30Black}X_{i(jk)} +$

$\gamma_{40Hispanic}X_{i(jk)} + u_{0j} + u_{0k} + e_{i(jk)}$ (13)

Model 5 (school and neighborhood) included both the school and neighborhood Level-2 covariates, in addition to the Level-1 covariates. The Level-1 formula (14) in model 5 is the same model as in model 2.

Level 1: $y_{i(jk)} = \beta_{0(jk)} + \beta_{biosex(jk)}X_{i(jk)} + \beta_{grade(jk)}X_{i(jk)} + \beta_{Black(jk)}X_{i(jk)} +$

$\beta_{Hispanic(jk)}X_{i(jk)} + e_{i(jk)}$ (14)

At Level-2 (formula 15), the Level-1 intercept, $\beta_{0(jk)}$, was modeled as a random effect and a function of the same school and neighborhood-level covariates as in models 3 and 4, respectively. γ_{00} now represents the grand mean BMI z-score when the Level-1 and the Level-2 school and neighborhood covariates are set to zero and have the same interpretation as in models 3 and 4. The interpretation of the school and neighborhood-level gammas remain the same as in models 3 and 4, respectively. The interpretation of the random effect parameters (u_{0j} and u_{0k}) is the same as in the previous model, except that it now controls for the Level-1 and the Level-2 school and neighborhood predictors. The interpretation of the gammas, representing Level-1 predictors as fixed effects at Level-2, remains the same in model 5 as in model 2. Formula 16 is the combined Level-1 and Level-2 formulas for the school and neighborhoods model.

Level 2: $\beta_{0(jk)} = \gamma_{00} + \gamma_{01schpov}T_j + \gamma_{02schsegblack}T_j + \gamma_{03schoolseghisp}T_j + \gamma_{01residentialblack}T_k + \gamma_{02residentialhisp}T_k + \gamma_{03neighborhoodedu}T_k + u_{0j} + u_{0k}$ (15)

$$\beta_{biosex(jk)} = \gamma_{10biosex}$$

$$\beta_{grade(jk)} = \gamma_{20grade}$$

$$\beta_{Black_{(jk)}} = \gamma_{30Black}$$

$$\beta_{Black_{(jk)}} = \gamma_{40Hispanic}$$

Combined Level-1 and Level-2: $y_{i(jk)} = \gamma_{00} + \gamma_{01schpov}T_j + \gamma_{02schsegblack}T_j + \gamma_{03schseghisp}T_j + \gamma_{01residentialblack}T_k + \gamma_{02residentialhisp}T_k + \gamma_{03neighborhoodedu}T_k + \gamma_{10biosex}X_{i(jk)} + \gamma_{20grade}X_{i(jk)} + \gamma_{30Black}X_{i(jk)} + \gamma_{30Hispanic}X_{i(jk)} + X_{i(jk)} + u_{0j} + u_{0k} + e_{i(jk)}$ (16)

The final step in the model building process was to add two different sets of interaction terms, in Model 6 and Model 7, to determine if individual youth race/ethnicity moderated the relationship between segregation and youth BMI z-score. Model 6 included the addition of an interaction term between school segregation and youth race/ethnicity (Black school segregation*Black, Black school segregation*Hispanic, Hispanic school segregation*Black, Hispanic school segregation*Hispanic). Model 7 included the addition of an interaction term between residential segregation and youth race/ethnicity (Black residential segregation*Black, Black residential segregation*Hispanic, Hispanic residential segregation*Black, Hispanic residential segregation*Hispanic).

Three different ICCs were calculated from the unconditional model to determine the percentage of variance in youth BMI z-score that was attributable to schools and neighborhoods versus individual characteristics: school only, neighborhood only, and school and neighborhood. The equations for each of the ICCs are detailed below.

Schools only: $\mathrm{ICC} = \frac{\tau_{j_{00}}}{\tau_{j_{00}} + \tau_{k_{00}} + \sigma^2}$ (17)

Neighborhood only: $\mathrm{ICC} = \frac{\tau_{k_{00}}}{\tau_{j_{00}} + \tau_{k_{00}} + \sigma^2}$ (18)

Schools and neighborhoods: $\mathrm{ICC} = \frac{\tau_{j_{00}} + \tau_{k_{00}}}{\tau_{j_{00}} + \tau_{k_{00}} + \sigma^2}$ (19)

Pseudo R^2 was calculated for each model to determine the proportion of variance they explain. Next, pseudo R^2 comparisons for each of the nested models were made to determine if the proportion of variance accounted for by each subsequent model is more significant than the previous model. Pseudo R^2 increases with the additional of parameters to the model because it is influenced by the degrees of freedom associated with the model.[175,179] Therefore, to determine if the contribution of the additional parameters added to each model in the model building process was statistically significant, likelihood ratio tests using the chi-squared statistic were performed on the differences -2 Log Likelihood for each of the model comparisons. If the difference in the -2 Log Likelihood was statistically significant the increase in pseudo R^2 indicated the additional parameters improved model fit, but if the difference is not statistically significant the more parsimonious model was preferable. In addition to likelihood ratio tests, Akaike's Information Criterion, or AIC, were also used to determine the best fitting model, where lower AIC indicated a better fitting model.[175]

Aim 2a: Do neighborhood social and economic environments, health and built environments, and education environments mediate the association between residential segregation and youth obesity?

To accomplish aim 2, this study utilized mediation analysis to determine if and to what extent neighborhood 1) social and economic environments, 2) health and built environments, and 3) education environments mediate the association between residential segregation and youth obesity. Mediation is the process by which a third variable serves as the pathway that transmits the association between the independent and dependent variables of interest.[180] This variable can fully, or partially, explain the association the focal relationship. Meditation models can include multiple mediators that act in parallel or are sequentially dependent upon each other.[181] Originating in Social Psychology research, using the method proposed by Baron and Kenny in 1986, mediation analysis experienced significant growth in public health and other fields of health research in the subsequent decades.[181]

The neighborhood-level characteristics that were utilized as mediators are the aforementioned COI environment z-scores: social and economic environment z-score, health and built environment z-score, and education z-score. The predictor of interest was residential segregation (Black and Hispanic residential segregation LQs) and the outcome of interest was youth BMI (z-score). Given that aim 1 identified differential impacts of segregation at the neighborhood level for both Black and Hispanic residential segregation by youth race/ethnicity, the model-building and mediation testing process was completed, separately, for four different combinations of segregation and youth race/ethnicity. The four combinations were as follows: Black residential segregation among Black youth compared to White youth, Black segregation among Hispanic youth compared to White youth, Hispanic segregation among Black youth compared to White youth, and Hispanic segregation among Hispanic youth compared to White youth. Final covariates included in each of the mediation analyses were different for Black and Hispanic youth, where race/ethnicity (Black versus White, Hispanic versus White) and grade (2^{nd}=0, 5^{th}=1, 8^{th}=2, 9^{th}=3) were significant for models including Black and Hispanic youth, but biological sex (male=0, female=1) was only significant for models including Black youth.

Preliminary study analyses indicated that approximately 4% of the variation in youth BMI z-score was attributable to variation across neighborhood environments based on calculation of the ICC. After the inclusion of individual and neighborhood level predictors, the unexplained variation by neighborhood was close to zero. Therefore, single level models were utilized for all analyses, which allowed for the use of the PROCESS macro, developed by Hayes.[180] PROCESS macro is a modeling tool that can handle complex mediation models, involving simultaneous inclusion of multiple mediators that produces estimates of direct and indirect effects for the predictor of interest along with bootstrapping confidence intervals.

Prior to completing the mediation analyses, descriptive statistics were obtained for each of the study variables. Bivariate analyses were then used to assess the correlations between the study predictors, outcome, mediating variables, and individual controls. Additionally, the data were examined for any major violations of assumptions for Ordinary Least Squares (OLS) regression. All analyses were conducted using SAS version 9.4.[177]

PROCESS macro was then used to examine the effects of the potential mediators, using parallel a mediation model. In parallel mediation analysis, the predictors effects on the dependent variable are modeled through two or more mediators, where the mediators do not causally influence each other.[180] While the inclusion of multiple mediators increases the complexity of interpreting mediation analyses, mediating variables are rarely completely uncorrelated with each. Therefore, including multiple mediators can reduce the risk of confounding or epiphenomenal influences that may be present with a single mediator. It does, however, come with the risk of increased sampling variance and reduced power for tests of indirect tests. Figure 3.5 outlines the conceptual model of the mediating pathways that was used in this study .

PROCESS macro uses a set of OLS regression equations (shown in formulas 20 through 24 without controls) to calculate the total effect of X (residential segregation) on Y (BMI z-score), the direct effect of X on Y, independent of the mediators, and the indirect effect of on Y transmitted through M (neighborhood environment z-scores).

Effect of X on Y: $Y = \beta_0 + \beta_c X + e_{y1}$ (20)

Effect of X on Y, controlling M_1, M_2, M_3: $Y = \beta_0 + \beta_{c'} X + \beta_{b1} M_1 + \beta_{b2} M_2 + \beta_{b3} M_3 + e_{y2}$ (21)

Effect of X on M_1: $M_1 = \beta_0 + \beta_{a1} X_1 + e$ (22)

Effect of X on M_2: $M_2 = \beta_0 + \beta_{a2} X_2 + e_2$ (23)

Effect of X on M_3: $M_3 = \beta_0 + \beta_{a3} X_3 + e_3$ (24)

The coefficients for the predictors ($\beta_{a1,}$ $\beta_{a2,}$ β_{a3}) and the coefficients for the potential mediators ($\beta_{b1,}$ $\beta_{b2,}$ β_{b3}) are then used to estimate the indirect effects of the potential mediators on the association between the predictor and the outcome of interest, where $\beta_{a1}\,\beta_{b1}$, $\beta_{a2}\,\beta_{b2}$, and $\beta_{a3}\,\beta_{b3}$ are the partial indirect effects of M_1, M_2, and M_3, respectively.[180] The products of the coefficients are then summed to obtain the total mediating effect. To test the significance of the direct and indirect mediating effects, lower and upper confidence limits are then calculated using the bootstrap resampling method (n=5000), where significant effects are those that do not contain zero in the confidence interval. Bootstrapping is a non-parametric approach that is preferred over the normal theory approach (also known as the Sobel test), as it makes so assumption about the shape of the sampling distribution of the coefficients, is more robust to error, and yields more accurate confidence interval estimates.[180]

3.6 PROTECTION OF HUMAN SUBJECTS

Risks to Subjects/Participants

The risks of participation in this study were minimal. The only known risk to participants was breach of confidentiality of data. Specifically, the greatest risk of youth identification was through home address; however, upon initial receipt of data from the school district, the first step was to geocode home address using ArcGIS and delete home address from the database to protect youth anonymity. Additionally, study participants were identified by a unique identifier provided by the school district, not by name or any other personally identifiable information.

Adequacy of Protection against Risks

To minimize risk of breach of anonymity, numerous procedures were enacted to protect data confidentiality. The study researcher followed data storage and safety guidelines outlined by the school district, which are detailed in the data and safety monitoring section below. Additionally, the geo-coding and removal of data was conducted by one of the primary study investigators. Once addresses were recoded and removed from the dataset, no personally identifiable information was included in the final study database and individual youth were only identified by a unique numeric ID assigned by the district.

Potential Benefits to the Subjects and Others

While this study did not pose any potential benefits to the youth whose data are included in the study sample, this research was applied in nature and is designed to benefit the community from which the data are sourced. The goal is to aid a local healthy eating and active living coalition in identifying contextual PSE determinants of youth obesity and corresponding PSE levers to reduce rates of youth obesity and its associated disparities, by "making the healthy choice the easy choice." Additionally, this research has potential to inform researchers and policy makers in addressing the structural and systemic barriers that produce inequities in obesity and chronic disease by race/ethnicity and income.

Data Safety and Monitoring

Once address was geocoded and removed from the study database, the final study database did not include any personally identifiable information. During the duration of the study, the original raw files that contain address were kept on an external, password protected hard drive that was stored in a locked file, behind a locked door. These files were only accessible to the study PI and CO-Is. Once final data analyses are complete, the original, raw data files will be destroyed and only the database stripped of identifiers will be retained by the study team. Student research assistants only had access to files that were stripped of identifiers, including address and date of birth.

In accordance with the school district's recommendations, data were not stored on any mobile device. The de-identified study database was stored on a secured university network drive that was only be accessible to the PI, Co-Is, and student research assistants. This database can only be accessed when hard wired into the university network or through VPN access. No persons other than PI, CO-Is and student research assistants were permitted access to the contents of the data files. The data are encrypted and password protected with the following minimum requirements: AES, 256-bit, strong password (min 8 characters, no dictionary word. Needs to be a mixture of upper/lower case, numbers, and special characters). Further, the password will not be communicated in email.

Documentation of IRB Approval and CITI training

This study received official IRB approval through a letter from Furman University's Institutional Review Board. The study researcher has completed the Social and Behavioral Research CITI training module for the protection of human subjects. The researcher's CITI training number is 21479594.

Table 3.1 Summary of study measures, data sources, and operational definitions

Level	Variable	Data Source	Operational Definition
Individual Youth	Youth Weight Status	Youth height and weight data, collected twice annually as part of Fitnessgram	Continuous, BMI z-score created using CDC youth BMI percentiles
	Demographics (gender, race and ethnicity, grade)	Data verified by parent/guardians once annually in Powerschools	Gender (male=0, female=1), Race/ethnicity (White=0, Black=1, Hispanic=2), Grade (2^{nd}=0, 5^{th}=1, 8^{th}=2, 9^{th}=3)
School Level	School Segregation	Publicly available data from the State Department of Education website	Continuous, Location Quotient, proportion of one racial or ethnic group within a school divided by proportion of that group in the broader school district, calculated for Black and Hispanic separately
	School Poverty	Publicly available data from the State Department of Education website	Continuous, School Poverty Index as calculated by a composite index of students attending a school who receive free lunch, Medicaid, are in foster care, or who are homeless
Neighborhood	Neighborhood of Residence	Data verified by parent/guardians once annually in Powerschools	Geocoded using Arc GIS to the census tract level using youth address
	Neighborhood Residential Segregation	Census Bureau Data from American Community Survey	Continuous, Location Quotient, proportion of one racial or ethnic group within a census tract divided by proportion of that group in the county district, calculated for Black and Hispanic separately
	Social and Economic Environment	COI 2.0 social and economic domain z-score	Continuous, z-score
	Health and Built Environment	COI 2.0 health and environment domain z-score	Continuous, z-score
	Education Environment	COI 2.0 education environment z-score	Continuous, z-score
	Educational Attainment	Census Bureau Data from American Community Survey	Percent of neighborhood residents at the census tract level who have a high school diploma or less; Continuous

Table 3.2 Childhood Opportunity Index 2.0 component indicators

Domain	Component Indicator	Operational Definition
Social and Economic	***Economic opportunties***	
	Employment rate	Percent adults ages 25-54 who are employed
	Commute duration	Percent workers commuting more than one hour one way
	Economic and social resources	
	Poverty rate	Percent individuals living in households with incomes below 100% of the FPL
	Public assistance rate	Percent households receiving cash public assistance or Food Stamps/SNAP
	Homeownership rate	Percent owner-occupied housing units
	High-skill employment	Percent individuals 16+ in management, business, financial, computer, engineering, science, education, legal, community service, health, health tech, arts and media occupations
	Median household income	Median income of all households
	Single-headed households	Percent family households that are single parent headed
Health and Built Environment	***Healthy Environments***	
	Access to healthy food	Percent households without a car located further than a half-mile from the nearest super-market
	Access to green space	Percent impenetrable surface areas such as rooftops, roads, or parking lots
	Walkability	EPA Walkability Inex
	Housing vacancy rate	Percent housing units that are vacant
	Toxic Exposures	
	Hazardous waste dump sites	Average number of Superfund sites within a 2-mie radius
	Industrial pollutants	Index of tox chemicals released by industrial facilities
	Airborne microparticles	Mean estimated microparticle concentration
	Ozone concentration	Mean estimated 8-hour average ozone concentration
	Extreme heat exposure	Summer days with maximum temperature above 90F
	Health Resources	
	Health insurance coverage	Percent individuals 0-64 with health insurance coverage
	Early Childhood Education	
	ECE Centers	Number of ECE centers within a 5-mile radius
	High Quality ECE Centers	Number of NAEYC accredited centers within a 5-mile radius
	ECE enrollment	Percent 3- and 4-year-olds enrolled in nursery school, preschool, or kindergarten
	Elementary Education	Percent 3rd graders proficient on standardized reading tests, converted to NAEP scale

Education Environment	Third grade reading proficiency	Percent 3rd graders proficient on standardized math tests, converted to NAEP scale
	Third grade math proficiency	
	Secondary and postsecondary education	
	High school graduation rate	Percent ninth graders graduating from high school on time
	Advanced Placement course enrollment	Ratio of students enrolled in at least one AP course to the number of 11th and 12th graders
	College enrollment in nearby institutions	Percent 18–24-year-olds enrolled in college within 25-mile radius
	Educational and social resources	
	School Poverty	Percent students in elementary schools eligible for free or reduced-price lunches, reversed
	Teacher experience	Percent teachers in their first and second year, reversed
	Adult Educational Attainment	Percent adults age 25 and over with a college degree or higher

Table 3.3 Summary of the model building process

Model	Predictor Variables and Covariates
Model 1 (Null)	None
Model 2 (Control)	Grade, Race/ethnicity, biological sex
Model 3 (School)	Grade, Race/ethnicity, School Poverty, Black School Segregation, Hispanic School Segregation
Model 4 (Neighborhood)	Grade, Race/ethnicity, Black Residential Segregation, Hispanic Residential Segregation, Educational Attainment
Model 5 (Combined School and Neighborhood Model)	Grade, Race/ethnicity, School Poverty Index, Black School Segregation, Hispanic School Segregation, Black Residential Segregation, Hispanic Residential Segregation, Neighborhood Educational Attainment
Model 6 (Combined Model with interaction between Hispanic school seg. and youth race/ethnicity)	School Poverty Index, Black School Segregation, Hispanic School Segregation, Black Residential Segregation, Hispanic Residential Segregation Educational Attainment, Hispanic Residential segregation*Black, Hispanic Residential segregation*Hispanic
Model 7 (Combined Model with interaction between Hispanic residential seg. and youth race/ethnicity)	School Poverty Index, Black School Segregation, Hispanic School Segregation, Black Residential Segregation, Hispanic Residential Segregation, Educational Attainment, Hispanic Residential segregation*Black, Hispanic Residential segregation*Hispanic

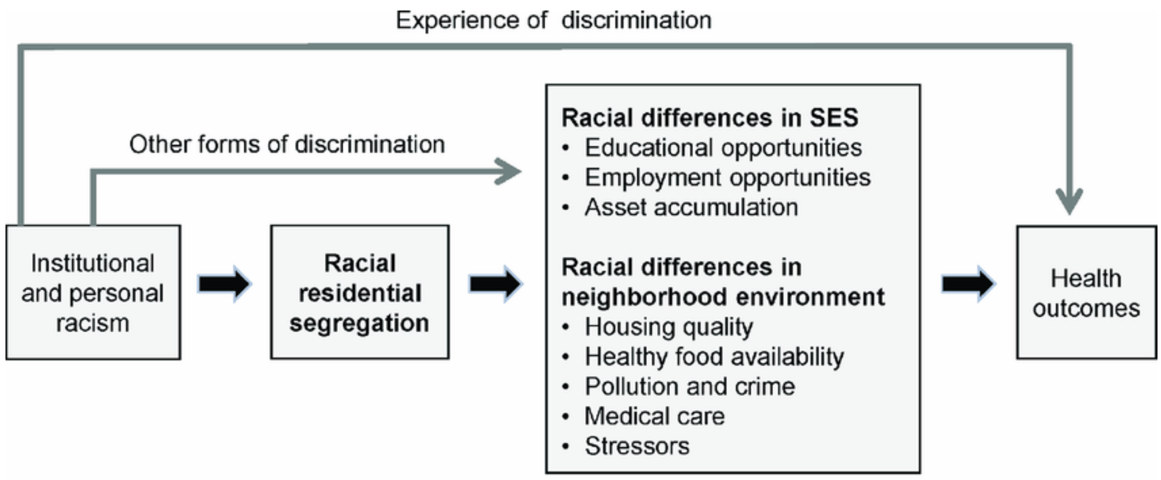

Figure 3.1 Racism, racial residential segregation, and health

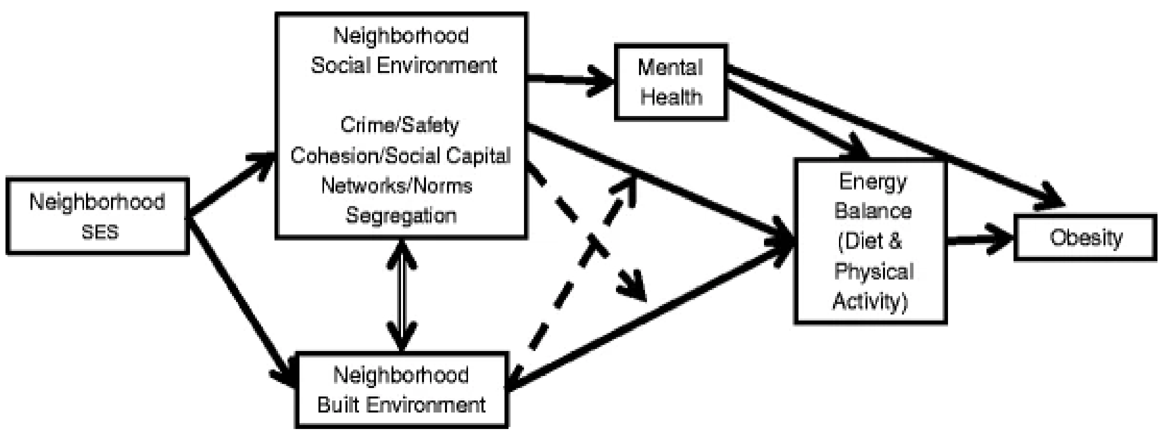

Figure 3.2 Conceptual model of the relationship between neighborhood social and physical environments with obesity

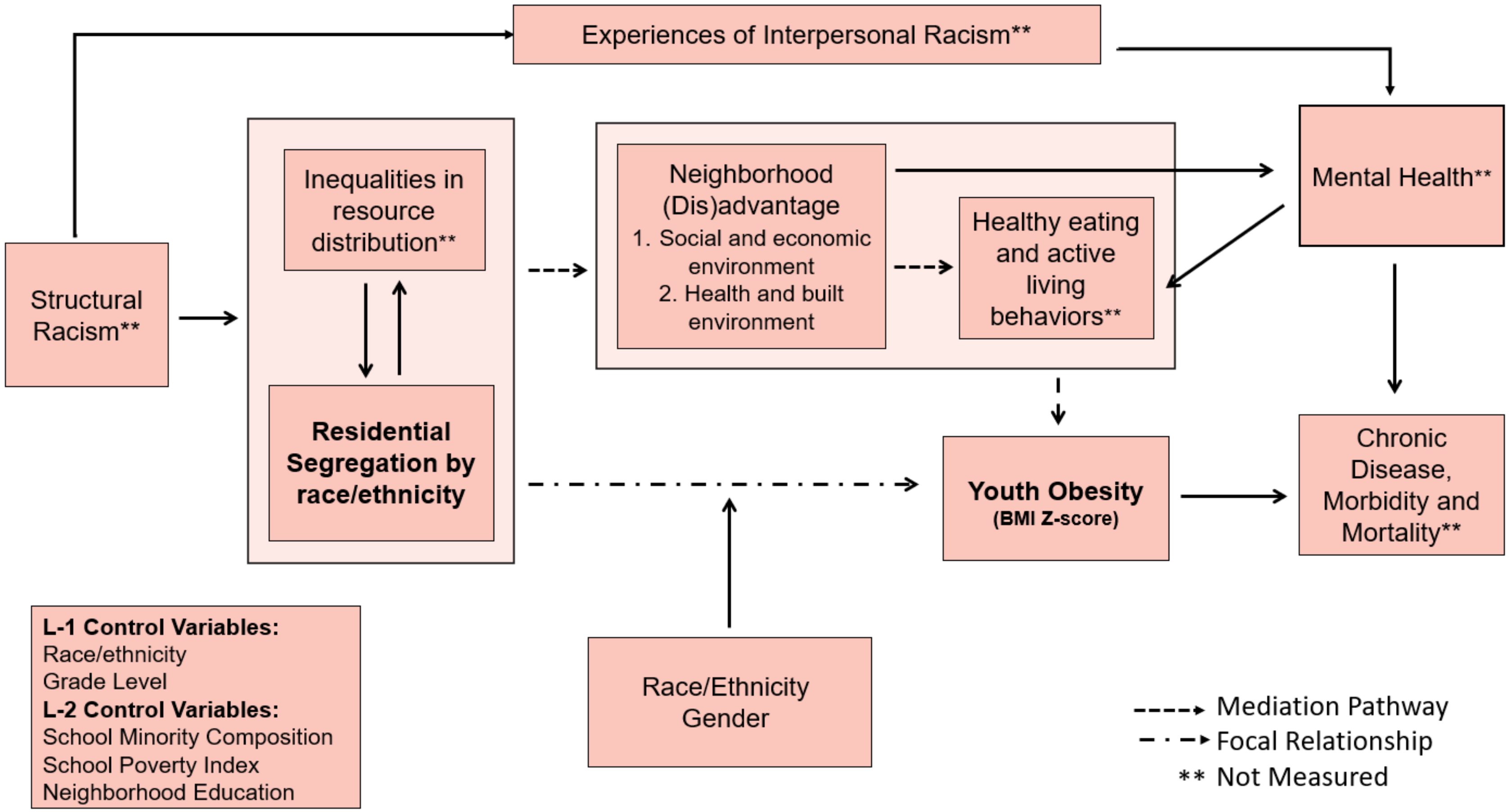

Figure 3.3 Adapted conceptual model of how neighborhood residential segregation affects youth obesity

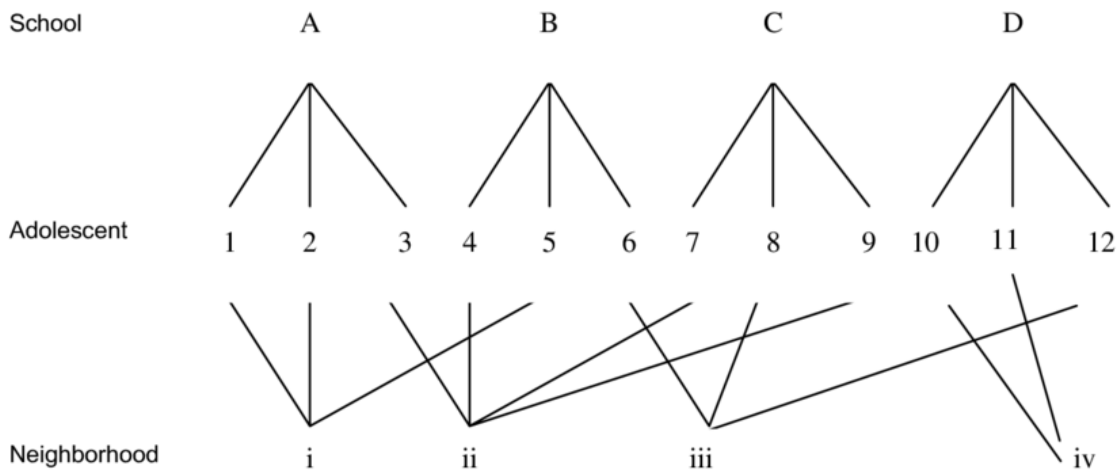

Figure 3.4 Schematic diagram of a cross-classified multilevel model showing nonhierarchical membership of neighborhoods and schools

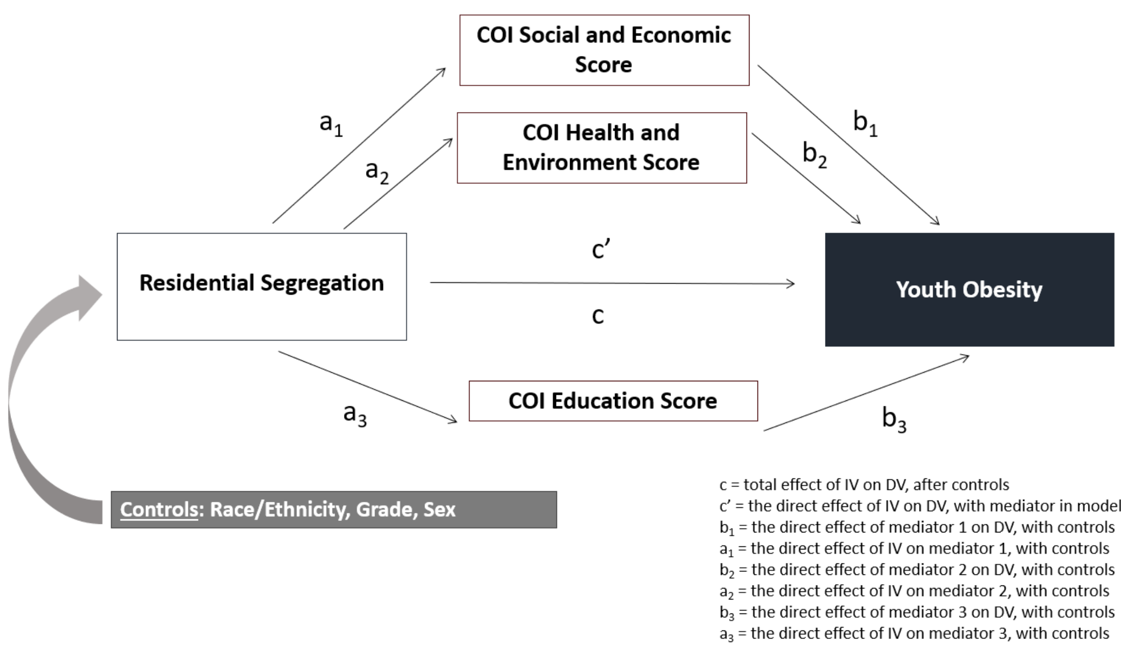

Figure 3.5 Mediation pathway of neighborhood social/economic, health/built, and education environments on the association between residential segregation and youth obesity

CHAPTER FOUR: MANUSCRIPT ONE

EXAMINING THE ASSOCIATIONS BETWEEN SCHOOL AND NEIGHBORHOOD RESIDENTIAL SEGREGATION WITH YOUTH OBESITY[1]

[1] Fair, M. L., Kaczynski, A. T., Monroe, C., Powers, A. R., Rudisill, A. To be submitted to *Journal of Ethnicity and Health*.

Abstract

Youth obesity remains a significant public health problem, with disparities by race/ethnicity that contribute to health inequities across the life course. Obesity is a complex system involving multiple levels and settings. Schools are important settings for the prevention of youth obesity and characteristics such as poverty and minority composition have been linked to youth obesity risk. Less attention has been paid to the role of school segregation in health disparities. Neighborhoods are also settings where health inequities emerge and are sustained. Residential segregation is one important neighborhood feature that influences social and built environments that impact youth obesity. Using individual youth data from a local school district (n=11,364) and census tract ACS survey data, a series of cross-classified, multi-level linear models were used to explore associations between school and neighborhood residential segregation with youth obesity. The moderating effects of segregation on the association between race/ethnicity and youth BMI z-score were examined. Results indicated Black (b=-0.10, $p<.05$) and Hispanic (b=-0.11, $p<.05$) school segregation were inversely associated with youth obesity, and individual race/ethnicity was a potential moderator of the association between Hispanic school segregation and youth BMI z-score. Black (b=0.04 $p<.05$) and Hispanic (b=0.06, $p<.001$) residential segregation were positively associated with youth obesity, and individual race/ethnicity was a potential moderator in the association between Hispanic residential segregation and youth obesity but did not improve model fit. Given the dearth of literature on the relationship between segregation and youth obesity, future research should continue to explore this area and consider the system level determinants that shape school and neighborhood opportunities and health behaviors.

Introduction

Youth obesity remains a significant public health problem in the United Sates (US), with significant disparities by income, education, and race/ethnicity that contribute to subsequent inequities in chronic disease and morbidity for certain groups.[1-4] While rates of youth obesity have shown signs of plateauing over the last decade, preliminary evidence indicates that the Covid-19 pandemic has reversed these promising trends.[182] In a nationwide study, Springer et al. found that youth obesity prevalence has increased over the last two years and the trajectory of weight gain has accelerated, especially among youth who had overweight or obesity at the beginning of the pandemic.[183] These same youth are also more likely to identify as Black or Hispanic, therefore, pandemic driven increases in youth obesity incidence and prevalence has exacerbated youth obesity disparities.

Youth obesity contributes to significant morbidity and mortality across the life course.[1,3] In the short term, youth who have obesity are more likely to experience musculoskeletal issues, asthma, glucose intolerance, dyslipidemia, and mental health issues, including depression and anxiety.[5,79] Youth who are obese are more likely to experience obesity as adults, at increased risk for heart disease, stroke, lung disease, some cancers, and most recently, more likely to die of complications related to Covid-19.[3,81,182]

Obesity is the result of a complex system involving multiple levels of influence and multiple settings where children and their families eat, play, learn, work, and play.[12] Therefore, understanding and developing interventions to reduce population level youth obesity prevalence requires simultaneous exploration of the settings that influence youth obesity, including schools and neighborhoods.[25,101,149] Schools are important settings for the prevention and reduction of youth obesity because of their important influence on food and physical activity opportunities and behaviors.[76,183] School-level poverty and minority composition have been linked to numerous health outcomes, including youth obesity, even after controlling for individual youth characteristics. School racial/ethnic segregation, while interrelated, is distinct from minority composition and has been less studied.[124,184]

An equally important context and rapidly growing area of research in which health behaviors, outcomes, and inequities emerge is neighborhoods.[22,23,25,110] Previous research has linked neighborhood socioeconomic and built environment characteristics to youth obesity risk, including features such as access to parks and greenspace, neighborhood walkability, fast-food density, crime, neighborhood socioeconomic status and educational attainment.[23,26,63] One neighborhood feature that has been linked to numerous health outcomes and inequities is residential segregation.[21,185,186] Williams and Collins proposed residential segregation as a fundamental cause of health inequities through the primary pathways of lack of investment and resources in communities of color that lead to disadvantages in opportunities for education, income, and health.[34]

There is a growing body of evidence on the relationship between residential segregation and obesity; however, there is inconsistency in the research findings by demographic characteristics including race/ethnicity and gender.[40,54,55,144] Positive associations between living in a residentially segregated neighborhood and obesity risk were most consistent among Black women, followed by Back men.[40] Among Hispanic and White men and women, studies have found a variety of positive, negative, and null associations, which researchers have attributed to methodological issues such as measurement and analysis as well as the potential protective factor living in a segregated neighborhood may have such as allocation of resources or protection against acculturation.[40,55]

The evidence exploring segregation and youth obesity remains very limited. Only one study to our knowledge, conducted by Ryabov, was identified that has explored the association between residential segregation and youth obesity as the focal relationship.[56] The author found that after controlling for individual and family-level covariates, residential segregation explained between 5% and 20% of the difference in obesity and chronic disease risk for Black and Hispanic children, compared to White youth, and that children living in more residentially segregated communities had greater odds of having obesity compared to children who lived in non-residentially segregated communities, regardless of their individual race or ethnicity.[56] Another 2016 study, examining individual, school, and county level effects on youth obesity risk, including school minority composition and residential segregation, found that schools with a higher proportion of minority students had higher rates of youth obesity, but the effect was not statistically significant after controlling for school poverty and residential segregation.[57]

Despite the recognition that youth obesity is a complex system and calls to examine the influences of multiple settings, including schools and neighborhoods in concert, previous research has commonly studied these settings in siloed contexts.[23,162] One reason for this isolated approach in studying environments that influence youth obesity risk, is the limitations of traditional statistical methods, such as single level regression.[93] Multi-level regression allows researchers to consider youth who are nested within levels, such as students nested within classrooms that are nested within schools.[93,175] Multi-level models can be extended to cross-classified multi-level models (CCMM), which allow for non-hierarchical data structures such as youth who are cross-classified across neighborhoods and schools.

Study Purpose

Using CCMMs, the purpose of this study was to 1) Determine the relative influences of school and neighborhood environments on youth obesity, and 2) Examine the associations between school and neighborhood residential segregation and youth obesity, after controlling for associated individual, school, and neighborhood characteristics.

Study Methods

Study Setting and Sample

This study occurred in a large southeastern county in the US, which is home to a unified school district, which is the 44th largest in the nation and covers more than 800 square miles and contains 111 census tracts. The district contains 51 elementary schools, 20 middle schools, 14 high schools, and 16 special schools or centers that serve more than 76,000 students.[187] The county is predominantly non-Hispanic White (76.3%), followed by Black (18.4%) and Hispanic (9.5%) residents. Approximately 87.3% of residents over the age of 25 have their high school diploma and 33.3% have a bachelor's degree or higher. The median household income is $60,351 and approximately 10.6% of individuals live in poverty.[60]

Data Collection

The youth level data used in this study, including height, weight, and demographics, were part of routine data collection conducted by the school district during the 2016-2017 academic year. Specifically, data were obtained from the school district's *FitnessGram* and *PowerSchools* databases as part of a data sharing agreement with a local university. Youth height and weight data were collected and entered by trained physical education teachers, twice annually, in the *FitnessGram* database.[64] Demographic data were submitted by parents or guardians annually and updated throughout the academic year via the *PowerSchools* database online platform.[167] School-level data, including minority composition and poverty for the 2016-2017 academic year were obtained from data that are publicly available on the State Department of Education and the local school district websites.

Neighborhood-level (census tract) data were obtained from the US Census Bureau American Community Survey (ACS) 2014-2018 five-year estimates via the National Historical Geographic Information Service (NHGIS).[168] The ACS measures included racial and ethnic composition and educational attainment at the census tract and county level. As is described further in the measures section below, racial/ethnic composition were used to calculate residential segregation. Student address, from the school district dataset, was geo-coded to the census tract level and used to join neighborhood level measures and youth level data using a unique identifier provided by the school district.

Inclusion criteria at the individual youth level included having complete height, weight, and demographic data for the spring 2017 data collection time point. Exclusion criteria included youth who identified as belonging to a racial or ethnic category other than Black, Hispanic, or White, as the race/ethnicity categories within the "Other category" were small and heterogeneous.

Measures

Level-1 Outcome

The continuous outcome of interest in this study was youth BMI z-score, which was computed from youth height, weight, age in months, and biological sex data using protocols developed by the CDC.[169] CDC BMI z-scores are recommended over BMI percentiles as they incorporate a smoothing and transformation process that makes them more comparable across youth of different ages (months) and sex.[188]

Level-2 Predictors

The primary predictor of interest was neighborhood-level (census tract) residential segregation by race/ethnicity, as measured by the Location Quotient (LQ). The LQ, a measure of separateness, is a localized measure of segregation that is calculated by taking the quotient of the proportion of one group at the local level (census tract) divided by the proportion of that group at the broader area (county).[21,172] If the proportion of one group in a local areal unit is the same as that for the overall area, the area overall LQ will equal zero. If the local proportion is greater than regional proportion LQ will be greater than 1, and if the proportion in a local area is less than the overall area, the LQ will be less than one. The LQ has a theoretical range of zero to infinity, but less than .85 or greater than 1.2 represent approximately 1 standard deviation above or below an LQ of one and are used as cutoffs for under and overrepresentation, respectively.[172] The LQ has several advantages, including its ease of calculation and interpretation.[172,173] Neighborhood residential segregation LQs were calculated for Black and Hispanic residential segregation, separately. They were then grand mean centered to improve interpretability of the parameter estimates and treated as continuous measures in study analyses.

A secondary predictor of interest was school-level segregation, which was also calculated using the LQ, separately, for Black and Hispanic school segregation. Specifically, school segregation was calculated by taking the quotient of the proportion of one group within a school divided by the proportion of that group in the school district. The school segregation LQs were also grand mean centered to increase interpretability in study analyses and treated as continuous variables.

Study Covariates

Level-1 covariates. The individual covariates obtained from the school district that were used in this study included biological sex (male=0, female=1), grade (0=2nd, 1=5th, 2=8th, 3=9th), and race/ethnicity (White=0, Black=1, Hispanic=2).

Level-2 school covariate. School covariates included an index of school poverty. The School Poverty Index (SPI) is calculated, annually, by the state department of education and is the proportion of students who are 1) living in poverty as defined by the federal poverty guidelines; 2) students participating in TANF, Medicaid, or SNAP; or 3) students who are identified as a foster child or homeless.[62] The SPI was treated as a continuous measure, with scores ranging from zero to 100, where zero represents the complete absence of students who live in poverty and 100 indicates that all students live in poverty. The SPI was grand mean centered and treated as a continuous measure.

Level-2 neighborhood covariate. The neighborhood covariate included in this study was educational attainment. Neighborhood educational attainment was calculated using the percentage of individuals who have a high school diploma or less within the census tract. The educational attainment variable was grand mean centered and treated as a continuous measure.

Analytic Approach/Model Building Process

Descriptive statistics were obtained for the outcome variable of interest, youth BMI z-score, and each of the level-1 and level-2 predictors, including the distribution of the level-1 characteristics across level-2 units. Bivariate analyses were used to assess the correlation between the main outcome variable, BMI z-score, and the main outcome of interest, residential segregation, along other relevant individual, school, and neighborhood characteristics using individual students as the unit of analyses. All analyses were conducted in SAS v9.4.[177]

A series of CCMMs were used to examine the associations between residential and school segregation with youth BMI z-score, while controlling for individual, school, and neighborhood covariates. CCMMs are an extension of hierarchical, multi-level models (MLM) that account for non-hierarchal, cross-classified data structures, such as students across schools and neighborhoods.[93] The primary advantage off CCMMs over MLMs is that they allow for simultaneous inclusion of non-hierarchical settings such as neighborhoods and schools.

This analysis included two levels, with individual youth as the Level-1 units who were cross-classified between two Level-2 units, neighborhoods and schools. Level-1 predictors included the main outcome of interest, youth BMI *z*-score, and youth demographic characteristics. Youth demographic characteristics were treated as fixed effects and included biological sex, grade, and race/ethnicity.

The Level-2 units in this analysis were school and neighborhood characteristics, which were treated as random effects. Treating school of attendance and neighborhood as random effects allowed the influence of these two environments to vary, rather than remaining equal, across all schools and all neighborhoods.[93,162] School-level characteristics included school-level poverty, as measured by the SPI, and school segregation, as measured by the Black and Hispanic school segregation LQs. The primary neighborhood-level characteristic of interest was residential segregation as measured by the Black and Hispanic residential segregation LQs. One additional neighborhood characteristic included was educational attainment.

A series of seven CCMM models were used to examine the association between residential segregation and youth BMI z-score. The model building process is summarized in Table 4.1. Using PROC MIXED with maximum likelihood estimation and Satterthwaite degrees of freedom, the first model estimated was the unconditional model, which includes no Level-1 or Level-2 predictors. Model 2 included the addition of the level-1 covariates (grade, race/ethnicity, and biological sex). Model 3 included the addition of the Level-2 school predictors (Black and Hispanic school segregation) and the covariate, school poverty. Model 4 included the addition of the Level-2 neighborhood predictors (Black and Hispanic residential segregation) and covariates (educational attainment), in addition to the individual covariates. Model 5 all previous individual, school, and neighborhood variables included in both Model 4 and 5 into one model. Model 6 included the same variables as Model 5 and the addition of an interaction term between Hispanic school segregation and youth race/ethnicity. Model 7 included the same variables as model 5with the addition of an interaction term between Hispanic residential segregation and youth race/ethnicity.

Assessing model fit

To determine the variability in youth BMI z-score that was attributable to between-school and between-neighborhood effects, three different Intraclass Correlation Coefficients (ICC) were calculated using the unconditional model: schools only, neighborhood only, and schools and neighborhoods.

Pseudo R^2 and -2 Log Likelihood were used to determine model fit during the model building process. Pseudo R^2 was calculated for each model and then a series of comparisons were made for each of the nested models to determine if the proportion of variance accounted for by the parameters added to the complex model was justified. Specifically, model comparisons were made between Models 1 and 2, Models 2 and 5, Models 3 and 5, Models 4 and 5, Models 6 and 5, and Models 7 and 5. Finally, to determine if the change in pseudo R^2 was statistically significant, likelihood ratio tests using the chi-squared statistic were used to assess the change in -2 Log Likelihood for each of the model comparisons.

Results

Sample Characteristics

Sample characteristics of individual youth, schools, and neighborhoods are presented in Table 4.2. The final analytic sample included 11,364 youth from 80 schools and 111 census tracts. There was a range of 9 to 317 students per school and an average of 142 students per school (SD=66.68). There was a range of 2 to 368 students per neighborhood and an average of 108 students per neighborhood (SD=68.19). There were slightly more male youth (51.5) compared to female youth (48.4%). White students were the largest racial/ethnic group (61.3%), followed by Black (23.3%) and Hispanic (15.4%) youth. There was a similar amount of youth in second (36.6%) and fifth (39.2%) grade and a much smaller, but similar, amount of eighth (13.2%) and ninth (11.03%) grade youth. The average youth BMI z-score was 0.48 (SD=1.16), the average youth BMI percentile was 62.99 (SD=30.20), and most youth were in the normal weight category (66.9%), followed by overweight (17.8%), and obese (15.3%).

The average Black school segregation LQ was 0.97 (SD=1.00) and 0.99 (SD=0.91) for Hispanic school segregation, indicating the average school was not residentially segregated. The lowest Black school segregation LQ was 0.06 and the maximum was 2.72. The lowest Hispanic school segregation LQ was zero, indicating no Hispanic students attend the school, and the highest was 4.50, which equated to the Hispanic population within that census tract being 4.5 times higher than the average across all census tracts within the county. At the school level, the average SPI score was 53.33 (SD=20.41), with a minimum of 16.23 and a maximum of 91.69.

Similarly, at the neighborhood level, the average neighborhood was not segregated for both the Black (M=1.04, SD=0.89) and Hispanic (M=1.03, SD=0.91) residential segregation LQs. However, the maximum Black residential segregation LQ (4.69) was much higher compared to the maximum for the Black school segregation LQ. The maximum Hispanic residential segregation LQ was 4.32 and the lowest for both Black and Hispanic LQs was zero. The average percentage of residents in each census tract with a high school diploma or less was 24.7% (SD=9.18), while the maximum was 46.1% and the minimum was 5.6%

Bivariate Associations

Pearson correlation coefficients were obtained for all bivariate associations between the study independent and dependent variables (Table 4.3). The strongest bivariate associations were between combinations of school segregation, residential segregation, and school poverty, with the highest value for the correlations between Hispanic school segregation and poverty (r=0.71), Hispanic school and neighborhood segregation (r=0.68), Black school segregation and poverty (r=0.62), and Black school and neighborhood segregation (r=0.62). At the individual youth level, there was a small, positive correlation between youth BMI z-score and belonging to the Black (r=0.11) or Hispanic (r=0.12) race/ethnicity category. The association between belonging to the White category and BMI z-score was negative (r=-0.18). The correlations between BMI z-score and all school and neighborhood level predictors and covariates were small, but statistically significant, ranging from r=0.08 to r=0.16. The highest correlations of any variable with BMI z-score were between neighborhood educational attainment (r=0.16), followed by school poverty (r=0.14), Black residential segregation (0.11), Hispanic residential segregation (r=0.10), Hispanic school segregation (r=0.9), and Black school segregation (r=0.08).

Cross-classified Multilevel Regression

Results from the cross-classified, multi-level modeling process are presented in Table 4.4. The ICC calculated from the unconditional model indicated that 3.6% of the variability in youth BMI z-scores could be attributed to schools, 1.9% attributed to neighborhoods, and 5.5% attributed to schools and neighborhoods combined. The final and best fitting model was Model 6 which was the combined school and neighborhoods model in addition to an interaction between Hispanic school segregation and youth race/ethnicity.

Results from Model 2 demonstrated that youth race/ethnicity and grade were associated with youth BMI z-score and remained significant throughout the model building process. Specifically, being Black (*b*=0.369, p<.0001) or Hispanic (*b*=0.432, p<.0001) was associated with a higher BMI z-score, compared to White youth. By grade level, being in fifth (*b*=0.207, p<.0001), eighth (*b*=0.182, p<.001), or ninth (*b=0.152*, p<.05) grade was associated with a higher BMI z-score, compared to youth in second grade. Biological sex was not significant and did not improve the model fit in Model 2; therefore, it was dropped from Model 2 and all subsequent models.

In Model 3, which included only the school and individual measures, after controlling for individual characteristics, each of the school level predictors and covariates was statistically significant, with Black (*b*=-0.105, p<.05) and Hispanic (*b*=-0.092, p<.05) school segregation inversely associated with youth BMI z-score and school poverty (*b*=0.010, p<.0001) positively associated with youth BMI z-score. The school predictors and covariates were significant throughout the model building process, with the exception of Hispanic school segregation, which was not significant in the best fitting model (Model 6) that included an interaction term between Hispanic school segregation and youth race/ethnicity.

In Model 4, which included only the neighborhood and individual measures, after controlling for individual characteristics, there were significant and positive associations between both Hispanic residential segregation (*b*=0.050, p<.05) and educational attainment (*b*=0.008, p<.0001) with youth BMI z-score. The association between Black residential segregation and youth BMI z-score was not significant. While not statistically significant, Black segregation was retained in the subsequent models because it was important in addressing the study research questions. Neighborhood poverty was removed from the model and not included in further models as it was not statistically significant and did not improve model fit. Additionally, the Level-2 error variance in Model 4 attributed to the random intercept for neighborhoods was no longer significant, indicating the variables included in the model explained the variation in youth obesity at the neighborhood level.

In the combined schools and neighborhood model (Model 5), all of the previously included individual, school, and neighborhood predictors and covariates remained statistically significant. At the neighborhood level, after controlling for individual predictors and school predictors and covariates, there was a positive and statistically significant relationship between youth BMI z-score and Black residential segregation (*b*=0.037, p<.05), Hispanic residential segregation (*b*=0.057, p<.001), and proportion of neighborhood residents with a high school diploma or less (*b*=0.006, p<.001*).* Among school level covariates, Black (*b*=-0.102, p<.05*)* and Hispanic (*b*=-0.114, p<.05*)* school segregation were negatively associated with youth BMI z-score, while school poverty was positively associated with youth BMI z-score (*b*=0.007, p<.001*).*

Results from Model 6 indicate there was a statistically significant interaction between Hispanic school segregation and youth race/ethnicity (Black, *b*=-0.101 p<.0001; Hispanic *b*=-0.096, p<.001), net of all other study variables. This suggests that individual youth race/ethnicity may moderate the association between Hispanic school segregation and youth BMI z-score. As shown in Figure 4.1, overall, schools with the highest levels of Hispanic school segregation had the lowest youth BMI z-scores for youth of all race/ethnicities, net of all other study variables, and as Hispanic school segregation increased the magnitude of the decrease in BMI z-score varied by race/ethnicity. While Black youth had a slightly lower average BMI z-score than Hispanic youth, the average BMI z-score for both Black and Hispanic youth decreased at a similar rate as Hispanic school segregation increased. White youth had the lowest average BMI z-scores overall and decreased at a slower rate compared to Hispanic and Black youth as Hispanic school segregation increased, achieving similar average BMI z-scores as Black youth (and, to a lesser extent, Hispanic youth) at the highest levels of Hispanic school segregation.

Results from Model 7 indicated there was a statistically significant interaction between Hispanic residential segregation and youth race/ethnicity (Black, b=-0.127, $p<.0001$; Hispanic b=-0.097, $p<.001$), net of all other study variables. This suggests that individual race/ethnicity may moderate the association between Hispanic residential segregation and youth BMI z-score. As shown in Figure 4.2, neighborhoods with higher Hispanic residential segregation have higher youth BMI z-scores for all youth compared to neighborhoods with average or under-representation of the Hispanic population. Additionally, as Hispanic residential segregation increased, the rate of increase in youth BMI z-scores varied by youth race/ethnicity. White youth had the lowest BMI z-scores when Hispanics were the most underrepresented, but they also had the greatest rate of increase in BMI z-scores as Hispanic residential segregation increased. At the highest levels of Hispanic residential segregation, average BMI z-scores among White youth surpassed average BMI z-scores among Black youth and were similar to those of Hispanic youth. BMI z-scores among Black youth were higher, compared to those of

White youth, when Hispanics were underrepresented at the neighborhood level, but remained consistent as Hispanic residential segregation increased. Finally, BMI z-scores among Hispanic youth were the highest compared to White and Black youth at the lowest levels of Hispanic representation at the neighborhood level, but increased slightly as Hispanic residential segregation increased.

As shown in Table 4.5, model fit statistics, including change in pseudo R^2 and -2 Log Likelihood tests using the Chi-Square statistic among nested models, indicated that Model 6 that included an interaction term between Hispanic residential segregation and youth race/ethnicity was the best fitting model, when compared to Model 5. While yielding a statistically significant interaction term between Hispanic residential segregation and youth race/ethnicity and having lower -2 Log Likelihood and AIC values, the change in pseudo R^2 from Model 7 to 5 it was not statistically significant and therefore the more parsimonious model (Model 5) is the preferred model.

Discussion

Despite decades of research exploring the relationship between school and neighborhood influences on youth obesity risk, few studies have examined the associations between school and residential segregation by race/ethnicity and youth obesity.[56,57,59] Specifically, this study found that while the variability in youth BMI across schools and neighborhoods was small (5.5%), similar to previous youth obesity research, higher youth BMI z-scores were associated with being Black or Hispanic, increasing grade level, attending a school with higher poverty levels, and living in a neighborhood with lower educational attainment.[4,69] After controlling for individual, school, and neighborhood covariates, Black and Hispanic school segregation, as well as Black and

Hispanic residential segregation, were significantly associated with youth BMI z-score. These findings were supported by a 2018 study that found as county-level Black and Hispanic residential segregation increased, so did the odds of youth overweight or obesity.[56]

Black and Hispanic school segregation were negatively associated with youth obesity, but the association was stronger for Hispanic school segregation. The majority of previous research on the impacts of school segregation has focused on the intersection of school and economic segregation and how it limits access to resources and school quality, resulting in differential educational outcomes and economic wellbeing across the life course for youth of color.[21,41,189] For example, using more than 100 million test scores across 300 metropolitan areas, Reardon explored the association between racial segregation and academic achievement, finding that the greatest predictor of the academic achievement gap between predominantly White and Black schools was disparities in school-level poverty.[190]

This study's finding of Hispanic school segregation as a potential protective factor against youth obesity risk contrasts with much of the previous literature on school segregation; however, few studies have explored the impact of school segregation on health behaviors and health outcomes, when compared to those that have examined academic achievement and long-term educational attainment.[184] Piontak and Schulman investigated the effect of school poverty and segregation on youth obesity in a large nationwide sample. After controlling for Black and Hispanic residential segregation, they found that while youth in minority segregated schools were more likely to be obese, the relationship was not significant after controlling for school level poverty.[57] There are, however, two major differences between Piontak and Schulman’s study and the present study, including that in the former, racial composition, rather than a measure of segregation, was used as the predictor and Black and Hispanic school segregation were not examined separately.

In addition to exploring the association between school segregation and youth BMI z-score, this analysis reported a significant interaction term between Hispanic school segregation and individual youth race/ethnicity, providing preliminary evidence that individual race/ethnicity may moderate the relationship between Hispanic school segregation and youth obesity. After controlling for other study predictors and covariates, as Hispanic school segregation increased, youth BMI score decreased at a greater rate for Black and Hispanic youth. In combination with the existing research, detailed above, these findings suggest future research should explore the differential mechanisms by which school segregation by race/ethnicity may impact youth well-being and health. This should include both harmful pathways, such as limiting access to resources and opportunities for educational quality and or through supportive pathways, such as increased racial/ethnic peer interactions or positive cultural influences on school environments.[41,190-192]

While findings from this study or future directions of research are not intended to advocate for continued school segregation, from a policy and systems perspective, they do highlight that consideration should not only be given to increasing diversity of schools by changing the racial or ethnic composition of the school. Consideration should also be given to the mechanisms by which disparate educational or health outcomes emerge and can be addressed within schools, including system level factors and structural inequalities that result in differential investment in low income and predominantly minority schools. For example, using data from Black segregated schools in the southern US states, Frisvold and Golberstein found that improving school quality metrics such as student-teacher ratio, teacher wages, and length of school year, positively impacted several indicators of health later in life including self-rated health, smoking, obesity, and mortality.[124] This future area of study is further supported by the significant interaction term in this study between Hispanic school segregation and youth race/ethnicity, where, as BMI z-scores decreased for all youth as Hispanic school segregation increased and the fact that the magnitude of the decrease was greatest for Black and Hispanic youth.

In contrast to findings related to school segregation and youth obesity, Black and Hispanic residential segregation were significantly and positively associated with youth BMI z-score, with a stronger association with Hispanic residential segregation. Given that the literature on the relationship between youth obesity and residential segregation is extremely limited, with only two studies previously identified that examined this specific relationship and one study finding a positive association and the other no association, this study represents a significant advancement and demonstrates the need for additional research in the field.[56,57,59] Results from this study are also supported by the growing body of evidence on the relationship between residential segregation and adult obesity.[40,54,55,144] A number of previous studies have demonstrated strong evidence on the association between Black-White residential segregation and obesity risk among Black adults and among women, in particular.[55,144] Given that our study found no differences by biological sex, future research should consider how longitudinal impacts of residential segregation may vary by race/ethnicity, gender, and accumulation of exposure. For example, in a longitudinal study by Pool et al., higher than average exposure to high residential segregation over the study period had a 50% higher hazard of developing obesity.[51]

While this study found the strongest associations between Hispanic residential segregation and youth BMI z-score, the research exploring Hispanic-White residential segregation among Hispanic adults is more limited than Black-White segregation, with some studies indicating negative, null, or positive associations.[40,55] The Hispanic population is one of the fastest growing minority populations, and among youth, is the fastest growing population.[193] This population growth has not only occurred within cities and communities where Hispanic families have historically resided in the US, but included expansion of Hispanic populations into new areas across the US.[194] This population growth and expansion has also coincided with increased Hispanic-White residential segregation in recent decades. A longitudinal exploration of residential segregation from 1990 to 2020 found that while multi-group segregation and Black segregation decreased, segregation among Hispanic residents increased by 8%.[195]

Substantial neighborhood and youth obesity research has explored the relationship between socioeconomic and built environments and their potential to either inhibit or promote health lifestyles and youth obesity risk, also known as obesogenic environments.[23,145,149,196] Differential quality of neighborhood socioeconomic and built environments that either support or limit healthy eating and active living is one potential mechanism through which neighborhood segregation creates disadvantage within communities of color and perpetuates inequities in youth obesity.[25] Quality of neighborhood environments and access to opportunities transcends family-level SES among families of color.[34,45] For example, after controlling for family poverty levels, McArdle et al. found that 40% of Black youth and 32% of Hispanic youth lived in the bottom quintile of neighborhood opportunity for education, socioeconomic, and built environment opportunities, compared to only 9% of White youth.[41] Therefore, the current study should consider potential mediators or moderators on the association between residential segregation and youth obesity at the neighborhood level including socioeconomic and built environments. Black and Hispanic youth of all family income levels are more likely to live in segregated and/or low-income communities when compared to White youth. However, the inclusion of a significant interaction between Hispanic residential segregation and youth race/ethnicity suggested that after controlling for other study variables, as Hispanic residential segregation increased, BMI z-score for all youth increased and was relatively similar at the highest levels of segregation. While this interaction term did not improve model fit, it did provide preliminary evidence that segregated environments have negative health consequences for all youth.

These findings suggest that community and neighborhood development and infrastructure are important targets for policy and intervention to reduce youth obesity risk in segregated communities. Potential solutions include housing policies that provide opportunities for residential mobility and home ownership among Black and Hispanic families, including prioritizing sufficient inventory of affordable homes, mixed-income housing, and enhanced credit or down payment programs.[38,197] In addition to supporting mixed-income housing that fosters more diverse communities, the use of community-engaged or participatory processes could ensure these neighborhoods are not only affordable, but are designed to be culturally inclusive and supportive of opportunities for families of color.[56,198]

Limitations

This study provides significant contributions to the literature on the association between residential segregation and obesity, but it does have several limitations. The first is the cross-sectional nature of the data. This limits the study's ability to draw causal inferences about the relationship between residential segregation and youth obesity.[21,51] To support causal inferences, future studies should consider incorporating longitudinal measures of residential segregation. This may be of particular importance, given there is some evidence to indicate that the impact of neighborhood environments, including negative socioeconomic and built environment exposures, can arise out of residential segregation, and may create cumulative complexity that is time or dose dependent.[199-201]

The second limitation is that no individual-level control for family SES was available. This may be an important confounding variable in the observed associations between residential segregation and youth obesity, because of the significant amount of literature about the relationship between family level poverty and youth obesity.[6,57,69,202] Future studies should consider incorporating individual or family-level measures of SES, such as school lunch status, poverty, or income.

Another important limitation of this study is the modifiable areal unit problem (MAUP) or Uncertain Geographic Problem (UCGoP). This issue occurs when individuals are aggregated into population counts within areal units, including census tracts, that do not have an underlying mean such as a neighborhood.[21] Census tracts are still frequently used as neighborhood proxies because of the data that are available through sources such as the US Census Bureau. However, future studies should consider using and contrasting with spatial measures of residential segregation, such as Reardon et al.'s spatially continuous kernel densities to create neighborhoods that are defined as a point in space around a location like home address.[172,203]

Lastly, this study was focused on one large southeastern county in the US, which may limit the generalizability of findings to other locations across the country. The strength of association between residential segregation and youth obesity may vary by state or region if racial/ethnic intergroup relations are influenced by history and/or local context.[34,41,141] For example, in some localities, living in a residentially segregated neighborhood may serve as a protective factor for health outcomes through mechanisms such as limiting exposures to racism or discrimination.[21,52] Despite this limitation, the localized context of this study may be an important contribution to the literature as Kreiger et al. found that local segregation compared to city or MSA level segregation was more strongly associated with fatal and non-fatal assaults.[163] Future studies should consider opportunities where the relationship between residential segregation and youth obesity can be simultaneously explored at both the local and larger regional area.

Conclusions

In summary, this study highlights the importance of neighborhood and school environment characteristics, such as residential segregation and school segregation, in youth obesity risk. This study found significant associations between both school-level and neighborhood-level segregation, where school segregation was negatively associated with youth obesity and residential segregation was positively associated with youth obesity, after controlling for individual, school, and neighborhood covariates. The association was strongest for Hispanic segregation, with moderation analyses suggesting individual race/ethnicity may moderate the association between both Hispanic school and residential segregation and youth obesity risk. Overall, this study provides an important contribution to the literature on how the dynamics of segregation can vary by definitions and measures of residential segregation (Black versus White, Hispanic versus White, Minority versus White, etc.), context (school versus neighborhood environments), and individual youth racial or ethnic identity. These complex dynamics necessitate further exploration of this poorly studied research area and careful considerations of potential policy and built environment solutions to support healthy behaviors for youth and their families, including protective versus health harming impacts and their interplay across forms of segregation, context of segregation, and individual race/ethnicity.

Acknowledgments

This study would not have been possible without the support of LiveWell Greenville and their community partners. LiveWell Greenville is a local coalition working on equitable access to healthy eating and active living opportunities through policy, systems, and environment change in Greenville County, SC. We would also like to acknowledge Furman University's Institute for the Advancement of Community Health. The IACH has been a champion of this work and the partnership with LWG. The Institutional Review Board at Furman University approved the study, and all procedures followed were in accordance with the ethical standards of the IRB and the Helsinki Declaration of 1975, as revised in 2000.

Table 4.1 Summary of model building process

Model	Predictor Variables and Covariates
Model 1 (Null)	None
Model 2[a] (Control)	Grade, Race/ethnicity
Model 3 (School)	Grade, Race/ethnicity, School Poverty, Black School Segregation, Hispanic School Segregation
Model 4[b] (Neighborhood)	Grade, Race/ethnicity, Black Residential Segregation, Hispanic Residential Segregation, Educational Attainment
Model 5 (Combined School and Neighborhood Model)	Grade, Race/ethnicity, School Poverty Index, Black School Segregation, Hispanic School Segregation, Black Residential Segregation, Hispanic Residential Segregation Neighborhood Educational Attainment
Model 6[c] (Combined Model with interaction between Hispanic school seg. and youth race/ethnicity)	School Poverty Index, Black School Segregation, Black School Segregation, Black Residential Segregation, Hispanic Residential Segregation Educational Attainment, Residential segregation*Black, Residential segregation*Hispanic
Model 7[d] (Combined Model with interaction between Hispanic residential seg. and youth race/ethnicity)	School Poverty Index, School Segregation, Residential Segregation, Educational Attainment, Residential segregation*Black, Residential segregation*Hispanic

[a]Model initially included biological sex, the term was removed because it was not significant and did not improve model fit

[b]Model initially included neighborhood poverty, the term was removed because it was not significant and did not improve model fit

[c]Model initially included interaction term between Black school segregation and race/ethnicity, the term was removed because it was not significant and did not improve model fit

[d]Model initially included interaction term between Black residential segregation and race/ethnicity, the term was removed because it was not significant and did not improve model fit

Table 4.2 Descriptive statistics of youth, school, and neighborhood characteristics

	Percentage	Frequency
	Youth Characteristics (n=11364)	
Biological Sex		
Male	51.6%	5866
Female	48.4%	5498
Race/ethnicity		
White (ref)	61.3%	6970
Black	23.2%	2640
Hispanic	15.4%	1754
Grade		
Second	36.6%	4163
Fifth	39.2%	4453
Eighth	13.2%	1494
Ninth	11.0%	1254
Youth Weight Status		
Normal	66.9%	7601
Overweight	17.8%	2020
Obese	15.3%	1743

	M (SD)	Minimum	Maximum
	Youth Characteristics (n=11,364)		
Youth BMI z-score	0.48 (1.16)	-5.59	3.052
Youth BMI percentile	62.99 (30.20)	1.15	99.89
	School Characteristics (n=80)		
School Black LQ	0.97 (1.004)	0.06	2.72
School Hispanic LQ	0.99 (0.90)	0.00	4.50
School Poverty Index	53.33 (20.41)	16.23	91.69
	Neighborhood Characteristics (n=111)		
Black LQ	1.04 (0.89)	0	4.69
Hispanic LQ	1.03 (0.91)	0	4.32
Educational Attainment	24.65 (9.18)	5.60	46.10

Table 4.3 Bivariate associations between BMI z-score, youth demographic, school, and neighborhood characteristics (n=11364)

	1. BMIz	2. Black	3. Hispanic	4. White	5. Second	6. Fifth	7. Eighth	8. Ninth	9. S. Black LQ	10. S. Hisp LQ	11. Black LQ	12. Hispanic LQ	13. SPI	14. Edu Att.
1.	-													
2.	0.11*	-												
3.	0.12*	-0.22*	-											
4.	-0.18*	-0.63*	-0.54*	-										
5.	-0.08*	0.01	-0.00	-0.01	-									
6.	0.06*	-0.01*	0.01	0.00	-0.61*	-								
7.	0.03*	-0.01	-0.01	0.00	-0.30*	-0.31*	-							
8.	0.00	0.01	0.00	0.01	-0.28*	-0.28*	-0.14*	-						
9.	0.08*	0.28*	0.17*	-0.38*	0.03*	-0.02*	0.00	-0.01	-					
10.	0.09*	0.11*	0.39*	-0.39*	0.03*	-0.01	-0.01	-0.02*	0.42*	-				
11.	0.11*	0.42*	0.09*	-0.45*	0.02*	-0.01	-0.04*	-0.04*	0.62*	0.03*	-			
12.	0.100 *	0.04*	0.40*	-0.34*	0.01	0.00	-0.02	-0.01	0.52*	0.68*	0.16*	-		
13.	0.14*	0.17*	.28*	-0.37*	0.09*	0.06*	-0.03	-0.18*	0.62*	0.71*	0.37*	0.48*	-	
14.	0.16*	0.18*	0.18*	-0.30	0.04*	-0.01	0.03*	-0.07*	0.25*	0.34*	0.42*	0.37*	0.56*	-

*p<.05

Table 4.4 Cross classified models predicting the association between youth BMI z-score and individual, school, and neighborhood level characteristics

	Model 1 *b* (SE)	Model 2 *b* (SE)	Model 3 *b* (SE)	Model 4 *b* (SE)	Model 5 *b* (SE)	Model 6 *b* (SE)	Model 7 *b* (SE)
Fixed Effects							
Intercept	0.520***	0.227***	0.195***	0.230***	0.210***	0.226	0.224***
Race/eth (White=ref)	-						
Black	-	0.369***	0.369***	0.326***	0.328***	0.323*	0.326***
Hispanic	-	0.432***	0.431***	0.385***	0.389***	0.401*	0.398***
Grade							
Fifth	-	0.207***	0.206***	0.209***	0.208***	0.209*	0.209***
Eighth	-	0.182**	0.230**	0.190*	0.226**	0.224*	0.226**
Ninth	-	0.152*	0.260**	0.165*	0.247**	0.240*	0.247**
	Level 2 School and Neighborhood Characteristics						
School Black LQ	-	-	-0.105*	-	-0.102*	-0.109*	-0.101
School Hisp LQ	-	-	-0.092*	-	-0.114*	-0.051	-0.110*
SPI	-	-	0.010***	-	0.007***	0.008*	0.008*
Black LQ	-	-	-	0.029	0.037*	0.034*	0.025
Hispanic LQ	-	-	-	0.050*	0.057**	0.060*	0.127***
Education		-	-	0.008***	0.006**	0.006*	0.006*
	Cross Level Interactions						
School Hisp LQ*race							
S Hisp LQ*Black	-	-	-	-	-	-0.101*	-
S Hisp LQ*Hispanic	-	-	-	-	-	-0.097*	-
Neigh Hisp LQ*race							
Hisp LQ*Black	-	-	-	-	-	-	-0.127***
Hisp LQ*Hisp	-	-	-	-	-	-	-0.096**
Model Fit							
-2LL	35072.6	34760.4	34741.4	34712.2	34699.5	34687.8	34670.2

AIC	35080.6	34778.4	34765.4	34734.2	34727.5	34719.8	34706.2
Error Variance							
Level-1 Residual	1.255***	1.223***	1.230 ***	1.228***	1.228***	1.227*	1.226***
Level-2 School	0.048***	0.045***	0.033***	0.039***	0.032***	0.032*	0.033***
Level-2 Neighborhood	0.025***	0.005*	0.003*	0.000	0.000	0.000	0.000

*p<.05, **<p.001, ***p<.0001

Table 4.5 Model Fit Comparisons for cross classified random effects models predicting youth BMI z-score from residential segregation

	Model 7 to 5	**Model 6 to 5**	**Model 5 to 4**	**Model 5 to 3**	**Model 5 to 2**	**Model 2 to 1**
Δ Pseudo R^2	-0.002*	.012*	0.055*	0.055*	0.247*	0.305*
Δ -2 log likelihood (obtained X^2)	13.9	11.7	12.7	41.9	60.9	312.2
Δ in fixed effects (df)	2	2	3	3	6	5
X^2 critical value	5.991	5.991	7.815	7.815	12.592	11.071

*p<.05 Pseudo R^2 Model 7=0.5612 Pseudo R^2 Model 4=0.4562 Pseudo R2 Model 1=0
Pseudo R^2 Model 6=0.5497 Pseudo R^2 Model 3=0.4970
Pseudo R^2 Model 5=0.5519 Pseudo R^2 Model 2=0.0453

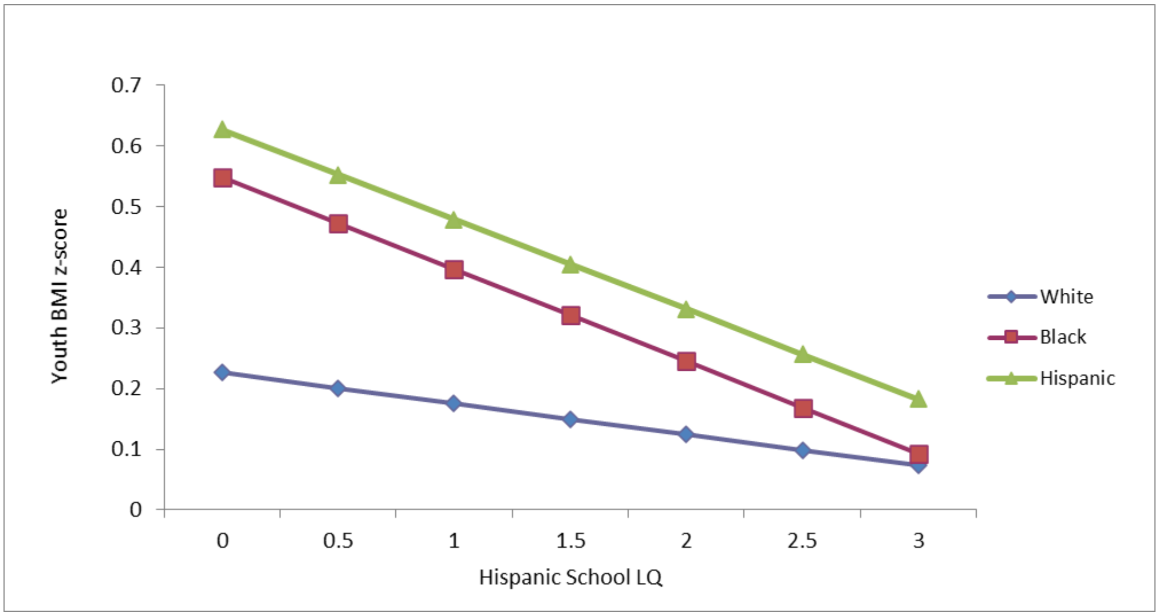

Figure 4.1 Plot of predicted BMI z-score with interaction between Hispanic school segregation and youth race/ethnicity

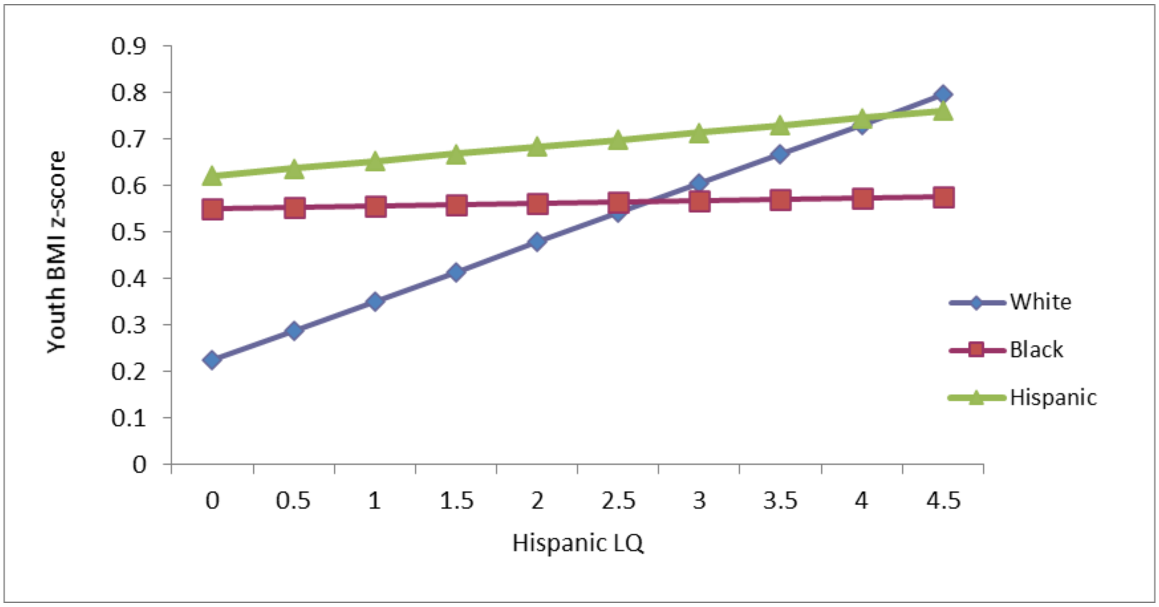

Figure 4.2 Plot of predicted BMI z-score with interaction between Hispanic residential segregation and youth race/ethnicity

CHAPTER FIVE: MANUSCRIPT TWO

SOCIAL AND ECONOMIC, HEALTH AND BUILT, AND EDUCATION ENVIRONMENTS AS MEDIATORS IN THE ASSOCIATION BETWEEN NEIGHBORHOOD RESIDENTIAL SEGREGATION AND YOUTH OBESITY[2]

[2] Fair, M. L., Kaczynski, A. T., Monroe, C., Powers, A. R., Rudisill, A. To be submitted to *Health and Place*.

Abstract

Youth obesity risk cannot be explained by individual and family risk factors alone. Neighborhood socioeconomic and built environments have emerged as important settings to examine the multiple contexts and factors that influence youth obesity risk and associated disparities by income, education, and race/ethnicity. Children and families of color are more likely to live in residentially and economically segregated communities that limit access opportunities and resources, including social and economic, health and built environment, and educational opportunities, and in turn impact economic mobility and health across the life course. This study used parallel mediation models to examine how neighborhood social/economic, health/built, and education environments, simultaneously, mediated the association between residential segregation at the census tract level and BMI z-score among school-age youth (n=11,364) in a large southeastern county in the US. Study analyses indicated that residential segregation indirectly affected youth BMI z-score through three domains of childhood opportunity, as measured by the Childhood Opportunity Index (social/economic, health/built, education environments), after controlling for individual youth characteristics. Further, the significance of the partial indirect effects of the three mediators varied by combinations of Black versus Hispanic segregation and individual race/ethnicity (Black versus Hispanic). Few studies have examined the association between residential segregation and youth obesity, and this is the first study to our knowledge that explored potential mediators of the association. This study has important implications for how structural determinants and systems influence investment and opportunity within neighborhoods that impact health outcomes such as obesity among minority youth.

Introduction

Prevalence of youth obesity in the United States (US), with significant disparities by demographic characteristics including race/ethnicity and socioeconomic (SES) status.[1,68] Black and Hispanic youth experience higher burdens of youth obesity and are more likely to be affected by its short and long-term negative health consequences.[4,69] Youth with obesity are also more likely to experience obesity as adults, resulting in increased chronic disease, morbidity, and mortality among Black and Hispanic youth and adults over the life course.[80,81]

Individual and family-level factors, such as genetics and health behaviors do not fully explain youth obesity and chronic disease risk.[12,67,84,92] Grounded in the social ecological model, the beginning of the 21st century witnessed a surge in research exploring the relationship between neighborhoods and health outcomes, including youth obesity.[22,207] Previous studies have linked neighborhood environment features to a number of health outcomes, including depression and mental health, cancer risk, and birth outcomes, but others have found mixed findings for certain health behaviors and outcomes, including physical activity and obesity.[40,113,115,116]

One important neighborhood concept, as it relates to youth obesity, is the "obesogenic environment," which can be defined as the neighborhood opportunities and exposures, or lack thereof, that contribute to obesity risk.[63,204,205] Obesogenic environments are comprised of both socioeconomic and built environment characteristics, such as a lack of parks/green space, high density of fast-food restaurants, low walkability, poverty, prevalent crime, and low social cohesion that can limit physical activity opportunities and increase access to calorically dense, nutritionally void foods.[23,40,117]

Decades of research have explored the important role of neighborhoods in health and obesity risk, with a primary focus on specific features of neighborhood environments. However, less research has considered the mechanisms by which neighborhood socioeconomic and environmental characteristics influence obesity risk.[33] Understanding these mechanisms is key to elucidating how neighborhoods create and sustain health disparities by SES and race/ethnicity. One such potential mechanism is residential segregation.[34]

Residential segregation is defined as the spatial separation of two or more social groups within a specified geographic area, such as a municipality, county, or metropolitan area.[33] Williams and Collins posited residential segregation as a fundamental cause of health inequities by race/ethnicity.[34] By definition, fundamental causes of health inequities: 1) involve access to resources that enable or prevent disease, 2) affect multiple health conditions through multiple pathways, and 3) are sustained or continue to emerge over time despite innovations in health interventions.[153] Specifically, Williams and Collins proposed that residential segregation leads to differences in educational opportunity and SES, which in turn drive the US health gradient by race/ethnicity.[34,47]

Supporting this theory of residential segregation as a fundamental cause is the concept of "Geography of Opportunity," introduced by Galster and Killen, where the social, economic, environmental, and cultural aspects of a neighborhood influence life chances and health outcomes beyond individual or family characteristics.[206,207] In the recent decades, significant research has explored the impact of early life opportunities across the life course.[149] One such line of inquiry is the Childhood Opportunity Index

(COI), which is a measure of neighborhood social and economic environment, health and built environment, and education environment opportunities that have been linked to child well-being, economic mobility, and health outcomes.[149] A recent study using component indicators of the COI 2.0 found that a higher number of high quality early childhood centers and greater access to healthy foods were associated with lower adiposity and improved metabolic risk factors among youth.[208]

Other studies have also explored the role of neighborhood socioeconomic environments on youth obesity risk and found similar results. Kramer et al. examined the geospatial distribution of youth obesity across the US and found that clustering of obesity in the south and Appalachian regions was linked to county characteristics, including physical activity environments, community SES, community health, and social marginalization.[209] Similarly, Bell et al. found that county level Black-White inequality in SES, home ownership, and unemployment were associated with obesity and obesogenic environments.[205]

While significant research has explored the intersections between residential segregation, neighborhood opportunity, and health, findings for the association between residential segregation and obesity are mixed and little research has specifically examined youth obesity.[40,56] Further, few studies have examined the specific pathways by which residential segregation impacts obesity risk among youth and adults.[30,58,150] Goodman et al. examined if health behaviors and health environments mediated the association between residential segregation and obesity, finding that dietary behaviors partially and food environments fully mediated the association.[58] In another study, Chang Martinez et al. proposed neighborhood disorder as a potential mediator in the association between Black-White residential isolation and obesity. They found that physical disorder (e.g., vacant houses, housing code violations, etc.), but not social disorder (e.g., crime), partially mediated the association for women but not for men.[150]

Study Purpose

The purpose of this study was to explore whether neighborhood social and economic environments, health and built environments, and education environments mediated the associations between residential segregation and youth obesity. Specifically, this study hypothesized that 1) social/economic, health/built, and education environments would mediate the relationship between residential segregation and youth obesity, and 2) the significance of the mediating effects would vary by type of segregation (Black versus Hispanic) and individual youth race/ethnicity (Black versus White).

Methods

Study Setting and Sample

The community of study was a large county in the southeastern US that covers more than 800 square miles and contains 111 census tracts, with pockets of urban and rural areas. It is also the most populous county in the state with approximately 523,542 residents, 22.9% of which are children 18 years of age and younger and 6.2% are children 5 years of age and younger.[165] The county is predominantly non-Hispanic White (76.3%), followed by Black (18.4%) and Hispanic (9.5%) residents. Approximately 87.3% of residents over the age of 25 have their high school diploma and 33.3% have a bachelor's degree or higher.[210] The median household income was $60,351 in 2019, and approximately 10.6% of individuals live in poverty. Poverty rates among children are higher, with 15.0% of children 18 years of age and younger living in poverty, 21.3% of children 5 years of age and younger living in poverty, and higher poverty rates among children and families of color.[165]

Inclusion criteria for the study included having complete height, weight, and demographic data for the spring 2017 data collection time point (discussed further below). Exclusion criteria included youth who identified as belonging to a racial or ethnic category other than Black, Hispanic, or White, as the race/ethnicity categories within the "Other category" were small and heterogeneity by racial/ethnic category and association with youth BMI z-score within the category limited its application in analysis and interpretation.

Data Collection and Measures

Data used in this study included individual youth variables and neighborhood measures at the census tract level. Individual youth data were obtained from a local school district's *PowerSchools* and *FitnessGram* databases as part of a datasharing agreement with a local university.[64,167] Individual youth variables obtained included youth height, weight, age in months, grade, biological sex, race/ethnicity, and home address.

Neighborhood-level (census tract) measures were obtained from two sources: 1) the Institute for Child, Youth and Family Policy at the Heller School for Social Policy and Management at Brandeis University, and 2) American Community Survey (ACS) data that was downloaded from the National Historical Geographic Information System (NHGIS) website.[66,168] As is described further below, neighborhood-level measures obtained included COI 2.0 social and economic, health and built environment, and education z-scores, as well as population estimates by race/ethnicity at the census tract and county-level. Individual and neighborhood-level datasets were joined into one database using a unique identifier for each youth provided by the school district and a census tract identifier.

Individual Measures

Youth Weight Status. Youth weight status was the dependent variable of interest and was operationalized in analyses as BMI z-score (a continuous variable). Youth height, weight, biological sex, and age in months were used to calculate youth BMI z-scores using standardized protocols developed by the Centers for Disease Control and Prevention.[169] BMI z-score is the number of standard deviation units above or below the mean of the sample distribution. It is widely used in youth obesity research as it accounts for uneven distributions and prevents extreme values from skewing the distribution.[171]

Youth Level Covariates. Youth level covariates included biological sex (male=0, female=1), grade (2nd=0, 5th=1, 8th=2, 9th=3), and race/ethnicity (White=0, Black=1, Hispanic=2). The "Other" race category was not included in study analyses because of the small sample sizes for certain race/ethnicity categories and heterogeneity across categories (i.e., includes Asian, American Indian, etc.), which limited its use in analysis and interpretation.

Neighborhood Level Measures

Black and Hispanic residential segregation. The independent variable of interest, or predictor, in this study was neighborhood residential segregation by race/ethnicity (Black and Hispanic). Neighborhood residential segregation, at the census tract level, was calculated using the Location Quotient (LQ). The LQ is a localized measure of residential segregation that is calculated by dividing the proportion of one group at the local level (census tract) by the proportion of that group in the broader area (county).[21,172] If the proportion of one group in a local area is equivalent to the larger area, the LQ will equal one, if the local proportion is greater than regional proportion the LQ will be greater than one, and if the proportion is less than the regional proportion the LQ will be less than one. The LQ can range from zero to infinity, where less than 0.85 is the standard cutoff for underrepresentation of a population and 1.2 is overrepresentation, or segregation, of a population.[172] Neighborhood residential segregation LQs were calculated for Black and Hispanic residential segregation, separately, and then grand mean centered to improve interpretability in study analyses.

Neighborhood social/economic, health/built, and education environments. Three dimensions of the neighborhood environment were included as mediators in this study – the social and economic environment, the health and built environment, and the education environment – which were operationalized using 2015 nationally normed domain z-scores from the COI 2.0. The COI 2.0 measures and maps the quality of resources and conditions at the neighborhood level that influence childhood health and development.[66] The COI 2.0 calculates scores for approximately 72,000 census tracts in the US at two time points, which are approximately 2010 and 2015. The exact year and range of individual data points can vary based on the indicator and source.

The COI 2.0 includes three domains –social and economic, health and built environment, and education. The three domains are constructed from 29 component indicators that are conceptually and empirically linked to the domain score and are detailed in Table 5.1. To create the COI domain scores, each of the component indicators are standardized using z-scores to create a uniform measurement scale and then weighted based on their association with select health and economic outcomes. A detailed description of the development, detailed methodology, and validation of the index can be found in the COI 2.0's Technical Documentation document.[66]

Analytic Approach

Mediation is the process by which a third variable serves as the causal pathway in the association between the independent and dependent variables of interest. This variable can fully, or partially, explain the association the focal relationship.[181] To accomplish aim 2, this study utilized mediation analysis to determine if, and to what extent, neighborhood 1) social and economic environment, 2) health and built environment characteristics, and 3) education environments mediated the association between residential segregation and youth obesity. Specifically, the 2015 COI social and economic environment, health and built environment, and education environment z-scores were included as potential mediators in the association between each of Black and Hispanic residential segregation (residential segregation LQs) and the outcome of interest, youth BMI z-score. Figure 3.5 outlines the conceptual model of the mediation pathway that was used in this study.

Given that previous research has identified the differential impacts of segregation by youth race/ethnicity, the model building and mediation testing process was completed, separately, using four different combinations of residential segregation and youth race/ethnicity.[40] The four combinations were as follows: Black residential segregation among Black youth compared to White youth, Black segregation among Hispanic youth compared to White youth, Hispanic segregation among Black youth compared to White youth, and Hispanic segregation among Hispanic youth compared to White youth. Final covariates included in each of the mediation analyses were different for Black and Hispanic youth; where race/ethnicity (White=0, Black=1, Hispanic=2) and grade (2^{nd}=0, 5^{th}=1, 8^{th}=2, 9^{th}=3) were significant for models including Black and Hispanic youth, while biological sex (male=0, female=1) was only significant for models including Black youth.

Preliminary study analyses indicated that approximately 4% of the variation in youth BMI z-score was attributable to variation across neighborhood environments, based on calculation of the Intraclass Correlation Coefficient. However, after the inclusion of individual and neighborhood-level predictors, the unexplained variation by neighborhood was approximately 0.5%. Therefore, single-level linear models were utilized for all analyses, allowing for the use of PROCESS macro in SAS version 9.4, developed by Hayes.[180] PROCESS macro is a modeling tool that can handle complex mediation models, including multiple mediators, simultaneously, and produces estimates of direct and indirect effects along with bootstrapping confidence intervals.

Prior to completing the mediation analyses, descriptive statistics were obtained for each study variable. Bivariate analyses were then used to assess the association between the main outcome variable, youth obesity (BMI *z*-score), and the mediators (COI social/economic environment z-score, health/built environment z-score, education environment z-score). Data were also examined for violations of assumptions of Ordinary Least Squares Regression. All analyses were conducted using SAS version 9.4.[177]

PROCESS macro uses the product of the coefficients approach to determine the indirect effect of the predictor (Black or Hispanic LQ) that is transmitted through the hypothesized mediator(s) onto the outcome (BMI z-score).[180] PROCESS macro uses a set of OLS regression, to calculate the total effect of X (residential segregation) on Y (BMI z-score), the direct effect of X on Y independent of the mediators, and the indirect effect of X on Y transmitted through M (neighborhood social/economic, health/built, and education environments). It also produces lower and upper confidence limits for the direct and indirect effects, which are calculated using the bootstrap resampling method (n=5000). The confidence intervals can be used to test the significance level of mediating effects, where significant effects are those that do not contain zero in the confidence interval.[180,211] Bootstrapping is a non-parametric approach that is preferred over the normal theory approach, also known as the Sobel test, as it makes no assumption about the shape of the sampling distribution of the coefficients and is more robust to error and yields more accurate confidence interval estimates for the indirect effect.[180]

Results

Sample Characteristics

Sample characteristics of individual youth and neighborhoods are presented in Table 5.2. The final study sample included 11,364 youth in second, fifth, eighth, and ninth grade from 111 census tracts or neighborhoods. Each census tract contained an average of 108 (SD=68.19) youth, with a minimum of 2 and a maximum of 368 youth per census tract. There were slightly more male youth (51.52%) in the sample than female youth (48.38%). White youth (61.3%) were the largest racial/ethnic group, followed by Black (23.3%) and Hispanic (15.4%) youth. The distribution of youth by grade was as follows: second (36.63%), fifth (39.19%), eighth (13.15%), and ninth (11.03%) grade.

At the neighborhood level, the average Black LQ was 1.04 (SD=0.89) and the average Hispanic LQ was 1.03 (SD=0.91), indicating that, on average, census tracts in the county are not residentially segregated. The maximum Black residential segregation LQ was 4.69, which translates to the Black population within that census tract being 4.69 times higher than the Black population in the county. Similarly, the maximum Hispanic residential segregation LQ was 4.32, while the lowest Black and Hispanic residential segregation LQs were zero, indicating no Black or Hispanic residents live within the census tract. The average neighborhood COI social and economic environment z-score was 0.06 (SD=0.18), with a minimum score of -0.49 and maximum score of 0.34. The average neighborhood COI health and built environment z-score was -0.001 (SD=0.05), with a minimum score of -0.20 and a maximum score of 0.06. The average neighborhood COI education environment z-score was 0.03 (SD=0.06), with a minimum score of -0.10 and a maximum score of 0.14.

Bivariate Statistics

Pearson correlation coefficients were obtained for all study independent and dependent variables. There was a small, positive correlation between youth BMI z-score and belonging to the Black ($r=0.11$, $p<.0001$) or Hispanic ($r=.12$, $p<.0001$) race/ethnicity category. There were also small, but positive correlations between youth BMI z-score and Black residential segregation ($r=0.11$, $p<.0001$) as well as Hispanic residential segregation ($r=0.10$, $p<.0001$). The COI social/economic environment z-score ($r=-0.15$, $p<.0001$), the health/built environment z-score ($r=-.12$, $p<.0001$), and the education environment ($r=-0.16$, $p<.0001$) z-score had small, negative correlations with BMI z-score. Each of the COI domain z-scores also had large and negative correlations with both Black residential segregation (social/economic r=-0.64, p<.0001; health/built environment=-0.56, p<.0001; education r=-0.50, p<.0001) and Hispanic residential segregation (social/economic r=-0.56, p<.0001; health/built environment r=-0.57, p<.0001; education r=-0.50, p<.0001).

Mediation Analyses

Table 5.3 presents the best fitting parallel mediator models for the association between Black segregation and BMI z-score among Black youth, compared to White youth. As shown in Table 5.4, there was a positive and statistically significant association for the total effect (c) of Black residential segregation on BMI z-score among Black youth (*b*=0.065, 95% CI [0.015, 0.094]), after adjusting for individual controls. With the addition of the three parallel mediators, the direct effect (c') of Black residential segregation on BMI z-score among Black youth was no longer significant (*b*=-0.018, 95% CI [-0.053, 0.018]). There was a positive and statistically significant association for total indirect effect ($a_1b_1+a_2b_2+a_3b_3$) of Black residential segregation (*b*=0.082, 95% CI [0.061, 0.105]) on BMI z-score among Black youth, transmitted through the three parallel mediators. When examining the significance of the partial indirect effects (a_1b_1, a_2b_2, a_3b_3) by mediator using bootstrap sample (n=5000) confidence intervals, after adjusting for the other mediators and controls in the model, the partial indirect effect of the health/built environment z-score was not significant (*b*=-0.015, 95% CI [-0.041, 0.011]). The partial indirect effects were significant for the social/economic z-score (*b*=0.036, 95% CI [0.004, 0.067]) and the education environment z-score (*b*=0.062, 95% CI [0.046, 0 .078]). As shown in Table 5.3, when examining the component coefficients of the significant partial indirect effects, as Black residential segregation increased, the social/economic environment z-score decreased (a_1=-0.114), and in turn when social/economic z-score increased, BMI z-score decreased (b_1=-.316). Similarly, as Black residential segregation increased, the education environment z-score decreased (a_3=-0.030), and in turn when the education z-score increased, BMI z-score decreased (b_3=-2.069).

Table 5.5 presents the best fitting parallel mediator models for the association between Black segregation and BMI z-score among Hispanic youth, when compared to White youth. As shown in Table 5.4, there was a significant and positive association for the total effect (c) of Black residential segregation (*b*=0.040, 95% CI [0.003, 0.077]) on BMI z-score among Hispanic youth, after adjusting for individual controls. With the addition of the three parallel mediators, the direct effect (c') of Black residential segregation on BMI z-score among Hispanic youth was still significant, but the direction of the association changed, and the strength of the association was reduced (*b*=-0.078, 95% CI [-0.121, -0.034]). There was a significant and positive association for the total indirect effect ($a_1b_1+a_2b_2+a_3b_3$) of Black residential segregation (*b*=0.118, 95% CI [0.094, 0.142]) on BMI z-score among Hispanic youth, transmitted through the three parallel mediators. When examining the significance of the partial indirect effects (a_1b_1, a_2b_2, a_3b_3) by mediator using bootstrap sample (n=5000) confidence intervals, after adjusting for other mediators and controls in the model, the partial indirect effects for all three COI z-scores were significant: social/economic z-score (*b*=0.041, 95% CI [0.005, 0.077]), health/built z-score (*b*=-0.041, 95% CI [-0.064, -0.018]), education environment z-score (*b*=0.118, 95% CI [0.094, 0.143]). As shown in Table 5.5, when examining the component coefficients of the significant partial indirect effects, as Black residential segregation increased, the social/economic environment z-score decreased (a_1=-0.120), and in turn when social/economic environment z-score increased, BMI z-score decreased (b_1=-.338). Conversely, while Black residential segregation increased, the health/built environment z-score decreased (a_2=-0.025), but as the health/built environment z-score increased, BMI z-score also increased (b_2=1.667). Like the social/economic environment z-score, as Black residential segregation increased, the education environment z-score decreased (a_3=-0.041), and in turn when the education z-score increased, BMI z-score decreased (b_3=-2.860).

Table 5.6 presents the best fitting parallel mediator models for the association between Hispanic segregation and BMI z-score among Black youth, when compared to White youth. As shown in Table 5.4, there was a significant and positive association for the total effect (c) of Hispanic residential segregation (*b*=0.078, 95% CI [0.047,0.110]) on BMI z-score among Black youth. With the addition of the three parallel mediators, the direct effect (c') of Hispanic residential segregation was not significant (*b*=0.007, 95% CI [-0.028, 0.042]). There was a significant and positive association for total indirect effect (a_1b_1+a_2b_2+ a_3b_3) of Hispanic residential segregation (*b*=0.071, 95% CI [0.055,0.088]) on BMI z-score among Black youth, transmitted through the three parallel mediators. When examining the significance of the partial indirect effects (a_1b_1, a_2b_2, a_3b_3) by mediator using bootstrap sample (n=5000) confidence intervals, after adjusting for covariates and other mediators in the model, only the partial indirect effect of the education environment z-score was significant (*b*=0.061, 95% CI [0.045, 0.077]). As show in Table 5.6, when examining the component coefficients of the significant partial indirect effect, as Hispanic residential segregation increased, the education environment z-score decreased

(a_3=-0.30), and in turn when the education z-score increased, BMI z-score decreased (b_3=-2.309).

Table 5.7 presents the best fitting parallel mediator models for the association between Hispanic segregation and BMI z-score among Hispanic youth. As shown in Table 5.4, there was a significant and positive association for the total effect (c) of Hispanic residential segregation (*b*=0.082, 95% CI [0.052, 0.114]) on BMI z-score among Hispanic youth. With the addition of the three parallel mediators, the direct effect (c') of Hispanic residential segregation on BMI z-score among Hispanic youth was no longer significant (*b*=0.035, 95% CI [-0.003, 0.072]). There was a significant and positive association for total indirect effect (a_1b_1+a_2b_2+a_3b_3) of Hispanic residential segregation (*b*=0.047, 95% CI [0.023, 0.071]) on BMI z-score among Hispanic youth, transmitted through the three parallel mediators. When examining the significance of the partial indirect effects (a_1b_1, a_2b_2, a_3b_3) by mediator using bootstrap sample (n=5000) confidence intervals, after adjusting for covariates and other mediators in the model, the partial indirect effect of the social/economic environment z-score was not significant (*b*=0.18, 95% CI [-0.013, 0.049]). The partial indirect effects were significant for the health/built z-score (*b*=-0.056, 95% CI [-0.084, -0.027]) and the education environment z-score (*b*=0.085, 95% CI [0.067 0.104]). As shown in table 5.7, when examining the component coefficient of the significant partial indirect effects, as Hispanic residential segregation increased, the health/built environment z-score decreased (a_2=-0.029); however, when the built/health environment z-score increased, BMI z-score increased (b_2=1.916). For the education environment z-score, as Hispanic residential segregation increased, the education environment z-score decreased (a_3=-0.032), and in turn when the education z-score increased, BMI z-score decreased (b_3=-2.68).

Discussion

There is preliminary evidence to support the association between residential segregation and youth obesity, but there is little to no evidence about the potential mediators in this association.[56,57,59] The current study's findings provide an important contribution to the field of youth obesity research in this area. Overall, results indicate that residential segregation indirectly effects youth BMI z-score through a number of neighborhood environment characteristics, including the social/economic, health/built, and education environments, after controlling for individual youth characteristics. Additionally, the significance of mediators varied by Black versus Hispanic segregation and by individual race/ethnicity (Black versus Hispanic).

After the addition of the three parallel mediators, the direct effect of residential segregation only remained significant for Black segregation among Hispanic youth. The partial indirect effect of the education environment was the only mediator that was significant for all combinations of residential segregation by race and by individual youth race/ethnicity. The indirect effect of the social/economic environment was significant for Black segregation among Black and Hispanic youth, compared to White youth, but was not significant for Hispanic segregation. Conversely, the health/built environment was significant for Black and Hispanic segregation, but only among Hispanic youth.

The limited amount of research that has specifically explored not only the association between residential segregation and youth obesity, but the potential mediators of this association make it difficult to make specific comparisons with prior research.

However, previous studies have found significant associations with overall childhood opportunity, as measured by the COI index, and its component indicators, with a number of health outcomes, including asthma, birth outcomes, and cardiometabolic risk factors.[149,208] Additionally, previous research has identified neighborhood environment features, including access to healthy food and physical disorder (e.g., housing vacancy or code violations), as mediators in the association between residential segregation and adult obesity.[30,51] Future research is needed to confirm the present study findings of neighborhood environments as potential mediators in the association between residential segregation and youth obesity as well as if there are consistent patterns that continue to emerge at the intersection of type of residential segregation (Black versus Hispanic) and individual youth race/ethnicity.

The findings from this study have important policy and practice implications for the prevention and reduction of youth obesity and disparities by race/ethnicity. Local governments should consider if and how decision-making processes or policies encourage inequitable financial and infrastructure investments in low-income communities and/or communities of color. This could include differences in local resources provided to schools or built environment investment, such as safe streets and sidewalks, parks and green space, or incentives to encourage farmer’s markets or fresh produce in corner stores.[212] In exploring and remedying inequitable community investments, local governments should also consider the unintended consequences and develop preventative solutions to protect against gentrification that often occurs when historically disadvantaged communities experience new investment.[213] Potential strategies could include efforts to involve community voice and preserve home ownership, such as community benefit agreements between developers and community groups or coalitions or reducing or freezing property taxes for long-term residents.[212,214]

Limitations

Census tracts are frequently used as approximations for neighborhood boundaries because they are previously defined units that have readily available and large amounts of demographic, health, and built environment data. However, census tracts may suffer from the modifiable areal unit problem (MAUP) or Uncertain Geographic Problem (UCGoP), which occurs when individuals are aggregated into population counts within areal units that do not necessarily have an underlying mean such as a neighborhood.[21] Future studies should consider spatial measures of residential segregation that define neighborhoods around specific points such as home address, an example of which is Reardon et al.'s spatially continuous kernel densities.[172,203]

Mediation analysis is also not without its limitations. Frequently cited concerns include failure to account for unmeasured confounding, selection bias, model misspecification, and missing data.[180,215] To address these limitations, this study included multiple parallel mediators that address a broad range of social determinants of health, as well as relevant individual-level covariates. However, individual or family level estimate of SES was not available as part of the datasharing agreement with the local school district and is an important potential confounder in this study, given the well-documented relationship between poverty and youth obesity.[83,154] This important individual-level covariate could provide one potential explanation for why models including all study variables did explain a large proportion of the variability in youth BMI z-score and as well as why the amount of variance explained by neighborhoods using the clustering variable of census tracts was quite small (i.e. approximately 0.5%).

Another major limitation of this study is that it used cross sectional data, which limits the ability to draw inferences about causality in the relationship between residential segregation and youth obesity, as well as the effect of potential mediators on the association.[23] In addition to residential segregation emerging through multiple mechanisms that can have differential impacts (i.e., protective vs harmful), previous research has identified exposure over time as a potential explanation for variations in associations between residential segregation and obesity by race/ethnicity and sex.[51,55] For example, Pool et al. found that Black women who lived in highly segregated neighborhoods were 30% more likely to be obese at follow-up compared to Black women who lived in low to medium segregated neighborhoods.[51] Future mediation analysis should consider the application of repeated measures designs using programs such as MLMED that was developed to for SPSS to accommodate these multi-level designs and account dose effect response.[216]

As previously mentioned, the mediation analyses used in this study did not account for cross-classified nature of data. Multi-mediation programs such as the aforementioned MLMED for SPSS have been developed to handle hierarchical data.[216] The available multi-level mediation macros and programs that have been developed and are currently available did not accommodate the 2-2-1 structure of the study data (i.e., both the predictor and mediator were at level two, or the neighborhood level, while the outcome of interest was at the level one or individual level).[218] Therefore, application of multi-level mediation analysis for the current study data would require additions to current programs for mediation using Hierarchical Linear Modeling or the use of Structural Equation Modeling.[216]

Conclusions

Despite these limitations, this study has several strengths. Few studies have examined the relationship between residential segregation and youth obesity. Further, this study is the first to our knowledge that explores the mediating effect of neighborhood environments on the association between residential segregation and youth obesity. Given the limited amount of research exploring the relationship between residential segregation and youth obesity along with the mechanisms, both direct and indirect, that influence the association, significantly more studies are needed in this area. Building upon the strengths of the current study, this research should consider 1) nationally representative samples at units smaller than the county level, 2) longitudinal measures to bolster causality and explore how time and dose affect the association, and 3) the addition of individual/family level controls and other important confounders to strengthen the inferences made about potential neighborhood environment mediators. Lastly, even though the study was not greatly influenced by clustering of subjects across neighborhoods (i.e., multi-level), future research should consider application of multi-level linear regression modeling or structural equation modeling in the exploration of potential mediators in the association between residential segregation and youth obesity.

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Table 5.1 Childhood opportunity index component indicators

Domain	Component Indicator	Operational Definition
Social and Economic	***Economic opportunities***	
	Employment rate	Percent adults ages 25-54 who are employed
	Commute duration	Percent workers commuting more than one hour one way
	Economic and social resources	
	Poverty rate	Percent individuals living in households with incomes below 100% of the FPL
	Public assistance rate	Percent households receiving cash public assistance or Food Stamps/SNAP
	Homeownership rate	Percent owner-occupied housing units
	High-skill employment	Percent individuals 16+ in management, business, financial, computer, engineering, science, education, legal, community service, health, health tech, arts and media occupations
	Median household income	Median income of all households
	Single-headed households	Percent family households that are single parent headed
Health and Built Environment	***Healthy Environments***	
	Access to healthy food	Percent households without a car located further than a half-mile from the nearest super-market
	Access to green space	Percent impenetrable surface areas such as rooftops, roads, or parking lots
	Walkability	EPA Walkability Inex
	Housing vacancy rate	Percent housing units that are vacant
	Toxic Exposures	
	Hazardous waste dump sites	Average number of Superfund sites within a 2-mie radius
	Industrial pollutants	Index of tox chemicals released by industrial facilities
	Airborne microparticles	Mean estimated microparticle concentration
	Ozone concentration	Mean estimated 8-hour average ozone concentration
	Extreme heat exposure	Summer days with maximum temperature above 90F
	Health Resources	
	Health insurance coverage	Percent individuals 0-64 with health insurance coverage
	Early Childhood Education	
	ECE Centers	Number of ECE centers within a 5-mile radius
	High Quality ECE Centers	Number of NAEYC accredited centers within a 5-mile radius
	ECE enrollment	Percent 3- and 4-year-olds enrolled in nursery school, preschool, or kindergarten
	Elementary Education	Percent 3rd graders proficient on standardized reading tests, converted to NAEP scale

Education Environment	Third grade reading proficiency	Percent 3rd graders proficient on standardized math tests, converted to NAEP scale
	Third grade math proficiency	
	Secondary and postsecondary education	
	High school graduation rate	Percent ninth graders graduating from high school on time
	Advanced Placement course enrollment	Ratio of students enrolled in at least one AP course to the number of 11th and 12th graders
	College enrollment in nearby institutions	Percent 18–24-year-olds enrolled in college within 25-mile radius
	Educational and social resources	
	School Poverty	Percent students in elementary schools eligible for free or reduced-price lunches, reversed
	Teacher experience	Percent teachers in their first and second year, reversed
	Adult Educational Attainment	Percent adults aged 25 and over with a college degree or higher

Table 5.2 Descriptive statistics of youth, school, and neighborhood characteristics

	Percentage	Frequency	
	Youth Characteristics (n=11364)		
Biological Sex			
Male	51.6%	5866	
Female	48.4%	5498	
Race/ethnicity			
White (ref)	61.3%	6970	
Black	23.2%	2640	
Hispanic	15.4%	1754	
Grade			
Second	36.6%	4163	
Fifth	39.2%	4453	
Eighth	13.2%	1494	
Ninth	11.0%	1254	
Youth Weight Status			
Normal	66.9%	7601	
Overweight	17.8%	2020	
Obese	15.3%	1743	
	M (SD)	**Minimum**	**Maximum**
	Youth Characteristics (n=11,364)		
Youth BMI z-score	0.48 (1.16)	-5.59	3.052
Youth BMI percentile	62.99 (30.20)	1.15	99.89
	Neighborhood Characteristics (n=111)		
Black LQ	1.04 (0.89)	0	4.69
Hispanic LQ	1.03 (0.91)	0	4.32
COI Social and Econ Env	0.06 (0.18)	-0.49	0.34
COI Health and Built Env	-0.001 (0.05)	-0.16	0.06
COI Education Env.	0.03 (0.06)	-0.10	-0.14

Table 5.3 Multiple mediating model of social and economic environments, health and built environments, and education environments on association between Black residential segregation and BMI z-score among Black youth (n=9,610)

	Consequent											
	M_1 (Social/Econ Env.)			**M_2 (Health/Built Env.)**			**M_3 (Education Env.)**			**Y (BMI z-score)**		
Antecedent	Coeff	*SE*	*95% CL*	Coeff	*SE*	*95% CL*	Coeff.	*SE*	*95% CL*	Coeff.	*SE*	*95% CL*
X (Black LQ)	**-0.114[a]**	**0.002**	**-0.117, -0.110**	**-0.026[b]**	**0.000**	**-0.027, -0.025**	**-0.030[c]**	**0.001**	**-0.027, -0.025**	-0.180	0.018	-0.053, 0.018
M_1 (Soc/Econ)	--	--	--	--	--	--	--	--	--	**-0.316[d]**	**0.143**	**-0.595, -0.036**
M_2 (Hlth/Built)	--	--	--	--	--	--	--	--	--	0.585[e]	0.507	-0.408, 1.579
M_3 (Education)	--	--	--	--	--	--	--	--	--	**-2.069[f]**	**0.267**	**-2.592, -1.546**
Constant	**0.0780**	**0.003**	**0.075, 0.085**	**0.005**	**0.001**	**0.004, 0.006**	**0.039**	**0.001**	**0.036, 0.041**	**0.303**	**0.026**	**0.253, 0.354**
	R^2=0.445 F (6, 9603) =1281.41, p<.0001			R^2=0.387 F (6, 9603)=1010.55, p<.0001			R^2= 0.505 F (6, 9603)=548.82, p<.0001			R^2=0.043 F (9, 9600)=47.78, p<.0001		

Bold values are confidence intervals that do not contain zero, Models adjusted for race/ethnicity (W=0, B=1), grade (2=0, 5=1, 8=2, 9=3), Sex (male=0, female=1)

[a]effect of residential segregation on social/econ environment, a_1

[b]effect of residential segregation on health/built environment, a_2

[c]effect of residential segregation on social/econ environment, a_3

[d]effect of social/econ env. on BMI through res. segregation, b_1

[e]effect of residential segregation on health/built environment, b_2

[f]effect of residential segregation on social/econ environment, b_3

Table 5.4 Regression coefficients for total and direct effects of social/economic environments, health/built environments, and education environments on youth BMI z-score

	Black Seg/Black Youth (n=9610)		**Black Seg/Hispanic Youth** (n=8724)		**Hispanic Seg/Black Youth** (n=9610)		**Hispanic Seg/Hispanic Youth** (n=8724)	
	Coeff (se)	95% CI	Coeff (se)	95% CI	Coeff (se)	95% CI	Coeff (se)	95% CI
Total Effect[a]	**0.065 (0.015)**	**0.015, 0.094**	**0.040 (0.019)**	**0.003, 0.077**	**0.078 (0.016)**	**0.047, 0.110**	**0.082 (0.015)**	**0.052, 0.111**
Direct Effect[b]	-0.018 (0.018)	-0.053, 0.018	**-0.078 (0.022)**	**-0.121, -0.034**	0.007 (0.018)	-0.028, 0.042	0.035 (0.019)	-0.003, 0.072
Total Ind. Effect[c]	**0.082 (0.011)**	**0.061, 0.105**	**0.118 (0.012)**	**0.094, 0.142**	**0.071 (0.009)**	**0.055, 0.088**	**0.047 (0.012)**	**0.023, 0.071**
Partial Ind. Effects[d]								
Social/ Econ	**0.036 (0.016)**	**0.004, 0.067**	**0.041 (0.018)**	**0.005, 0.077**	0.025 (0.013)	-0.000, 0.050	0.018 (0.016)	-0.013, 0.049
Health/ Built	-0.015 (0.013)	-0.041, 0.011	**-0.041 (0.012)**	**-0.064, -0.077**	-0.015 (0.011)	-0.037, 0.007	**-0.056 (0.015)**	**-0.084, -0.270**
Education	**0.062 (0.008)**	**0.046, 0.078**	**0.118 (0.013)**	**0.094, 0.143**	**0.061 (0.008)**	**0.045, 0.077**	**0.085 (0.010)**	**0.067, 0.104**

*Bold text indicates confidence interval does not contain zero

[a]Total effect of residential segregation on youth BMI z-score adjusting for individual controls (c)

[b]Direct effect of residential segregation on youth BMI z-score after the addition of mediators (c′)

[c]Total indirect effect residential segregation on youth BMI z-score transmitted through all mediators ($a_1b_1+a_2b_2+a_3b_3$)

[d]Partial indirect effect residential segregation on youth BMI z-score transmitted through individual mediators (a_1b_1, a_2b_2, a_3b_3)

Table 5.5 Multiple mediating model of social and economic environments, health and built environments, and education environments on association between Black residential segregation and BMI z-score among Hispanic youth (n=8724)

	Consequent											
	M_1 (Social/Econ Env.)			**M_2 (Health/Built Env.)**			**M_3 (Education Env.)**			**Y (BMI z-score)**		
Antecedent	Coeff	*SE*	*95% CL*	Coeff	*SE*	*95% CL*	Coeff.	*SE*	*95% CL*	Coeff.	*SE*	*95% CL*
X (Black LQ)	**-0.120[a]**	**0.000**	**-0.124, -0.116**	**-0.025**	**0.001**	**-0.026, -0.023**	**-0.041[c]**	**0.001**	**-0.043, -0.040**	**-0.078**	**0.022**	**-0.121, -0.034**
M_1 (Soc/Econ)	--	--	--	--	--	--	--	--	--	**-0.338[d]**	**0.150**	**-0.121, -0.043**
M_2 (Hlth/Built)	--	--	--	--	--	--	--	--	--	**1.667[e]**	**0.472**	**0.742, 2.591**
M_3 (Education)	--	--	--	--	--	--	--	--	--	**-2.860[f]**	**0.301**	**-3.451, -2.270**
Constant	**0.800**	**0.002**	**0.075, 0.086**	**0.006**	**0.001**	**0.004, 0.008**	**0.035**	**0.001**	**0.033, 0.037**	**0.035**	**0.026**	**0.278, 0.382**
	R^2=0.404			R^2=0.300			R^2= 0.312			R^2=0.055		
	F (6, 8718) =984.20, p<.0001			F (6, 8717) =615.14, p<.0001			F (6, 8717)= p<.0001			F (6, 8714) =, p<.0001		

Bold values are confidence intervals that do not contain zero, Models adjusted for race/ethnicity (W=0, H=1), grade (2=0, 5=1, 8=2, 9=3)

[a]effect of residential segregation on social/econ environment, a_1
[b]effect of residential segregation on health/built environment, a_2
[c]effect of residential segregation on social/econ environment, a_3
[d]effect of social/econ env. on BMI through res. segregation, b_1
[e]effect of residential segregation on health/built environment, b_2
[f]effect of residential segregation on social/econ environment, b_3

Table 5.6 Multiple mediating model of social and economic environments, health and built environments, and education environments on association between Hispanic residential segregation and BMI z-score among Black youth (n=9,610)

	Consequent											
	M_1 (Social/Econ Env.)			**M_2 (Health/Built Env.)**			**M_3 (Education Env.)**			**Y (BMI z-score)**		
Antecedent	Coeff	*SE*	*95% CL*	Coeff	*SE*	*95% CL*	Coeff	*SE*	*95% CL*	Coeff.	*SE*	*95% CL*
X (Hispanic LQ)	**-0.092[a]**	**0.003**	**0.088, 0.100**	**-0.022[b]**	**0.001**	**-0.023, -0.021**	**-0.030[c]**	**0.001**	**-0.031, -0.028**	0.007	0.018	-0.028, 0.042
M_1 (Soc/Econ)	--	--	--	--	--	--	--	--	--	**-0.276[d]**	**0.139**	**-0.549, -0.003**
M_2 (Hlth/Built)	--	--	--	--	--	--	--	--	--	0.681[e]	0.506	-0.311, 1.674
M_3 (Education)	--	--	--	--	--	--	--	--	--	**-2.039[f]**	**0.269**	**-2.566, -1.511**
Constant	**0.094**	**0.003**	**0.088, 0.100**	**0.008**	**0.000**	**0.007, 0.009**	**0.041**	**0.001**	**0.039, 0.043**	**0.303**	**0.026**	**0.253, 0.354**
	R^2=0.326			R^2=0.297			R^2= 0.233			R^2=0.043		
	F (6, 9603) =, p<.0001			F (6, 9603)=, p<.0001			F (6, 9603)= p<.0001			F (9, 9600)=, p<.0001		

Bold values are confidence intervals that do not contain zero, Models adjusted for race/ethnicity (W=0, B=1), grade (2=0, 5=1, 8=2, 9=3), biological sex (male=0, female=1)

[a]effect of residential segregation on social/econ environment, a_1

[b]effect of residential segregation on health/built environment, a_2

[c]effect of residential segregation on social/econ environment, a_3

[d]effect of social/econ env. on BMI through res. segregation, b_1

[e]effect of residential segregation on health/built environment, b_2

[f]effect of residential segregation on social/econ environment, b_3

Table 5.7 Multiple mediating model of social and economic environments, health and built environments, and education environments on association between Hispanic residential segregation and BMI z-score among Hispanic youth (n=8724)

	Consequent											
	M_1 (Social/Econ Env.)			**M_2 (Health/Built Env.)**			**M_3 (Education Env.)**			**Y (BMI z-score)**		
Antecedent	Coeff	*SE*	*95% CL*	Coeff	*SE*	*95% CL*	Coeff.	*SE*	*95% CL*	Coeff.	*SE*	*95% CL*
X (Hispanic LQ)	**-0.105[a]**	**0.002**	**-0.109,-0.102**	**-0.029[b]**	**0.000**	**-0.030, -0.028**	**-0.032[c]**	**0.001**	**-0.033, -0.030**	0.035	0.019	-0.003, 0.072
M_1 (Soc/Econ)	--	--	--	--	--	--	--	--	--	-0.168[d]	0.148	-0.459, 0.122
M_2 (Hlth/Built)	--	--	--	--	--	--	--	--	--	**1.916[e]**	**0.500**	**0.948, 2.883**
M_3 (Education)	--	--	--	--	--	--	--	--	--	**-2.681[f]**	**0.300**	**-3.267, -2.096**
Constant	**0.091**	**0.003**	**0.085, 0.096**	**0.006**	**0.001**	**0.005, 0.008**	**0.040**	**0.001**	**0.038, 0.042**	**0.330**	**0.026**	**0.278, 0.381**
	R^2=0.447			R^2=0.454			R^2= 0.300			R^2=0.054		
	F (6, 8717) =1171.81, p<.0001			F (6, 8717)=1207.87, p<.0001			F (6, 8717)=621.54, p<.0001			F (9, 8714)=55.00, p<.0001.		

Bold values are confidence intervals that do not contain zero, Models adjusted for race/ethnicity (W=0, H=1), grade (2=0, 5=1, 8=2, 9=3)

[a]effect of residential segregation on social/econ environment, a_1

[b]effect of residential segregation on health/built environment, a_2

[c]effect of residential segregation on social/econ environment, a_3

[d]effect of social/econ env. on BMI through res. segregation, b_1

[e]effect of residential segregation on health/built environment, b_2

[f]effect of residential segregation on social/econ environment, b_3

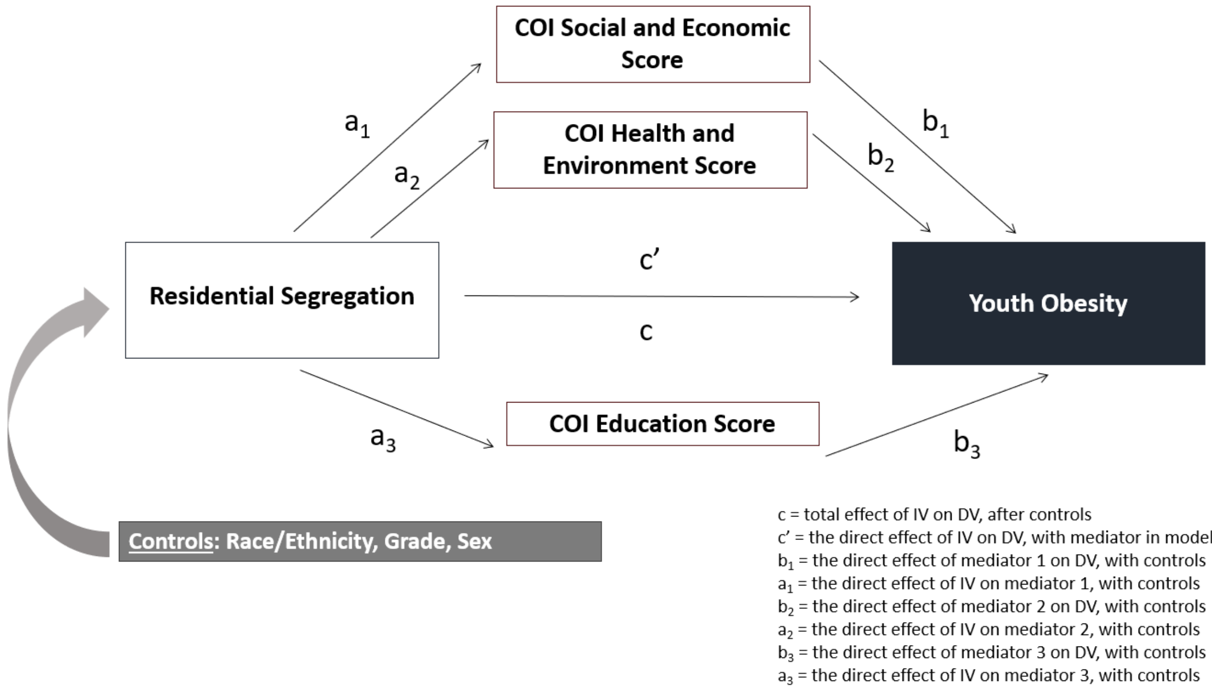

Figure 5.1 Mediation pathway of neighborhood social and physical environment on the association between residential segregation and youth obesity

CHAPTER SIX

DISCUSSION

This chapter provides a synthesis of major findings across the study aims, study strengths, and study limitations, as well as public health implications and directions for future research.

5.1 Summary of Major Findings

Beginning in the 1980s, youth obesity prevalence rates in the US began rising rapidly, almost tripling by the first decade of the 21st century.[1,4,68] Overall, youth obesity prevalence showed initial signs of plateauing in the early 2000s and even decreasing in some populations; however, the COVID-19 pandemic has renewed global and national concerns about the health consequences of youth obesity.[2,68,182] Preliminary evidence has shown that not only has the COVID-19 pandemic increased youth obesity prevalence, but it has reinforced existing disparities by socioeconomic status and race/ethnicity, with the trajectory of youth obesity increasing most rapidly among youth who are already overweight or obese.[182]

Neighborhoods are important settings where youth obesity behaviors are shaped through socioeconomic and built environments that can reinforce or prevent healthy lifestyle behaviors among youth and families.[23,174] Grounded in the social ecological model of youth obesity, significant research in recent decades has explored system and environmental determinants of youth obesity, including access to healthy foods, parks and green space access, complete streets and sidewalks, neighborhood poverty and education, social cohesion, etc.[12,174] Despite the breadth and depth of neighborhood youth obesity research, few studies have examined the association between residential segregation by race/ethnicity and youth obesity, or its intersectionality with school segregation.[40,56]

To address this important gap in the literature, this study explored two major aims. The first aim used cross-classified multilevel models (CCMM) to: 1) determine the relative influence of school and neighborhood environments on youth obesity, and 2) examine associations between school and neighborhood residential segregation with youth obesity. The second aim explored whether neighborhood social/economic environments, health/built environments, and education environments mediated the association between residential segregation and youth obesity. Aim 2 also explored if the significance of the mediators differed by type of residential segregation (Black versus Hispanic) and youth race/ethnicity (Black versus Hispanic).

Summary of Findings: Aim 1

Using a series of CCMMs, the first aim of this study explored the intersection of school and neighborhood segregation and their influences on youth obesity risk, after controlling for individual youth characteristics. ICCs, calculated from the unconditional model, found that about 3.6% of the variability in youth BMI z-scores could be attributed to schools, 1.9% could be attributed to neighborhoods, and 5.5% could be attributed to schools and neighborhoods combined. Results indicated that school and neighborhood segregation were associated with youth obesity, where school segregation was negatively associated with youth BMI z-score, and residential segregation was positively associated with youth BMI z-score. The association between segregation and youth BMI z-score was stronger for Hispanic school and residential segregation, when compared to Black school and residential segregation. Furthermore, in the best fitting model (Model 6), Black school segregation was no longer significant after the addition of an interaction term between Hispanic school segregation and individual youth race/ethnicity.

The addition of two interaction terms (i.e., Hispanic school segregation and youth race/ethnicity, Hispanic residential segregation and youth race/ethnicity) suggested that individual race/ethnicity may moderate the associations between both Hispanic school and neighborhood segregation with youth obesity risk. However, while significant, the addition of the interaction term for Hispanic residential segregation did not improve model fit, when compared to the combined schools and neighborhood model (Model 5). Interaction terms between Black school or Black residential segregation with youth race/ethnicity were not included in the final, best fitting models as they were not statistically significant and did not improve model fit.

The amount of research exploring the association of residential segregation youth obesity, in particular, is extremely limited, but there is a larger and growing body of research that has explored associations with adult obesity.[56,57,59] These studies have found differences in the strength and significance of associations by race/ethnicity, with the strongest evidence for positive associations between Black segregation and obesity among Black individuals.[40,51] Research exploring the associations between Hispanic residential segregation and obesity among Hispanic adults has found positive, null, and even negative (i.e., protective factor) associations.[150] For example, Kershaw et al. found that living in higher Hispanic-White residential segregation decreased the odds of obesity among Mexican American women.[55] The current study found that Hispanic residential segregation was associated with increased obesity risk for both Black and Hispanic youth. Similarly, Ryabov found the odds of having obesity was greater for Black youth living in higher Black-White segregation and for Hispanic youth living in higher Hispanic-White segregation, when compared with their White peers.[56]

There is a significant body of research about the relationship between school racial composition and academic achievement, educational attainment, and economic mobility, through the mechanisms of school quality and poverty.[41,189,190] Less research has explored the relationship between school racial composition and health outcomes, including youth obesity, and few studies have used measures of school segregation instead of minority composition.[184] One study examining the associations of both school poverty and segregation with youth obesity found that, after controlling for Black and Hispanic residential segregation, youth in minority segregated schools were more likely to be obese, but the relationship was not significant after controlling for school-level poverty.[57] In contrast, this study found that the associations between Black and Hispanic school segregation and youth obesity were significant and negative, after controlling for school poverty and residential segregation. The only exception was Black school segregation, after the addition of an interaction term between Hispanic segregation and youth race/ethnicity. One potential explanation for the differences in findings is that this study used a true measure of segregation (LQ) for schools and neighborhoods, while the study conducted by Piontak et al. used a categorical measure derived from percent minority students attending the school.[57,59]

Summary of Findings: Aim 2

Parallel mediation analysis was used in aim 2 to determine if, and to what extent, neighborhood social/economic environments, health/built environments, and education environments mediated the association between residential segregation and youth obesity, after controlling for individual youth characteristics. Additionally, this aim explored if the strength and significance of the hypothesized mediators varied at the intersection of Black versus Hispanic segregation and among Black versus Hispanic youth. Results of the parallel mediation analyses showed that residential segregation indirectly affects youth BMI z-score through three different neighborhood environments: the social/economic environment, the health/built environment, and the education environment, even after controlling for individual youth characteristics. The significance of the three neighborhood environments as mediators varied by the four combinations of Black or Hispanic segregation with individual race/ethnicity (Black or Hispanic). The direct effect of residential segregation was only significant for Black segregation among Hispanic youth, after the addition of the three parallel mediators. When examining the partial indirect effects of the three mediators, only the education environment was significant for all combinations of Black or Hispanic residential segregation with individual youth race/ethnicity. The partial indirect effects of the social/economic environment were significant for Black segregation among Black and Hispanic youth but were not significant for Hispanic segregation among Black or Hispanic youth. The partial indirect effects of the health/built environment were significant for both Black and Hispanic segregation, but only among Hispanic youth.

While many studies have explored the role of neighborhood social and built environments in the association between residential segregation and health, few studies have examined youth obesity as the outcome of interest.[30,55,58,217] Goodman et al. examined the role of healthy eating behaviors and environments as mediators of the association between residential segregation and adult obesity at both the county and census tract level.[58] The retail food environment at the census tract level was a significant mediator of the association between residential segregation and adult BMI, but the mediating effect was not significant for county-level food environments. In combination with the current study's findings, these results suggest that local environments, when compared to cities or counties, may play a greater role in influencing access to opportunities and behaviors that encourage healthy weights. In support, Kreiger et al. reached a similar conclusion about county versus census tract level effects, finding that there was a stronger association between residential segregation and assaults at the census tract level compared to the county level.[163]

6.2 Study Strengths

One clear contribution of the present research is that it adds to the extremely limited evidence on the relationship between residential segregation and youth obesity. In a review of the literature, only a handful of related studies were found, and only one explored the association between residential segregation and youth obesity as the focal relationship.[56,57,59] This one study found that as residential segregation increased, youth obesity increased, and residential segregation explained up to 20% of the differences in the prevalence of overweight, obesity, and obesity-related chronic disease between Black and Hispanic children, compared to their White peers.[56] Further, given the limited amount of research exploring school segregation and health and that the majority of studies have used racial composition as a proxy for segregation, this study provides an important contribution to better understanding how school segregation influences youth obesity risk, as well as how the effects of school and residential segregation may intersect.

Another strength of this study was the use of CCMMs in aim 1 that allowed for the simultaneous inclusion of youth who were cross classified across schools and neighborhoods. While the variation in youth obesity that was attributable to schools and neighborhoods was small, previous research comparing single level regression and hierarchical multi-level models with CCMMs found that the application of CCMMs decreases potential violations of assumptions and errors, including power to detect treatment or covariant effects, less accurate estimates of variances, inflated Type I errors, and errors in interpreting tests for statistical significance.[174]

Several other study strengths are related to the sample. The first is the large sample size that included data collected by trained physical education teachers for more than 11,000 youth from 111 census tracts and 80 different elementary, middle, or high schools across the county. Specifically, the sample included a robust number of level-2 units across both schools and neighborhoods and a large overall sample size that was reflective of the county racial/ethnic composition. Additionally, the level-1 units used in this study are census tracts and not counties, as the latter is the case for many studies examining the association between residential segregation. Using census tracts as the local unit of analysis allowed for a better understanding of the variability within communities, rather than variability across counties. This distinction may be important as

Acevedo-Garcia et al. found that 90% of the variation in childhood neighborhood opportunity occurs within metropolitan areas, rather than across metropolitan areas.[149] Similarly, in a multi-level study of residential segregation across communities in the US, Arcaya et al. found that 80% of the variability in residential segregation occurred within MSAs rather than across MSAs.[161]

Another strength is that this study used the LQ, a localized measure of residential segregation that accounts for under or over-representation at the census tract level.[21,172] The evidence base on the association between residential segregation and obesity has suffered from methodological concerns, including both variability and quality of measures, making comparison across studies difficult.[21,36,40] Many studies exploring the associations between residential segregation and obesity have used racial composition as a proxy for residential segregation.[21,40] For example, in a 2015 critical review of the evidence for the relationship between residential segregation and adult obesity among Black Americans, six out of eleven of the identified studies used percent Black/African American as the measure of residential segregation.[40] This is problematic given that residential segregation scholars have demonstrated that high or low minority composition does not always align with validated measures of residential segregation.[126]

6.3 Study Limitations

While the findings from the two study aims have several strengths, there are some limitations worth noting. One of the first limitations is the cross-sectional nature of the data, which limits the ability to draw causal inferences about the association between residential segregation and youth obesity.[23,51] Therefore, future research should consider longitudinal data and measures that allow for more most robust causal inferences, including exploration of how timing of and duration of exposure impact youth obesity risk and burden over the life course.

This study could also have limitations related to the modifiable areal unit problem (MAUP) or Uncertain Geographic Problem (UCGoP), which occurs when census tracts are somewhat arbitrarily defined and may not represent actual neighborhoods to which residents identify as belonging to.[21,22] Still, census tracts are frequently used as neighborhood proxies given the large amounts of data that are readily available through sources such as the US Census Bureau.[21,172] Similar to the MAUP, one limitation of the LQ is that while it considers each smaller areal unit (census tract) in comparison to the broader population distribution (county), it does not consider clustering of units across the broader regional area.[218] Future studies should consider using and contrasting spatial measures of residential segregation, such as Reardon et al.'s spatially continuous kernel densities that create and define neighborhoods as a point in space around a location like home address.[21,172]

Mediation provides an important analytic tool to better understand the mechanisms by which neighborhood characteristics can influence youth obesity as well as the corresponding levers to create healthier community environments.[180] However, there are potential limitations and sources of error that should be carefully considered when interpreting mediation analyses, including failure to account for unmeasured confounding, selection bias, model misspecification, and missing data.[180,215] In response to these concerns, this study included three parallel mediators that encompassed a broad range of neighborhood environment characteristics. One potential confounder not available in the study dataset was an adequate control for individual SES, which is a well-documented predictor of youth obesity.[68,69] In addition to acting as a potential confounder of the mediation analyses in aim 2, lack of individual SES as a study covariate could impact the results of the multi-level analyses in aim 1, particularly in cases where there is significant amount of income incongruity with the neighborhood mean. Future studies should consider leveraging datasets that contain measures of individual SES.

6.4 Public Health Implications and Future Research

This research was designed to be applied in nature, in partnership with a community coalition working to create equitable access to healthy eating and active living for all residents through policy, systems, and environment change, with the goal of reducing youth obesity prevalence. Findings from this study will be shared with the coalition to support advocacy for equitable neighborhood environments with local government officials and community stakeholders. This research will add to the coalition's growing work around community power building that considers the historical and contemporary determinants within community systems and government that drive disparities in youth obesity by race/ethnicity and SES. Findings from this study will also be used, in combination with existing maps of census tract level youth obesity, access to physical activity opportunities (parks, trails, walkability), and food security risk, to help identify priority neighborhoods where children and families of color live at the intersection of segregation and limited access to healthy eating and active living opportunities.

One important finding in the current study that has implications for future research was that the association between residential segregation and youth obesity varied by type of segregation (Black versus Hispanic) and individual youth race/ethnicity

(Black versus White). It is well understood that residential segregation does not emerge through one mechanism and, therefore, has different implications for health and wellbeing that intersect with both individual race/ethnicity and the historical and contemporary racial dynamics of communities.[21,38] Despite this recognition, many of the studies examining the association of residential segregation have examined Black-White and Hispanic-White segregation, separately, and only in congruence with individual race/ethnicity (i.e., Black-White segregation among Black individuals).[40,55,56] Future research is needed that explores the interaction between type of racial residential segregation and individual race/ethnicity. This includes examining Black-White and Hispanic-White segregation individually and in combination, as well as the effects of individual race/ethnicity on youth obesity risk across these types of racial residential segregation (congruence versus incongruence).

This study provided an important contribution to the field of neighborhood research by examining the role of social/economic, health/built, and education environments as potential mediators of the association between residential segregation and youth obesity. While the effects of the mediators varied by Black versus Hispanic segregation and among Black and Hispanic youth, each of the three environments were significant in at least one of the four combinations of residential segregation by race/ethnicity and individual youth race/ethnicity. These findings have important policy and practice implications for local, state, and federal government. For example, government officials and stakeholders should consider how infrastructure investments in neighborhood environments (i.e., physical activity and nutrition built environments) or resources/opportunities (i.e., high quality childcare or schools) could alleviate the impacts of residential segregation, as well as how biased infrastructure investments sustain the intersection of neighborhood deprivation and residential segregation.[212,213]

While this study leveraged a large and robust dataset from within one large county in the southeastern US, it may limit generalizability to other areas that differ by population density, racial composition, urbanicity, historical racial/ethnic dynamics, and so on. Residential segregation is a complex and changing system that includes both historical and contemporary processes; therefore, both the mechanisms by which segregation emerge and how they impact youth obesity and other health outcomes may differ by city, state, or region.[38,125] For example, the Jim Crow era laws may have created different patterns of segregation and limited opportunity in rural and suburban communities across the southern states, compared to the segregation that emerged in large urban northeastern cities with the growth of manufacturing and immigration.[38] Both of these historically rooted mechanisms have lasting implications for current generations, but are also continuing to shift and change in unique ways (i.e., growing rates of new Latinx populations in some southern states compared to well established and large communities across other regions of the US).[33,150,194] Therefore, future studies should consider not only nationally representative samples to better understand the overall impacts of youth segregation, but also consider historical and emerging patterns of residential segregation that are unique to region and population demographics.

6.5 Conclusions

Overall, this study provides an important contribution to the literature on how the dynamics of segregation can vary by how we define and measure residential segregation (Black versus White, Hispanic versus White, Minority versus White, etc.), by context (school versus neighborhood environments), and by individual youth race/ethnicity. These complex dynamics necessitate further exploration of this poorly studied research area and careful consideration of potential policy and built environment solutions to support healthy behaviors for youth and their families, including protective versus health-harming impacts and their interplay across forms of segregation, context of segregation, and individual race/ethnicity.

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