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Not So Black and White: The Racial/Ethnic Structure of School Environments and Cardiometabolic and Reproductive Health across the Life Course

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By

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Master of Public Health

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An abstract of
A dissertation submitted to the Faculty of the
James T. Laney School of Graduate Studies of Emory University
in partial fulfillment of the requirements for the degree of
Doctor of Philosophy in Epidemiology
2020

Abstract

Not So Black and White: The Racial/Ethnic Structure of School Environments and Cardiometabolic and Reproductive Health across the Life Course
By Kya N. Grooms

Background: Given the public health burden of adverse cardiometabolic and reproductive outcomes, it is critical to investigate the social determinants of that occur in adolescence and childhood. We sought to investigate the association between the racial structure of schools and three connected outcomes: adolescent obesity (*Aim 1*), inflammation in early adulthood (*Aim 2*), and experiences of preterm birth (PTB) (*Aim 3*).
Methods: We utilized data from the National Study of Adolescent to Adult Health. School racial composition was assessed using the proportion of Black/non-White students, and racial diversity. District-level segregation was assessed using the Black-White dissimilarity and exposure indices. Adolescent obesity was categorized as not overweight/obese (< 85th percentile) and overweight/obese (≥ 85th percentile). Inflammation was classified using two categories of high-sensitivity C-reactive protein concentrations: (1) low/average: ≤ 3 mg/L, and (2) high: 3-10 mg/L. PTBs were defined as any birth that occurred prior to 37 gestational weeks. The predicted prevalences of the outcomes were estimated from cluster- and sample-weight adjusted logistic regression models. Models were stratified by race/ethnicity and adjusted for school-, neighborhood-, and individual-level characteristics.
Results: In Aim 1, Asian/Native American/Other adolescents who attended schools with 26-50% Black students were 2.11 times more likely to be overweight/obese (95% CI: 1.08, 4.15) than those who attended schools with 0-10% Black students. Non-Hispanic Black adolescents who attended schools with 0-10% non-White students were 1.83 times more likely to be overweight/obese (95% CI: 1.07, 3.11) than those who attended schools with 0-10% non-White students. In Aim 2, there were no meaningful associations observed between school environments and inflammation. In Aim 3, Hispanic mothers were 0.05 times as likely (95% CI: 0.00, 0.56) to experience a PTB for every one-unit change (0-100%) in the proportion of Black students in a school.
Conclusion: There were racial/ethnic differences in the types of schools attended that U.S. adolescents attended. The relationships between the racial structure of school environments and cardiometabolic and reproductive health across the life course also varied by race/ethnicity.

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Acknowledgements

I would like to acknowledge the following people who were instrumental in the completion of this dissertation and my PhD: Shakira Suglia, ScD, MS; Michael Kramer, PhD, MS; Tomeka Davis, PhD; Jena Black; and Julie Gazmararian. This dissertation would not have been possible without the following funding sources: Laney Graduate School; Emory Graduate Diversity Fellowship; HRSA Maternal and Child Health Epidemiology PhD Training Award (T03MC07651); and NIH T32 Reproductive, Pediatric, and Perinatal Pre-Doctoral Training Award (T32D052460).

Table of Contents

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This dissertation is dedicated to my two amazing grandmothers – Eva S. Morton and Lillie S. Grooms – who passed away during my doctoral program. Both women attended segregated schools in the South, and achieved educational accomplishments above and beyond their parents' imaginations. They have been my constant inspiration throughout this doctoral program, as well as throughout my entire life. I have achieved this for both of them.

Table of Contents

Not So Black and White: The Racial/Ethnic Structure of School Environments and Cardiometabolic and Reproductive Health across the Life Course

Chapter 1: Background & Significance

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SPECIFIC AIMS

Specific Aim 1: *Investigate the multilevel associations between the racial/ethnic structure of school environments in adolescence and the prevalence of adolescent obesity.*

Hypothesis 1a: Adolescents who attend schools with a higher proportion of Black students, higher proportion of non-White students, or with less racial/ethnic diversity will have an increased likelihood of adolescent obesity, after adjusting for individual-, school-, and neighborhood-level characteristics.

Hypothesis 1b: The associations between the racial/ethnic structure of school environments in adolescence and the prevalence of adolescent obesity will differ by an individual's race/ethnicity.

Specific Aim 2: *Examine the association between the racial/ethnic structure of school environments in adolescence and the risk of inflammation in early adulthood.*

Hypothesis 2a: Adolescents who attend schools with a higher proportion of black students, non-white students, less racial/ethnic diversity, and higher levels of racial segregation will have higher C-reactive protein (CRP) levels in early adulthood, even after adjusting for individual-, school-, and neighborhood-level characteristics.

Hypothesis 2b: The association between the racial/ethnic structure of school environments in adolescence and the risk of inflammation in early adulthood will differ by an individual's race/ethnicity.

Specific Aim 3: *Examine the association between school racial composition in adolescence and the risk of preterm birth (PTB) in early adulthood.*

Hypothesis 3a: Adolescents who attend schools with a higher proportion of black students, non-white students, or with less racial/ethnic diversity, and higher levels of racial segregation will have an increased risk of PTB in early adulthood, even after adjusting for individual-, school-, and neighborhood-level characteristics.

Hypothesis 3b: The association between the racial/ethnic structure of school environments in adolescence and the risk of PTB in early adulthood will differ by an individual's race/ethnicity.

BACKGROUND & SIGNIFICANCE

Cardiometabolic and Reproductive Outcomes: Finding Common Ground

The seemingly disparate collection of cardiometabolic and reproductive health outcomes under investigation in this dissertation may have their origins in early life/adolescence, and may share common etiologic pathways. Specifically, adolescent obesity, cardiometabolic risk, and experiences of preterm birth (PTB) are all linked to the dysregulation and disruption of inflammatory processes. Furthermore, cardiometabolic risk and adverse pregnancy outcomes represent a set of related outcomes that can inform us not only about the development of risk across an individual's life, but also how a contextual exposure can potentially impact population health across three connected periods of emerging adulthood.

Population Burden of Adolescent Obesity

The prevalence of obesity among United States (U.S.) adolescents, aged 12-19 years old, has increased in recent years[1], with approximately 20.6% of adolescents being obese[2-3]. Family history, health behaviors (e.g., physical activity and diet), and socioeconomic characteristics are all risk factors for obesity[4-6]. Being of black or Hispanic race/ethnicity or having a lower socioeconomic status (SES) are associated with a higher risk of adolescent obesity[2-4,6-11]. In addition, the prevalence of adolescent obesity varies by geographic area and school characteristics. Adolescents living in southern states are more likely to be obese than those living in other regions of the country[12]. Adolescents living in non-urban areas are also more likely to be obese than

those living in urban areas[2,5,13]. Finally, adolescent obesity is higher in schools with a higher proportion of students from racial/ethnic minorities and low-SES households[14].

Being obese can also have adverse health consequences during adolescence that carry on into adulthood. Compared to those who are normal weight, obese children and adolescents are more likely to be obese as adults and may have higher levels of inflammatory biomarkers, such as C-reactive protein and interleukin-6[9,12,15-19]. Obesity that occurs during childhood and adolescence can increase the risk of type 2 diabetes, high blood pressure, high cholesterol, cardiovascular disease, and mortality in adolescence and adulthood[1,4,9,15,17-18,20-,27]. Given these life course implications and that more than 1 in 5 U.S. adolescents are obese, identifying relevant socio-contextual exposures and underlying biosocial mechanisms are important in addressing this large public health burden.

Population Burden of Cardiometabolic Risk

Though there have been considerable declines in mortality due to advances in medical treatments and public health preventions[28], cardiovascular disease (CVD) is still the leading cause of death in the U.S.[3] Approximately 92.1 million U.S. adults, or about 33% of the country's population, are living with CVD. Known risk factors of CVD include, but are not limited to: hypertension, inflammation, smoking, a family history of CVD, being overweight/obese, lack of physical activity, and a poor diet[3,29]. Although CVD has decreased in recent years, racial/ethnic, geographic, and socioeconomic disparities still persist in the U.S. Blacks, persons with lower SES, and those who live in the South have an increased cardiovascular risk[30-38]. Also, adverse are-level

characteristics, such as low neighborhood SES, are associated with an increased risk of CVD[39-42].

Cardiometabolic risk can also accumulate across the life course. Given that overweight children are likely to become obese adults[6,15,17], they will have a higher cardiovascular risk since obesity plays a significant role in the development of CVD. It is thought that obesity is associated with heart disease through an increased insulin resistance experienced among obese persons; obesity results in cardiometabolic dysfunction[43]. Furthermore, since higher levels of inflammatory biomarkers are associated with a higher cardiometabolic risk[44-50], we will use inflammation in early adulthood as our second outcome in this proposed study.

Population Burden of Preterm Birth

Approximately 11.4% of U.S. infants were born preterm in 2013[51], which is relatively high compared to other developed nations[52-53]. Though PTBs, defined as an infant born < 37 gestational weeks[53], have decreased in the last decade due to the reduction of population-wide risk factors and the development of medical interventions and policies[54], the health implications of PTB span the entire life course.

Preterm delivery is often a result of infection or inflammation, hemorrhage, and maternal stress[53,55]. Demographic risk factors for PTB include: race/ethnicity, SES, maternal age, marital status, and maternal obesity, among others[53]. In fact, black infants are more likely to be born preterm than white infants[51,53,56-58]. Furthermore, there are also established socioeconomic disparities in PTB[59-63]. These socioeconomic differences may vary by maternal race/ethnicity, in that having a higher SES may be

more beneficial for the reproductive health of white women than black women[62-63]. Thus, investigating the social determinants of PTB will help to decrease this racial gap.

Similar to adolescent obesity and cardiometabolic risk, PTBs may also have adverse consequences spanning across the life course. Being born preterm is also associated with neonatal and infant mortality[53,60], as well as attention-deficit/hyperactivity disorder and other behavioral problems in childhood[64-66]. It has been theorized that the association between PTB and health in later life is a result of "fetal programming." In fact being born preterm can have a detrimental impact on the pathophysiology and structure of organs, tissues, and systems relevant to the development of disease in adulthood[67-69].

Inflammatory Pathways and Processes

Inflammation is an underlying physiologic process, which may begin in adolescence and have long-lasting implications for the three seemingly unrelated outcomes experienced across the life course – adolescent obesity, cardiometabolic dysfunction, and poor perinatal outcomes. Persistent and long-term external stressors, such as socio-contextual exposures, can have negative consequences for one's sympathetic nervous system and hypothalamic-pituitary-adrenal (HPA) axis. Repeated stress on the body may lead to biological "wear and tear" (i.e., allostatic load) and hyperactivity of adaptive responses, resulting in increases in cortisol levels and the disruption of other inflammatory processes. Though the activation of the HPA axis may have some protective value when facing acute stressors, the dysregulation of this system can prove to be detrimental. Constant activation, particularly from socioeconomic stressors, can result in increased allostatic load, which may accumulate across one's life

course and produce harmful chronic health outcomes[57,70-72]. More specifically, when the body is faced with an external stressor, the sympathetic nervous system and HPA axis are activated, causing catecholamines and cortisol to be released. However, if one's body stays in a heightened state or still perceives a stressor to be present, such as attending a disadvantaged school or living in a violent neighborhood, the body is unable to return to homeostasis. Persistent activation of this system results in increased levels of stress hormones (e.g., cortisol) and inflammatory biomarkers (e.g., C-reactive protein (CRP))[71-72].

CRP is a biomarker of systemic inflammation and is associated with an increased risk of CVD, stroke, and cardiovascular-related mortality[44-50]. It is thought that CRP can bind to low density lipoprotein and deposit in the walls of the arteries creating a buildup of plaque, thus playing a direct role in the development of atherosclerosis[73-77]. Elevated levels of CRP, defined as a CRP concentration > 3.0 mg/L, have also been found to be associated with an increased risk of type 2 diabetes among women[78]. Thus, elevated levels of inflammatory biomarkers, such as CRP, can have adverse effects on cardiometabolic health. The national prevalence of elevated CRP has decreased in recent years[48,79]. In fact, the proportion of U.S. adults living with elevated CRP levels has decreased from 36.7% to 32.9% between 1999 and 2010. However, in early adulthood, there still remains a large population burden, with the prevalence of elevated CRP levels among adults ages 20-29 and 30-39 years old at 26.7% and 30.2%, respectively, in 2010[80]. Additionally, compared to whites and adults with a high SES, blacks and those with a lower SES are more likely to have elevated levels of inflammatory biomarkers[80-83]. Adults with lower educational attainment are also more likely to have higher levels of CRP[84]. Given this relatively large burden among young

adults and the associated disparities, inflammation is a relevant proxy for cardiometabolic risk in Specific Aim 2 of this dissertation.

Stress and Inflammation

Higher levels of inflammatory biomarkers have been found to be associated with higher risks of obesity[9,18-19,21,85], CVD[46,49-50], and PTB[53,55,86] in adulthood. Due to its role in the development of atherosclerosis, inflammation can increase the risk of cardiometabolic outcomes[73-77]. In fact, it is believed that chronic social stressors can increase the risk of adolescent obesity by: (1) increasing cortisol secretion, which can result in an accumulation of fat and abdominal obesity; and (2) decreasing one's motivation for and participation in physical activity due to feelings of intense stress[87].

Regarding PTB, women who experience chronic stressors across the life course may not be able to regulate their inflammatory and endocrine responses when confronted with a prenatal stressor. This dysregulation due to psychological and social stress can result in increased levels of the corticotropin-releasing hormone produced by the placenta, increasing the risk of preterm delivery[55,57,59,88-89]. Furthermore, Geronimus' "weathering" hypothesis suggests that black women have worse birth outcomes than white women due to the declines in reproductive potential that results from social, economic, and political disadvantages and stressors experienced repeatedly across the life course[90].

From Segregation to Desegregation to Resegregation of U.S. Schools

Segregation

In the investigation of the impact of the racial/ethnic structure of school environments on population health across the life course, it is important to understand the historical context and evolution of school segregation in the U.S. In *Plessy v. Ferguson* (1896), the Supreme Court ruled that the separation of people by race in public places in the U.S. was legal. In fact, the Court reasoned that, as long as the separate facilities were equal, laws requiring blacks to utilize separate public facilities from whites were not unconstitutional. Though facilities for blacks were generally of lower quality than white facilities, the doctrine of "separate, but equal" was enacted into a federal law under *Plessy*. Until the Civil Rights Act of 1964, Jim Crow laws provided the legal basis for racial segregation in many domains of everyday life (e.g., schools, transportation, restaurants, etc.) in states across the country[91-92]. In fact, during this era of legal segregation, black schools were actually inherently unequal to white schools. Compared to black schools, white schools received more funding, offered more courses, and had smaller class sizes[93]. This era of "separate, but equal" produced inequitable educational opportunities for black students in the U.S.

Desegregation

Brown v. Board of Education of Topeka (1954) abolished *de jure* school segregation based on race in public schools and deemed the doctrine of "separate, but equal" to be unconstitutional based on its violation of basic human rights guaranteed under the 14th Amendment[94]. Though *Brown* was the landmark case in undermining a long-standing history of legal, racial segregation in American public schools, plans on how and when to desegregate were not detailed in this decision. Even after the Court decided on *Brown II* (1955), which stated that school systems should desegregate "with

all deliberate speed," implementation plans were still not explicitly defined[95]. Even by 1964, ten years after the *Brown* decision, a majority of black students in the South still attended majority-black schools[93]. School desegregation proved to be a slow process.

It was not until *Green v. County School Board of New Kent County* (1968) when specific guidelines were established to assist U.S. public schools in their desegregation efforts. The *Green* decision forced an end to "dual" (i.e., separate and segregated) school systems, in an effort to create a single, integrated school system in each district. Unitary school systems were what school districts should strive to accomplish and they should attempt to achieve desegregation across the following domains: facilities, staff, faculty, extracurricular activities, and transportation[96]. Encouraging unitary status would create racially integrated, or more diverse, schools that could have had beneficial effects. Students attending less segregated schools have higher levels of academic achievement, experience less racial discrimination, and have more college resources and opportunities[97].

In an effort to further enforce the desegregation of public schools in the South, *Swann v. Charlotte-Mecklenberg Board of Education* (1971) specified that schools must make reasonable efforts to desegregate, including through the use of busing[98]. However, busing was a potentially harmful exposure for children during this period of desegregation. Busing forced a child to leave one's neighborhood to go to an unfamiliar or "foreign" neighborhood. Though black students might have been exposed to better educational opportunities/resources and wider social networks through busing, the violent white opposition created safety concerns and increased experiences of heightened interracial contact and discrimination[93]. Finally, *Keyes v. Denver School District No. 1* (1973) proved to be the first Supreme Court case to encourage the creation

of desegregated schools in the North and West, where school desegregation plans had not been previously mandated by the courts[99].

Resegregation

Although court-ordered efforts were temporarily successful in reducing *de jure* school segregation, U.S. public schools began to resegregate in the late 1970s due to persistent residential segregation and a few, critical Supreme Court decisions that released school systems from their court-ordered mandates to integrate. These decisions effectively helped to unravel the successful desegregation policies that were put in place during the *Brown* era[93,100-102]. In fact, *Milliken v. Bradley* (1974) made school desegregation in northern metropolitan areas difficult by preventing school districts from drawing on majority white, suburban areas to integrate majority black, inner-city schools[103]. *Riddick v. School Board of the City of Norfolk, Virginia* (4th Cir. 1986), *Board of Education of Oklahoma v. Dowell* (1991), and *Freeman v. Pitts* (1992) allowed local governments to formally end desegregation efforts in southern school districts. Even if schools in those districts had not reached complete desegregation under *Green*'s guidelines, as long as the Court declared that they had "achieved" unitary status, the school districts were free from court-ordered desegregation mandates[104-106].

Most notably, these Court decisions did not take into account the long-standing effects of slavery and segregation that had been entrenched in our society and pervasive throughout our education system. As a result, the 1980s and 1990s saw an increase in the segregation of public schools in various regions across the U.S.[102] Given the evolution from segregation to desegregation, and back to the segregation of U.S. public schools in recent years, the racial/ethnic structure of schools and school systems is not

only a timely socioeconomic risk factor, but it is also a plausible, social determinant of population health that needs further investigation.

The Structure of School Environments in Adolescence

Racial/Ethnic Composition of U.S. Schools

Given the historical evolution of school segregation and court-ordered desegregation efforts in the South, the racial/ethnic composition of U.S. schools has changed dramatically since the *Brown* decision in 1954. In 1995, the same year during which Wave I of Add Health was conducted, 40.5% of metropolitan public schools, 61.8% of city public schools, and 25.6% of suburban public schools consisted of minority students. Particularly of note, between 1989 and 1995, public schools located in cities saw significant decreases in the white student enrollment, as well as increases in the minority student enrollment[107]. Understanding the current state of U.S. schools will help in our investigation of the impact of the racial/ethnic structure of school environments on adolescent obesity.

Both school segregation and school racial composition are constructs of the racial/ethnic structure of school environments. However, in this proposed study, we will conceptualize the racial/ethnic structure of schools and school systems using school racial composition and school entropy. While school racial segregation represents a regional phenomenon of the sorting of students of various races/ethnicities across schools within a geographic area, school racial composition measures the absolute racial/ethnic make-up of the student population, while school entropy measures the level of racial/ethnic diversity within a school. These two entities assess students'

experiences interacting with their peers of other racial/ethnic groups. More specifically, school racial composition and school entropy attempts to represent the daily-life stressors experienced by students and the general intergroup contact between racial/ethnic groups within a school.

Implications of the Racial Composition and Diversity within Schools

Recent literature has established a potentially harmful link between the racial/ethnic structure of schools and academic outcomes[108-113]. One study, using Add Health data, found that a school's racial/ethnic composition is associated with a student's academic achievement. These researchers suggested that this association could be explained by one's school socioeconomic characteristics. Schools with a higher proportion of students of color may have fewer socioeconomic and academic resources, which may negatively affect academic achievement[109]. There are also racial differences associated with this relationship[108,110,112]. Hanushek et al., (2009) found that the higher the percentage of black students at a school, the lower black students' academic achievement in mathematics. However, this study did not find any significant impact of school racial composition on white students' achievement[108]. Furthermore, the type of racially segregated school may also matter for academic achievement. Roscigno (1998) found that attending a predominantly black school was associated with lower academic achievement, while attending a predominantly white school was associated with higher academic achievement. These differences could be due to the better quality of teachers and higher levels of cultural and social capital that persists more in predominantly white schools than in predominantly black schools[111].

The racial structure of schools experienced during adolescence may also have effects on an individual's health that could accumulate across the life course. According to the perpetuation theory, persons who experience segregation in early life are more likely to have segregated experiences in adulthood. It is possible that school segregation experienced during adolescence can perpetuate into other domains of everyday life in adulthood[114]. Students attending racially segregated schools may have heightened beliefs of other racial/ethnic groups due to harmful experiences of racial/ethnic discrimination or lack of contact with other racial/ethnic groups. For students of color, this negative intergroup contact could result in attendance at historically black colleges and universities or the purchasing of homes in more segregated neighborhoods[97,114-116]. Segregation in other domains of life in adulthood could lead to disadvantage and prevent access to critical health-relevant resources that occur more abundantly in less segregated spaces. Similar to the life course framework, the perpetuation theory suggests that socio-contextual risk factors experienced in early life, such as racially segregated school environments, may send you down a path of segregation in later life.

Role of Residential Segregation

The effects of residential segregation on population health have been extensively investigated in the public health literature. In fact, racial residential segregation is a known cause of health disparities through its effects on the following factors: individual and neighborhood SES, inadequate educational and employment resources and opportunities, healthy behaviors and lifestyles, and access to health care[117]. It has been widely thought that racial residential segregation plays a role in the racial/ethnic structure of U.S. public schools. The racial/ethnic composition within a geographic area

can impact school segregation and the racial/ethnic make-up of the student population[118-119]. Yet, due to neighborhood gentrification, school choice, regional differences, and other processes, the correlation between racial residential segregation and the racial/ethnic structure of school environments is not complete[120].

In fact, if all students attended their neighborhood schools, school racial composition would be almost identical to the neighborhood racial composition. However, as a result of attendance in private, charter, and magnet schools, school racial composition is often different than the neighborhood racial composition. Also, there may be neighborhoods with a high proportion of households without any school-age children. In these areas, the racial/ethnic make-up of the resident population will not be equal to the racial/ethnic make-up of the student population in the neighborhood schools. Furthermore, it is possible that there may be lower levels of racial segregation in metropolitan schools if wealthy and white families did not have school choice[121]. As a result of white parents sending their children to private schools, some metropolitan areas may be racially diverse, while the public schools are predominantly black[120]. Though there is an inherent link between the distribution of racial/ethnic groups within a residential area and the racial composition of school within those areas, the correlation is not complete. In this study, we will attempt to determine whether there are independent effects of school racial composition and diversity and adolescent obesity, even after accounting for the demographic characteristics of the neighborhoods in which the schools reside.

How Does the Racial/Ethnic Structure of School Environments Get Under the Skin?

Social Epidemiologic Theory

Fundamental Causes

Given the associated racial/ethnic and socioeconomic disparities, there is a need for a more comprehensive understanding of the social, economic, and political contexts of these three distinct, yet connected, cardiometabolic and reproductive outcomes. Socioeconomic conditions are considered to be fundamental causes of disease because they are often associated with access to critical health-relevant resources, such as wealth, economic capital, social support, and political power[122-123]. Given its ties to inequitable school resources and opportunities, it is possible that the racial/ethnic structure of school environments is a fundamental cause of health, particularly in adolescence. Racially homogenous schools have higher dropout rates, offer fewer advanced courses, and lack high-caliber teachers[102,117]. Thus, students attending these segregated schools lack an access to important socioeconomic resources needed to live healthy and successful lives in adolescence, as well as in adulthood.

Biological Embodiment

The social embodiment of disease theory suggests that humans biologically embody the socioeconomic characteristics of the environments in which they live. Humans are literally products of the social, economic, and political environments in which they reside and interact[124-125]. Harmful life experiences, such as living in a socioeconomically disadvantaged neighborhood, experiences of sexual trauma or racial

discrimination, and even attending segregated schools, can prove to be detrimental to one's health. It is possible that the effects of attending segregated school environments could be biologically embodied. This contextual factor could constrain an individual's acquisition of important socioeconomic resources and their ability to develop critical social capital needed to prevent chronic health outcomes. Therefore, adolescents could biologically internalize the adverse health consequences of attending racially homogenous schools. Furthermore, since education is a critical means for increased social capital in adulthood[126], inadequate educational opportunities in segregated schools during adolescence could result in health inequities in adolescence, across the life course, and even across generations.

Life Course Framework

In order to fully understand the manifestation of health disparities, it is critical to examine how socio-contextual risk factors impact one's health across multiple stages of the life course: adolescence, early adulthood, and the reproductive age. The life course framework suggests that the timing of an exposure affects the development of disease and early-life socioeconomic factors can impact health in adulthood[127]. It has been theorized that the health effects of social and economic exposures can accumulate across the life course, and that differences in early-life socioeconomic conditions, such as educational attainment, can result in differential health trajectories from childhood into adulthood, leading to population health disparities[128-129].

According to this life course framework of population health, adolescence is also a "critical period" of human development, which is marked by substantial bran growth, as well as important social and emotional changes[127,130-133]. The Adolescent Pathway

model also suggests that adverse socioeconomic factors that are experienced during adolescence will not only result in poor adult health outcomes, but can also result in health inequalities across populations. In fact, this model also theorizes that socioeconomic disadvantage experienced at an early age can negatively impact health-relevant behaviors and resources, and result in the differential access to critical social capital and social networks, ultimately producing health disparities[131].

Additionally, it is possible that the racial/ethnic structure of school environments experienced during adolescence can have adverse consequences on cardiometabolic and reductive health in emerging adulthood. Experiences of social and economic disadvantage during childhood and adolescence can increase the risk for chronic health outcomes in adulthood[127,131,133]. Socioeconomic disadvantage in childhood increases one's risk of all-cause and cause-specific mortality[127-128,134], obesity and type 2 diabetes[128,131,135], CVD[128,135-137], and inflammation[128,138-139] in adulthood. Also, socioeconomic disadvantage, potentially experienced in racially segregated schools, could have adverse effects on pregnancy outcomes. Recent literature has suggested that contextual factors in early life may have greater consequences on the reproductive health of women across their life course than social disadvantage only experienced during pregnancy[57,62]. Given the critical development that occurs, any adverse exposures experienced during this period can have detrimental consequences on the physiologic structure and function of organs and tissues[127]. Thus, attending racially segregated school environments may affect the risk and development of chronic diseases in adolescence and in later life, as well as impact the acquisition of healthy behaviors and socioeconomic resources that are needed for optimal health in emerging adulthood.

Potential Biosocial Mechanisms

The Individual Level

School racial/ethnic composition and diversity may impact chronic health outcomes, such as adolescent obesity, through its effects on individual health behaviors and life style factors. The racial/ethnic composition of a school may impact chronic disease risk by limiting an adolescent's access to quality dietary options, ultimately preventing one's agency to make healthy food choices. In fact, high schools that have a higher percentage of black students are more likely to be located in close proximity to a fast food restaurant. Thus, adolescents attending these racially segregated that are close to fast food restaurants have poorer eating habits and are ultimately more likely to be overweight/obese[140]. At the individual level, the racial/ethnic structure of school environments can constrain a student's ability to make healthy food choices and may prevent the development of healthy behaviors.

The Interpersonal Level

The racial/ethnic structure of schools may also impact adolescent obesity through stressful events and negative intergroup contact. The contact hypothesis suggests that students attending more diverse schools are more likely to have more positive relationships with students outside of their own race, especially as they enter adulthood. In more diverse schools, there is increased intergroup contact between students of different racial/ethnic backgrounds, which results in increased racial tolerance and positive attitudes, as well as decreased racial prejudice[97,141]. Furthermore, the cultural deficit theory suggests that in more integrated schools, black students may benefit from

the "lateral transmission" of academic values, goals, capital, and resources from their white peers[97,142]. Schools provide the place for students to learn how to interact with other students from diverse backgrounds and to develop social skills and social capital that are critical for their development as they transition into adulthood.

Although there are positive effects for black students who attend more diverse schools (e.g., more economic resources, advanced courses, college opportunities), it is important to highlight some of the harmful effects of discrimination that can occur in predominantly white schools. In fact, black students attending predominantly white schools in Add Health were found to have worse adult health outcomes compared to their white peers, due to the negative consequences of perceived racial discrimination that they experienced being the racial/ethnic minority[143]. Recent studies have also found that black and Hispanic students attending predominantly white schools are likely to experience racial discrimination from their peers and racial stereotypes from their teachers[144-147]. It is possible that these experiences of racial discrimination impact health through the negative physiological consequences of stress[101]. Repeated stress over time can result in increases in cortisol levels and the disruption of normal cardiometabolic functions[71-72]. Stress, resulting from racial discrimination[101,148], can lead to poor dietary behaviors and a reduction in physical activity, ultimately resulting in adolescent obesity[87,149]. Therefore, at the interpersonal level, school racial composition and diversity may potentially get under the skin through the beneficial effects of intergroup contact or the harmful biological effects of racial discrimination.

The School Level

The impact of the racial/ethnic distribution of students within schools on chronic health outcomes can also manifest itself at the school level. Racially segregated schools have higher dropout rates and lack high-caliber teachers[102,117]. Additionally, segregated schools result in an unequal access to educational opportunities. For example, predominantly black/Latino and low-income schools are more likely to have fewer advanced course and more remedial/vocational courses and less likely to have access to college-preparatory courses than predominantly white and high-income schools[97]. This differential access to critical resources can impact graduation rates and college attendance, ultimately resulting in detrimental consequences for a student's health-relevant resources as an adult, such as educational attainment, employment, and income.

The cultural deficit theory also suggests that black students benefit more academically by attending predominantly white schools, than predominantly black schools. Though there are potentially harmful effects of discrimination, this theory suggests that, in more integrated schools, there is a lateral transmission of academic values, goals, and resources from white students to black students[97,142]. Attending predominantly white schools may provide black students with greater social capital as they transition through various periods of their lives. It is also thought that attending more integrated colleges and universities may prove to be advantageous to black students in that they are more likely to pursue careers in which they are underrepresented and are more likely to be hired by employers upon graduation[97]. Finally, lower levels of education achieved in these lower-quality schools can impact

chronic disease risk through its effects on stress, social support, healthy behaviors and lifestyles, and one's SES accumulated across the life course[150-152]. Therefore, at the school level, school racial composition can get under the skin through its impact on socioeconomic potential and health-relevant resources, such as educational attainment and employment.

Public Health Contributions and Policy Importance

The academic and socioeconomic impacts of the racial/ethnic structure of schools have been investigated extensively in the sociology and educational studies literature. However, very few studies have investigated the impact of school segregation on cardiometabolic and reproductive health within a life course framework. This proposed study will not only fill this gap in the public health literature, but it will also contribute to the current social epidemiologic literature. Given the persistent racial and socioeconomic disparities and that humans can embody the social contexts in which they reside, the racial/ethnic structure of school environments is a plausible social determinant of population health. Current social epidemiologic studies are attempting to integrate macro-level determinants of health with the associated biosocial mechanisms in order to better understand the relationships between social, economic, and political exposures and various health outcome[153]. Thus, given the large public health burden of obesity, inflammation, and PTB, investigating the link between school segregation and these chronic and reproductive outcomes in this study will contribute to our understanding of the ways in which more large-scale social contexts can impact health across the life course.

The recent segregation of public schools in the U.S. again, beginning in the late 20th century, has made school segregation a timely and relevant exposure. The findings from this dissertation can also inform current educational policies. By understanding the social, economic, and political contexts, this dissertation can contribute to the contemporary educational policy debate on the health consequences of children attending segregated and racially homogenous schools. Given that we will examine the effects of school racial composition on health in adolescence and early adulthood, as well as those passed from mother to child through experiences of PTB, this dissertation will also provide an important life course perspective to existing zoning and educational policies.

References

1. Ogden CL, Carroll MD, Lawman HG, Fryar CD, Kruszon-Moran D, Kit BK, et al. Trends in obesity prevalence among children and adolescents in the United States, 1988-1994 through 2013-2014. *JAMA*. 2016; 315(21):2292-9.

2. Ogden CL, Fryar CD, Hales CM, Carroll MD, Aoki Y, Freedman DS. Differences in obesity prevalence by demographics and urbanization in US children and adolescents, 2013-2016. *JAMA*. 2018; 319(23):2410-8.

3. Benjamin EJ, Blaha MJ, Chiuve SE, Cushman M, Das SR, Deo R, et al. Heart disease and stroke statistics – 2017 update: A report from the American Heart Association. *Circulation*. 2017; 135(10):e146-e603.

4. Barlow SE, Expert Committee. Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity: Summary report. *Pediatrics*. 2007; 120 Suppl 4:S164-92.

5. Liu J-H, Jones SJ, Sun H, Probst JC, Merchant AT, Cavicchia P. Diet, physical activity, and sedentary behaviors as risk factors for childhood obesity: An urban and rural comparison. *Child Obes*. 2012; 8(5):440-8.

6. Singh GK, Kogan MD, Van Dyck PC, Siahpush M. Racial/ethnic, socioeconomic, and behavioral determinants of childhood and adolescent obesity in the United States: Analyzing independent and joint associations. *Ann Epidemiol*. 2008; 18(9):682-95.

7. Ogden CL, Carroll MD, Fakhouri TH, Hales CM, Fryar CD, Li X, Freedman DS. Prevalence of obesity among youths by household income and education level of head of household – United States 2011-2014. *MMWR Morb Mortal Wkly Rep*. 2018; 67(6):186-9.

8. Frederick CB, Snellman K, Putnam RD. Increasing socioeconomic disparities in adolescent obesity. *Proc Natl Acad Sci USA*. 2014; 111(4):1338-42.

9. Kelly AS, Barlow SE, Rao G, Inge TH, Hayman LL, Steinberger J, et al. Severe obesity in children and adolescents: Identification, associated health risks, and treatment approaches: A scientific statement from the American Heart Association. *Circulation*. 2013; 128(15):1689-712.

10. Miech RA, Kumanyika SK, Stettler N, Link BG, Phelan JC, Chang VW. Trends in the association of poverty with overweight among US adolescents, 1971-2004. *JAMA*. 2006; 295(20):2385-93.

11. Delva J, O'Malley PM, Johnston LD. Racial/ethnic and socioeconomic status differences in overweight and health-related behaviors among American students: National trends 1986-2003. *J Adolesc Health*. 2006; 39(4):536-45.

12. Singh GK, Kogan MD, van Dyck PC. A multilevel analysis of state and regional disparities in childhood and adolescent obesity in the United States. *J Community Health*. 2008; 33(2):90-102.

13. Johnson JA 3rd, Johnson AM. Urban-rural differences in childhood and adolescent obesity in the United States: A systematic review and meta-analysis. *Child Obes*. 2015; 11(3):233-41.

14. O'Malley PM, Johnston LD, Delva J, Bachman JG, Schulenberg JE. Variation in

obesity among American secondary school students by school and school characteristics. *Am J Prev Med*. 2007; 33(4 Suppl):S187-94.

15. Freedman DS, Mei Z, Srinivasan SR, Berenson GS, Dietz WH. Cardiovascular risk factors and excess adiposity among overweight children and adolescents: The Bogalusa Heart Study. *J Pediatr*. 2007; 150(1):12-17.e2.

16. Freedman DS, Khan LK, Serdula MK, Dietz WH, Srinivasan SR, Berenson GS. The relation of childhood BMI to adult adiposity: The Bogalusa Heart Study. *Pediatrics*. 2005; 115(1)22-7.

17. Srinivasan SR, Bao W, Wattigney WA, Berenson GS. Adolescent overweight is associated with adult overweight and related multiple cardiovascular risk factors: The Bogalusa Heart Study. *Metabolism*. 1996; 45(2):235-40.

18. Weiss R, Dziura J, Burgert TS, Tamborlane WV, Taksali SE, Yeckel CW, et al. Obesity and the metabolic syndrome in children and adolescents. *N Engl J Med*. 2004; 350(23):2362-74.

19. Ford ES, Galuska DA, Gillespie C, Will JC, Giles WH, Dietz WH. C-reactive protein and body mass index in children: Findings from the Third National Health and Nutrition Examination Survey, 1988-1994. *J Pediatr*. 2001; 138(4):486-92.

20. Reilly JJ, Kelly J. Long-term impact of overweight and obesity in childhood and adolescence on morbidity and premature mortality in adulthood: Systematic Review. *Int J Obes*. 2011; 35(7):891-8.

21. Norris AL, Steinberger J, Steffen LM, Metzig AM, Schwarzenberg SJ, Kelly AS. Circulating oxidized LDL and inflammation in extreme pediatric obesity. *Obesity*. 2011; 19(7):1415-9.

22. Baker JL, Olsen LW, Sørensen TI. Childhood body-mass index and the risk of coronary heart disease in adulthood. *N Engl J Med*. 2007; 357(23):2329-37.

23. Skinner AC, Perrin EM, Moss LA, Skelton JA. Cardiometabolic risks and severity of obesity in children and young adults. *N Engl J Med*. 2015; 373(14):1307-17.

24. Must A, Strauss RS. Risks and consequences of childhood and adolescent obesity. *Int J Obes Relat Metab Disord*. 1999; 23 Suppl 2:S2-11.

25. Gunnell DJ, Frankel SJ, Nanchahal K, Peters TJ, Davey Smith G. Childhood obesity and adult cardiovascular mortality: A 57-y follow-up study based on the Boyd Orr cohort. *Am J Clin Nutr*. 1998; 67(6):1111-8.

26. Must A, Jacques PF, Dallal GE, Bajema CJ, Dietz WH. Long-term morbidity and mortality of overweight adolescents: A follow-up of the Harvard Growth Study of 1922 to 1935. *N Engl J Med*. 1992; 327(19):1350-5.

27. Lauer RM, Clarke WR. Childhood risk factors for high adult blood pressure: The Muscatine Study. *Pediatrics*. 1989; 84(4):633-41.

28. Ford ES, Ajani UA, Croft JB, Critchley JA, Labarthe DR, Kottke TE, et al. Explaining the decrease in U.S. deaths from coronary disease, 1980-2000. *N Engl J Med*. 2007; 356(23):2388-98.

29. Bhatt H, Safford M, Glasser S. Coronary heart disease risk factors and outcomes in the twenty-first century: Findings from the REasons for Geographic and Racial Differences in Stroke (REGARDS) Study. *Curr Hypertens Rep*. 2015; 17(4):541.

30. Kulshreshtha A, Goyal A, Dabhadkar K, Veledar E, Vaccarino V. Urban-rural

differences in coronary heart disease mortality in the United States: 1999-2009. *Public Health Rep*. 2014; 129(1):19-29.

31. Graham G. Disparities in cardiovascular disease risk in the United States. *Curr Cardiol Rev*. 2015; 11(3):238-45.

32. Kurian AK, Cardarelli KM. Racial and ethnic differences in cardiovascular disease risk factors: A systematic review. *Ethn Dis*. 2007; 17(1):143-52.

33. Mensah GA, Mokdad AH, Ford ES, Greenlund KJ, Croft JB. State of disparities in cardiovascular health in the United States. *Circulation*. 2005; 111(10):1233-41.

34. Kramer MR, Valderrama AL, Casper ML. Decomposing Black-White disparities in heart disease mortality in the United States, 1973-2010: An age-period-cohort analysis. *Am J Epidemiol*. 2015; 182(4):302-12.

35. Vaughan AS, Quick H, Pathak EB, Kramer MR, Casper M. Disparities in temporal and geographic patterns of declining heart disease mortality by race and sex in the United States, 1973-2010. *J Am Heart Assoc*. 2015; 4(12). pii:e002567.

36. Winkleby MA, Kraemer HC, Ahn DK, Varady AN. Ethnic and socioeconomic differences in cardiovascular disease risk factors: Findings for women from the Third National Health and Nutrition Examination Survey, 1988-1994. *JAMA*. 1998; 280(4):356-62.

37. Winkleby MA, Jatulis DE, Frank E, Fortmann SP. Socioeconomic status and health: How education, income, and occupation contribute to risk factors for cardiovascular disease. *Am J Public Health*. 1992; 82(6):816-20.

38. Roth GA, Dwyer-Lindgren L, Bertozzi-Villa A, Stubbs RW, Morozoff C, Naghavi M, et al. Trends and patterns of geographic variation in cardiovascular mortality among US counties, 1980-2014. *JAMA*. 2017; 317(19):1976-92.

39. Diez Roux AV, Nieto FJ, Muntaner C, Tyroler HA, Comstock GW, Shahar E, et al. Neighborhood environments and coronary heart disease: A multilevel analysis. *Am J Epidemiol*. 1997; 146(1):48-63.

40. Diez Roux AV, Merkin SS, Arnett D, Chambless L, Massing M, Nieto FJ, et al. Neighborhood of residence and incidence of coronary heart disease. N Engl J Med. 2001; 345(2):99-106.

41. Nordstrom CK, Diez Roux AV, Jackson SA, Gardin JM, Cardiovascular Health Study. The association of personal and neighborhood socioeconomic indicators with subclinical cardiovascular disease in an elderly cohort: The cardiovascular health study. *Soc Sci Med*. 2004; 59(10):2139-47.

42. Sundquist K, Winkleby M, Ahlén H, Johansson SE. Neighborhood socioeconomic environment and incidence of coronary heart disease: A follow-up study of 25,319 women and men in Sweden. *Am J Epidemiol*. 2004; 159(7):655-62.

43. Abbasi F, Brown BW Jr, Lamendola C, McLaughlin T, Reaven GM. Relationship between obesity, insulin resistance, and coronary heart disease risk. *J Am Coll Cardiol*. 2002; 40(5):937-43.

44. Koenig W. Inflammation and coronary heart disease: An overview. *Cardiol Rev*. 2001; 9(1):31-5.

45. Buckley DI, Fu R, Freeman M, Rogers K, Helfand M. C-reactive protein as a risk factor for coronary heart disease: A systematic review and meta-analyses for the U.S. Preventive Services Task Force. *Ann Intern Med*. 2009; 151(7):483-95.

46. Koenig W, Sund M, Frölich M, Fischer HG, Löwel H, Döring A, et al. C-reactive protein, a sensitive marker of inflammation, predicts future risk of coronary heart disease in initially healthy middle-aged men: Results from the MONICA (Monitoring Trends and Determinants in Cardiovascular Disease) Augsburg Cohort Study, 1984-1992. *Circulation*. 1999; 99(2):237-42.

47. Emerging Risk Factors Collaboration, Kaptoge S, Di Angelantonio E, Lowe G, Pepys MB, Thompson SG, et al. C-reactive protein concentration and risk of coronary heart disease, stroke, and mortality: An individual participant meta-analysis. *Lancet*. 2010; 375(9709):132-40.

48. Rifai N, Ridker PM. High-sensitivity C-reactive protein: A novel and promising marker of coronary heart disease. *Clin Chem*. 2001; 47(3):403-11.

49. Cushman M, Arnold AM, Psaty BM, Manolio TA, Kuller LH, Burke GL, et al. C-reactive protein and the 10-year incidence of coronary heart disease in older men and women: The cardiovascular health study. *Circulation*. 2005; 112(1):25-31.

50. Ridker PM, Buring JE, Shih J, Matias M, Hennekens CH. Prospective study of C-reactive protein and the risk of future cardiovascular events among apparently healthy women. *Circulation*. 1998; 98(8):731-3.

51. Martin JA, Hamilton BE, Osterman MJ, Driscoll AK, Mathews TJ. Births: Final Data for 2015. *Natl Vital Stat Rep*. 2017; 66(1):1.

52. Beck S, Wojdyla D, Say L, Betran AP, Merialdi M, Requejo JH, et al. The worldwide incidence of preterm birth: A systematic review of maternal mortality and morbidity. *Bull World Health Organ*. 2010; 88(1):31-8.

53. Goldenberg RL, Culhane JF, Iams JD, Romero R. Epidemiology and causes of preterm birth. *Lancet*. 2008; 371(9606):75-84.

54. Schoen CN, Tabbah S, Iams JD, Caughey AB, Berghella V. Why the United States preterm birth rate is declining. *Am J Obstet Gynecol*. 2015; 213(2):175-80.

55. Wadhwa PD, Culhane JF, Rauh V, Barve SS. Stress and preterm birth: Neuroendocrine, immune/inflammatory, and vascular mechanisms. *Matern Child Health J*. 2001; 5(2):119-25.

56. Culhane JF, Goldenberg RL. Racial disparities in preterm birth. *Semin Perinatol*. 2011; 35(4):234-9.

57. Lu MC, Halfon N. Racial and ethnic disparities in birth outcomes: A life-course perspective. *Matern Child Health J*. 2003; 7(1):13-30.

58. MacDorman MF. Race and ethnic disparities in fetal mortality, preterm birth, and infant mortality in the United States: An overview. *Semin Perinatol*. 2011; 35(4):200-8.

59. Kramer MS, Goulet L, Lydon J, Séguin L, McNamara H, Dassa C, et al. Socioeconomic disparities in preterm birth: Causal pathways and mechanisms. *Paediatr Perinat Epidemiol*. 2001; 15 Suppl 2: 104-23.

60. Kramer MS, Séguin L, Lydon J, Goulet L. Socio-economic disparities in pregnancy outcome: Why do the poor fare so poorly? *Paediatr Perinat Epidemiol*. 2000; 14(3):194-210.

61. Peacock JL, Bland JM, Anderson HR. Preterm delivery: Effects of socioeconomic factors, psychological stress, smoking, alcohol, and caffeine. *BMJ*. 1995; 311(7004):531-5.

62. Blumenshine P, Egerter S, Barclay CJ, Cubbin C, Braveman PA. Socioeconomic disparities in adverse birth outcomes: A systematic review. *Am J Prev Med*. 2010; 39(3):263-72.

63. Braveman PA, Heck K, Egerter S, Marchi KS, Dominguez TP, Cubbin C, et al. The role of socioeconomic factors in Black-White disparities in preterm birth. *Am J Public Health*. 2015; 105(4):694-702.

64. Lindstrom K, Lindblad F, Hjern A. Preterm birth and attention deficit/hyperactivity disorder in schoolchildren. *Pediatrics*. 2011; 127(5):858-65.

65. Sucksdorff M, Lehtonen L, Chudal R, Suominen A, Joelsson P, Gissler M, et al. Preterm birth and poor fetal growth as risk factors of attention-deficit/hyperactivity disorder. *Pediatrics*. 2015; 136(3):e599-608.

66. Arpi E, Ferrari F. Preterm birth and behavior problems in infants and preschool age children: A review of the recent literature. *Dev Med Child Neurol*. 2013; 55(9):788-96.

67. Barker DJ. Fetal origins of coronary heart disease. *Br Heart J*. 1993; 69(3):195-6.

68. Barker DJ. Fetal programming of coronary heart disease. *Trends Endocrinol Metab*. 2002; 13(9):364-8.

69. Rogers LK, Velten M. Maternal inflammation, growth retardation, and preterm birth: Insights into adult cardiovascular disease. *Life Sci*. 2011; 89(13-14):417-21.

70. Singh-Manoux A, Ferrie JE, Chandola T, Marmot M. Socioeconomic trajectories across the life course and health outcomes in midlife: Evidence for the accumulation hypothesis? *Int J Epidemiol*. 2004; 33(5):1072-9.

71. McEwen BS, Seeman T. Protective and damaging effects of mediators of stress: Elaborating and testing the concepts of allostasis and allostatic load. *Ann N Y Acad Sci*. 1999; 896:30-47.

72. McEwen BS. Protective and damaging effects of stress mediators. *N Engl J Med*. 1998; 338(3):171-9.

73. Chang MK, Binder CJ, Torzewski M, Witztum JL. C-reactive protein binds to both oxidized LDL and apoptotic cells through recognition of a common ligand: Phosphorylcholine of oxidized phospholipids. *Proc Natl Acad Sci U S A*. 2002; 99(20):13043-8.

74. Zwaka TP, Hombach V, Torzewski J. C-reactive protein-mediated low density lipprotein uptake by macrophages: Implications for atherosclerosis. *Circulation*. 2001; 103(9):1194-7.

75. Black S, Kushner I, Smols D. C-reactive protein. *J Biol Chem*. 2004; 279(47):48487-90.

76. Hirschfield GM, Pepys MB. C-reactive protein and cardiovascular disease: New insights from an old molecule. *QJM*. 2003; 96(11):793-807.

77. Zhang YX, Cliff WJ, Schoefl GI, Higgins G. Coronary C-reactive protein distribution: Its relation to development of atherosclerosis. *Atherosclerosis*. 1999; 145(2):375-9.

78. Pradhan AD, Manson JE, Rifai N, Buring JE, Ridker PM. C-reactive protein, interleukin 6, and risk of developing type 2 diabetes mellitus. *JAMA*. 2001; 286(3):327-34.

79. Ford ES, Giles WH, Mokdad AH, Myers GL. Distribution and correlates of C-reactive protein concentrations among adult US women. *Clin Chem*. 2004; 50(3):574-81.

80. Ong KL, Allison MA, Cheung BM, Wu BJ, Barter PJ, Rye KA. Trends in C-reactive protein levels in US adults from 1999 to 2010. *Am J Epidemiol*. 2013; 177(12):1430-42.

81. Nazmi A, Victoria CG. Socioeconomic and racial/ethnic differentials of C reactive protein levels: A systematic review of population-based studies. *BMC Public Health*. 2007; 7:212.

82. Gruenewald TL, Cohen S, Matthews KA, Tracy R, Seeman TE. Association of socioeconomic status with inflammation markers in black and white men and women in the Coronary Artery Risk Development in Young Adults (CARDIA) study. *Soc Sci Med*. 2009; 69(3):451-9.

83. Dinwiddie GY, Zambrana RE, Doamekpor LA, Lopez L. The impact of educational attainment on observed race/ethnic disparities in inflammatory risk in the 2001-2008 National Health and Nutrition Examination Survey. *Int J Environ Res Public health*. 2015; 13(1):ijerph13010042.

84. Stepanikova I, Bateman LB, Oates GR. Systemic inflammation in midlife: Race, socioeconomic status, and perceived discrimination. *Am J Prev Med*. 2017; 52(1S1:S63-S76.

85. Monteiro R, Azvedo I. Chronic inflammation in obesity and the metabolic syndrome. *Mediators Inflamm*. 2010; 2010.

86. Cappelletti M, Della Bella S, Ferrazzi E, Mavilio D, Divanovic S. Inflammation and preterm birth. *J Leukoc Biol*. 2016; 99(1):67-78.

87. De Vriendt T, Moreno LA, De Henauw S. Chronic stress and obesity in adolescents: Scientific evidence and methodological issues for epidemiological research. *Nutr Metab Cardiovasc Dis*. 2009; 19(7):511-9.

88. Hobel CJ, Dunkel-Schetter C, Roesch SC, Castro LC, Arora CP. Maternal plasma corticotropin-releasing hormone associated with stress at 20 weeks' gestation in pregnancies ending in preterm delivery. *Am J Obstet Gynecol*. 1999; 180(1 Pt 3):S257-63.

89. Lockwood CJ. Stress-associated preterm delivery: The role of corticotropin releasing hormone. *Am J Obstet Gynecol*. 1999; 180(1 Pt 3):S264-6.

90. Geronimus AT. The weathering hypothesis and the health of African-American women and infants: Evidence and speculations. *Ethn Dis*. 1992; 2(3):207-21.

91. Plessy v. Ferguson, 163 U.S. 537 (1896).

92. Tischauser LV. *Jim Crow Laws: Landmarks of the American Mosaic*. Greenwood: Santa Barbara, CA; 2012.

93. Clotfelter CT. *After Brown: The Rise and Retreat of School Desegregation*. Princeton University Press: Princeton, NJ; 2004.

94. Brown v. Board of Education of Topeka, 347 U.S. 483 (1954).

95. Brown II, 349 U.S. 294 (1955).

96. Green v. County School Board of New Kent County, 391 U.S. 430 (1968).

97. Smrekar CE, Goldring EB, eds. *From the Courtroom to the Classroom: The*

Shifting Landscape of School Desegregation. Harvard Education Press: Cambridge, MA; 2009

98. Swann v. Charlotte-Mecklenberg Board of Education, 402 U.S. 1 (1971).

99. Keyes v. Denver School District No. 1, 413 U.S. 189 (1973).

100. Reardon SF, Owens A. 60 years after *Brown*: Trends and Consequences of school segregation. *Annu Rev Sociol*. 2014; 40:199-218.

101. Williams DR. Race, socioeconomic status, and health: The added effects of racism and discrimination. *Ann N Y Acad Sci*. 1999; 896:173-88.

102. Orfield G, Eaton SE, The Harvard Project on School Desegregation. *Dismantling Desegregation: The Quiet Reversal of Brown v. Board of Education*. New Press: New York, NY; 1996.

103. Milliken v. Bradley, 418 U.S. 717 (1974).

104. Riddick v. School Board of the City of Norfolk, Virginia 784 F.2d 251 (4th Cir. 1986).

105. Board of Education of Oklahoma v. Dowell, 498 U.S. 237 (1991).

106. Freeman v. Pitts, 503 U.S. 567 (1992).

107. Reardon SF, Yun JT, Menulty Eitle T. The changing structure of school segregation: Measurement and evidence of multiracial metropolitan-area school segregation, 1989-1995. *Demography*. 2000; 37(3):351-64.

108. Hanushek EA, Kain JF, Rivkin SG. New evidence about *Brown v. Board of Education*: The complex effects of school racial composition on achievement. *J Labor Economics*. 2009; 27(3):349-83.

109. Lee H. The effects of school racial and ethnic composition on academic achievement during adolescence. *Journal of Negro Education*. 2007; 76(2):154-72.

110. Goldsmith PA. All segregation is not equal: The impact of Latino and Black school composition. *Sociological Perspectives*. 2003; 46(1):83-105.

111. Roscigno VJ. Race and the reproduction of educational disadvantage. *Social Forces*. 1998; 76(3):1033-61.

112. Bankston C III, Caldas SJ. Majority African American schools and social injustice: The influence of de factor segregation on academic achievement. *Social Forces*. 1996; 75(2):535-55.

113. Braddock JH II, Dawkins MP. Long-term effects of school desegregation on southern Blacks. *Sociological Spectrum*. 1984; 4(4):365-81.

114. Wells AS, Crain RL. Perpetuation theory and the long-term effects of school desegregation. *Review of Educational Research*. 1994; 64(4):531-55.

115. McPartland JM, Braddock JH. Going to college and getting a good job: The impact of desegregation. In Hawley WD (ed.), *Effective School Desegregation: Equality, Quality and Feasibility*. Sage Publications: London, England; 1981, 141-54.

116. Braddock JH. The perpetuation of segregation across levels of education: A behavioral assessment of the contact-hypothesis. *Sociology of Education*. 1980; 53(3):178-86.

117. Williams DR, Collins C. Racial residential segregation: A fundamental cause of racial disparities in health. *Public Health Rep*. 2001; 116(5):404-16.

118. Rivkin SG. Residential segregation and school integration. *Sociology of Education*. 1994; 67(4):279-92.

119. Farley R. Residential segregation and its implications for school integration. *Law And Contemporary Problems*. 1975; 39(1):164-93.

120. Denton NA. The persistence of segregation: Links between residential segregation and school segregation. *Minnesota Law Review*. 1996; 80(4):795-824.
121. Saporito S, Sohoni D. Coloring outside the lines: Racial segregation in public schools and their attendance boundaries. *Sociology of Education*. 2006; 79(2):81-105.
122. Link BG, Phelan J. Social conditions as fundamental causes of disease. *J Health Soc Behav*. 1995; Spec No:80-94.
123. Phelan JC, Link BG, Tehranifar P. Social conditions as fundamental causes of health inequalities: Theory, evidence, and policy implications. *J Health Soc Behav*. 2010; 51 Suppl:S28-40.
124. Krieger N. Methods for the scientific study of discrimination and health: An Ecosocial approach. *Am J Public Health*. 2012; 102(5):936-44.
125. Krieger N. Theories for social epidemiology in the 21st century: An ecosocial perspective. *Int J Epidemiol*. 2001; 30(4):668-77.
126. Huang J, van den Brink HM, Groot W. A meta-analysis of the effect of education on Social capital. *Econ Edu Rev*. 2009; 28(4):454-64.
127. Kuh D, Ben-Shlomo Y, Lynch J, Hallqvist J, Power C. Life course epidemiology. *J Epidemiol Community Health*. 2003; 57(10):778-83.
128. Braveman P, Barclay C. Health disparities beginning in childhood: A life course perspective. *Pediatrics*. 2009; 124 Suppl 3:S163-75.
129. Power C, Hertzman C. Social and biological pathways linking early life and adult disease. *Br Med Bull*. 1997; 53(1):210-21.
130. Viner RM, Ozer EM, Denny S, Marmot M, Resnick M, Fatusi A, et al. Adolescence and the social determinants of health. *Lancet*. 2012; 379(9826):1641-52.
131. Due P, Krølner R, Rasmussen M, Andersen A, Trab Damsgaard M, Graham, et al. Pathways and mechanisms in adolescence contribute to adult health inequalities. *Scand J Public Health*. 2011; 39(6 Suppl):62-78.
132. Sawyer SM, Afifi RA, Bearinger LH, Blackemore SJ, Dick B, Ezeh AC, et al. Adolescence: A foundation for future health. *Lancet*. 2012; 379(9826):1630-40.
133. Viner RM, Ross D, Hardy R, Kuh D, Power C, Johnson A, et al. Life course epidemiology: Recognising the importance of adolescence. *J Epidemiol Community Health*. 2015; 69(8):719-20.
134. Smith GD, Hart C, Blane D, Hole D. Adverse socioeconomic conditions in childhood and cause specific adult mortality: Prospective observational study. *BMJ*. 1998; 316(7145):1631-5.
135. Nandi A, Glymour MM, Kawachi I, VanderWeele TJ. Using marginal structural models to estimate the direct effect of adverse childhood social conditions on onset of heart disease, diabetes, and stroke. *Epidemiology*. 2012; 23(2):223-32.
136. Kaplan GA, Salonen JT. Socioeconomic conditions in childhood and ischaemic heart disease during middle age. *BMJ*. 1990; 301(6761):1121-3.
137. Galobardes B, Lynch JW, Davey Smith G. Childhood socioeconomic circumstances and cause-specific mortality in adulthood: Systematic review and interpretation. *Epidemiol Rev*. 2004; 26:7-21.
138. Pollitt RA, Kaufman JS, Rose KM, Diez Roux AV, Zeng D, Heiss G. Early-life and adult socioeconomic status and inflammatory risk markers in adulthood. *Eur J Epidemiol*. 2007; 22(1):55-66.

139. Liu RS, Aiello AE, Mensah FK, Gasser CE, Rueb K, Cordell B, et al. Socioeconomic status in childhood and C reactive protein in adulthood: A systematic review and meta-analysis. *J Epidemiol Community Health*. 2017; 71(8):817-26.

140. Kwate NO, Loh JM. Separate and unequal: The influence of neighborhood and school characteristics on spatial proximity between fast foods and schools. *Prev Med*. 2010; 51(2):153-6.

141. Pettigrew T, Tropp I. Does intergroup contact reduce prejudice? Recent meta-analytic findings. In Oskamp S (ed.), *Reducing Prejudice and Discrimination: Social Psychological Perspectives*. Erlbaum: Mahwah, NJ; 2000. 93-114.

142. Gerard HB. School desegregation: The social science role. *American Psychologist*. 1983; 38(8):869-77.

143. Goosby BJ, Walsemann KM. School racial composition and race/ethnic differences in early adulthood health. *Health Place*. 2012; 18(2):296-304.

144. McCabe J. Racial and gender microaggressions on a predominantly-white campus: Experiences of black, Latina/o and white undergraduates. *Race, Gender & Class*. 2009; 16(1/2):133-51.

145. Juvonen J, Nishina A, Graham S. Ethnic diversity and perceptions of safety in urban middle schools. *Psychological Science*. 2006; 17(5):393-400.

146. Feagin JR, Vera H, Imani N. *The Agony of Education: Black Students at a White University* (2nd ed.). Routledge: New York, NY; 1996.

147. Allen WR. The color of success: African-American college student outcomes at Predominantly white and historically black public colleges and universities. *Harvard Educational Review*. 1992; 62(1):26-44.

148. Clark R, Anderson NB, Clark VR, Williams DR. Racism as a stressor for African Americans: A biopsychosocial model. *American Psychologist*. 1999; 54(10):805-16.

149. Pervanidou P, Chrousos GP. Stress and obesity/metabolic syndrome in childhood and adolescence. *Int J Pediatr Obes*. 2011; 6 Suppl 1:21-8.

150. Hahn RA, Truman BI. Education improves public health and promotes health equity. *Int J Health Serv*. 2015; 45(4):657-78.

151. Ross CE, Wu CL. The links between education and health. *American Sociological Review*. 1995; 60(5):719-45.

152. Ross CE, Wu CL. Education, age, and the cumulative advantage in health. *J Health Soc Behav*. 1996; 37(1):104-20.

153. Galea S, Link BG. Six paths for the future of social epidemiology. *Am J Epidemiol*. 2013; 178(6):843-9.

Not So Black and White: The Racial/Ethnic Structure of School Environments and Cardiometabolic and Reproductive Health across the Life Course

Chapter 2: Adolescent Obesity

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SPECIFIC AIM 1

Investigate the multilevel associations between the racial/ethnic structure of school environments in adolescence and the prevalence of adolescent obesity.

Hypothesis 1a: Adolescents who attend schools with a higher proportion of Black students, higher proportion of non-White students, or with less racial/ethnic diversity will have an increased likelihood of adolescent obesity, after adjusting for individual-, school-, and neighborhood-level characteristics.

Hypothesis 1b: The associations between the racial/ethnic structure of school environments in adolescence and the prevalence of adolescent obesity will differ by an adolescent's race/ethnicity.

ABSTRACT

Since schools play an important role in adolescent development, it is possible that the disparities in adolescent obesity may be driven by not only individual characteristics, but also by the racial/ethnic structure of their school environments. This study will examine the racial/ethnic differences in the associations between school racial/ethnic composition and diversity and adolescent obesity. We used data from Wave I (1994-5) of the National Longitudinal Adolescent to Adult Health Study (N=20,745). Using self-reported height and weight data, adolescent obesity was categorized as not overweight/obese (< 85th percentile) and overweight/obese (≥ 85th percentile). The predicted marginal prevalence of obesity, by school racial/ethnic composition (i.e., proportion Black and non-White students) and school racial/ethnic diversity (i.e., school entropy), were estimated from cluster- and sample-weight adjusted logistic regression models in SUDAAN. Models stratified by race/ethnicity were adjusted for school-level (size, type, urbanicity), neighborhood-level (poverty, racial composition), and individual-level (age, gender, parent's education, physical activity) characteristics. Adolescents identifying as Asian/Native American/Other who attended schools with 26-50% Black students were 2.11 times more likely to be obese (95% CI: 1.08, 4.15) than those who attended schools with 0-10% Black students. Non-Hispanic Black adolescents who attended schools with 11-25% non-White students were 1.83 times more likely to be obese (95% CI: 1.07, 3.11) than those who attended schools with 0-10% non-White students. Attending a school with moderate levels of Black and non-White students is associated with an increased prevalence of obesity among Asian/Native American/Other and non-Hispanic Black adolescents.

SIGNIFICANCE

The prevalence of obesity among U.S. adolescents, aged 12-19 years old, has increased in recent years[1], with approximately 20.6% of adolescents being obese[2-3]. Adolescents who are less physically active, have poor dietary habits, or have a family history of obesity are at a higher risk for developing obesity during adolescence[4-6]. Black and Hispanic adolescents and those having a lower family socioeconomic status (SES) are more likely to be overweight or obese[2-4,6-11]. Adolescents living in southern states and non-urban areas are more likely to be overweight or obese, compared to those living in other regions of the country and urban areas, respectively[2,5,12-13]. Additionally, being overweight or obese in adolescence can have adverse health consequences that carry on into adulthood, including type 2 diabetes, high blood pressure, high cholesterol, cardiovascular disease, and mortality[1,4,9,14-24].

Given the persistent disparities, identifying the relevant socio-contextual risk factors is critical to further understanding the underlying biosocial mechanisms in order to fully address this public health burden. In fact, since adolescents are shaped by their environments and peer social groups, the racial/ethnic structure of school environments could be one of these socio-contextual risk factors. Considering the historical context of school segregation in the U.S. is important to the investigation of the health impact of the racial/ethnic structure of school environments. *Plessy v. Ferguson* (1896) provided the legal basis for the racial segregation of public schools, which ultimately produced inequitable educational opportunities for black students in the U.S.[25-27]. *Brown v. Board of Education of Topeka* (1954) ended this era of "separate, but equal" and southern schools, in particular, were forced to desegregate[28]. However, due to

persistent residential segregation and other defining Supreme Court decisions that released school systems from their court-ordered mandates to integrate[29-32], U.S. public schools began to resegregate in the late 1970s[27,33-35]. The 1980s and 1990s saw significant decreases in the white student enrollment in urban schools, as well as increases in the minority student enrollment[36]. Given the evolution from segregation to desegregation, and back to resegregation of U.S. public schools in recent years, the racial/ethnic structure of schools and school systems is not only a timely socioeconomic risk factor, but it is also a plausible, social determinant of adolescent obesity that needs further investigation.

According to ecosocial theory, humans can biologically embody the characteristics of the contexts in which they live and interact[37-38]. Adolescents spend a considerable amount of time in school, which are places that should provide the foundation to increase economic resources and develop social capital. However, this is not always the case. Predominantly black or Hispanic schools have higher dropout rates and lack high-caliber teachers[35,39]. Additionally, students attending these racially-segregated schools are more likely to have fewer advanced courses and more remedial/vocational courses, and less likely to have access to college-preparatory courses than those attending predominantly white schools[40]. Recent literature has established a potentially harmful link between the racial/ethnic structure of schools and academic outcomes[41-46]. However, this association is dependent upon the type of racially segregated school environment. Due to fewer socioeconomic and academic resources available, schools with a higher proportion of minority students are more likely to have lower academic achievement[42,44]. On the other hand, schools that are predominantly white may have higher academic achievement due to the higher-caliber

teachers and higher levels of cultural and social capital[44]. Since we are products of our social, economic, and political environments, it is possible that adolescents could internalize the adverse consequences of attending racially-segregated and resource-deprived school environments.

Though there are important academic implications, little is known about the health consequences of attending racially homogenous schools during adolescence. It is possible that the racial/ethnic structure of schools may impact adolescent obesity through its impact on individual health behaviors. School racial/ethnic composition may limit an adolescent's access to quality dietary options, ultimately constraining his/her agency to make healthy food choices. High schools that have a higher percentage of black students are more likely to be located in close proximity to fast food restaurants than schools that are predominantly white[47]. These adolescents develop poorer eating habits and are more likely to be overweight/obese[48]. Furthermore, adolescent obesity is higher in schools with a higher proportion of students from racial/ethnic minorities and low-SES households[49].

Given the growing public health burden and disparities in the development of obesity, it is important to investigate the social and economic risk factors that contribute to this disease. Since schools play a large role in the social, emotional, and physical development of adolescents, understanding how the racial/ethnic structure of these environments impact chronic disease is critical in combatting this burden. Very few public health studies have explored the link between the racial/ethnic structure of schools and chronic health outcomes. To our knowledge, this is one of the first studies that will estimate the relationships between both school racial/ethnic composition and diversity, and adolescent obesity in a nationally-representative study. The primary

objective of this study is to determine if there are associations between school racial/ethnic composition and diversity experienced in adolescence and adolescent overweight/obesity. Our secondary objective is to investigate whether these associations differ by an individual's race/ethnicity.

METHODS

Study Design and Population

For this study, we used data from Wave I (1994-1995) of the National Longitudinal Study of Adolescent to Adult Health (Add Health). Beginning in 1994, Add Health was a longitudinal study that followed adolescents in grades 7-12 into adulthood. This study utilized a complex sampling design and stratification method in order to derive a nationally representative sample of U.S. high schools. Using a sampling frame based on a database from the Quality Education Data, Inc., Add Health derived a school-based sample of adolescents living in the U.S. Systematic sampling methods and implicit stratification were used to ensure that the schools in Add Health were representative of all U.S. schools regarding the following factors: region, urbanicity, size, type, and racial composition[50].

The sampled high schools had to include 11th graders and at least 30 enrolled students. Seventy-nine percent of sampled schools participated in the study, resulting in 80 high schools represented in the Add Health sample. Once high schools were sampled, feeder (middle) schools were identified to capture students in grades 7-8 from the 80 communities in which the high schools were located. These feeder schools were required to include the 7th grade and had to send at least 5 graduates to one of the 80 high schools in the Add Health sample. Altogether, there were 132 U.S. schools included in the Add Health sample: 80 high schools and 52 feeder schools. There was not a 1 to 1 ratio of high schools to feeder schools because some high schools included students from grades 7-12, and thus, separate feeder schools were not recruited for these high schools[50].

Given that this was a school-based sample, both in-school questionnaires and in-home interviews were administered. From the sampled schools, there were 90,118 students in grades 7-12 who participated in the in-school questionnaires during Wave I. Of these students, 20,745 adolescents were randomly selected, based on grade and sex, to be in the "core sample" to participate in the in-home interviews. Only those students who participated in the in-school questionnaires were eligible to be selected to participate in the in-home interviews. In addition to the random selection by grade and sex, investigators oversampled based on the following criteria: ethnicity, disability status, school saturation, and genetic factors (e.g., twins)[50].

Adolescent Obesity

The outcome of interest for this study was adolescent obesity, which was assessed using body mass index (BMI). Self-reported height and weight data were collected during the in-home interview of Wave I. Height was reported in feet and inches, while weight was reported in pounds. We converted the adolescents' height and weight data into meters and kilograms, respectively. BMI was calculated as kilograms/meters^2. Categories for adolescent obesity were determined using the criteria from the Centers for Disease Control and Prevention (CDC), which is based on standardized age- and sex-specific growth charts. Using the CDC's criteria, adolescent obesity was categorized as: (1) not overweight/obese ($< 85^{th}$ percentile), and (2) overweight/obese ($\geq 85^{th}$ percentile)[51-52].

Individual Race/Ethnicity

Adolescents reported their race/ethnicity during the in-home interview of Wave I. Individual race/ethnicity consisted of the following categories: Hispanic, Non-Hispanic White, Non-Hispanic Black, Asian/Pacific Islander, American Indian/Native American, and Other. We combined Asian/Pacific Islander, American Indian/Native American, and Other into one category.

School Racial/Ethnic Composition and Entropy

The exposures of interest for this study were school racial/ethnic composition and school entropy (i.e., diversity) within an adolescent's school during Wave I (1994-1995). We were not able to assess school segregation operating at the district level in Add Health, so we decided to focus on school racial/ethnic composition and diversity. Investigating the health impact of the proportion of black students within a school was important given the historical and political context of *Brown*, which was primarily decided to address Black-White segregation in U.S. public schools. In addition, given that the Hispanic population has increased in the U.S. in recent years and that Hispanics are experiencing higher levels of segregation[36], it was also necessary to incorporate the proportion of non-white students within a school. Furthermore, with these increases in minority student enrollment in U.S. public schools in the 1980s and 1990s[36], we thought it was critical to explore a measure of school diversity, across multiple racial/ethnic groups.

School racial/ethnic composition was defined in two ways: (1) the proportion of the total student population within each Add Health school that was non-Hispanic

Black; and (2) the proportion of the total student population within each Add Health school that was non-White. School entropy, a measure of racial/ethnic diversity within each Add Health school, was calculated using the following formula:

$$E_i = \sum_{r_i=1}^{n_i} Q_{r_i} \ln(\frac{1}{Q_{r_i}})$$

n_i represents the total number of racial/ethnic groups in the ith school. r_i represents the specific racial/ethnic group in the ith school. Q_{r_i} represents the total students in the ith school made up of the specific racial/ethnic group of interest. School entropy is an index that theoretically ranges from 0 (minimum diversity) to 1 (maximum diversity), after standardization. For this multi-group measure of entropy, a value of 0 was assigned to schools where a single racial/ethnic group was present. A value of 1 was assigned to schools where all racial/ethnic groups were represented equally in the student population[36].

School racial composition (proportion black students and proportion non-white students) was divided into five categories: (1) low: 0.00 – 0.10, (2) 0.11 – 0.25, (3) 0.26 – 0.50, (4) 0.51 – 0.75, and (5) high: 0.76 – 1.00. School entropy (multi-group, standardized) was also divided into five categories: (1) low: 0.02 – 0.10, (2) 0.11 – 0.25, (3) 0.26 – 0.50, (4) 0.51 – 0.75, and (5) high: 0.76 – 0.94. While school racial/ethnic composition was an absolute measure of the racial structure of a school, school entropy was a proxy for the levels of diversity within a school, and the degree to which the racial/ethnic groups within a school are represented equally.

Covariates: School

School-level covariates included: size, urbanicity, and type. Schools were divided into three categories, based on the definition of school size from the National Center for Education Statistics (NCES). These categories included: (1) *small*: 1 – 400 students, (2) *medium*: 401 – 1,000 students, and (3) *large*: 1,001 – 4,000 students. A school's urbanicity was determined according to the NCES and QED classification of the geographic areas in which the schools reside. This classification included:

(a) Central city of a Consolidated Metropolitan Statistical Area (CMSA) or Metropolitan Statistical Area (MSA) with population of 250,000 or more
(b) Central city of a CMSA or MSA, but not designated as a large central city
(c) Place within the CMSA or MSA of a large central city
(d) Place within the CMSA or MSA of a mid-size central city
(e) Place not within a CMSA or MSA, but with a population of 25,000 or more and defined as urban
(f) Place not within a CMSA or MSA with a population of at least 2,500, but less than 25,000
(g) Place not within a CMSA or MSA and designated as rural
(h) Place within a CMSA or MSA designated as rural

In this study, schools were categorized as: (1) *urban*: included schools located within items *(a)* and *(b)*; (2) *suburban*: included schools located within items *(c) – (f)*; and (3) *rural*: included schools located within items *(g)* and *(h)*. Regarding school type, schools were classified as either public or private.

Covariates: Neighborhood

During Wave III (2001-2002), neighborhood-level data – corresponding to the adolescents' areas of residence at Wave I (1994-1995) – was merged retrospectively in Add Health. This neighborhood data was derived from the 1990 U.S. Census[50]. Neighborhood-level covariates included: poverty and racial composition. For

neighborhood poverty, we used the proportion of residents in each adolescents' census tract who were living below the federal poverty level. Regarding the neighborhood racial composition, we used the proportion of residents who were non-Hispanic Black when the exposure of interest was the proportion black students within a school; and we used the proportion of residents who were non-White when the exposure of interest was the proportion of non-White students within a school.

Covariates: Individual

Individual-level covariates included: age, gender, parental education, and physical activity. An adolescent's age (years) and gender (male vs. female) was self-reported during the in-home interview of Wave I. The educational attainment levels of an adolescent's parents were self-reported by the parents in the in-home interview. In our study, parental education was determined to be the highest educational attainment of either the mother or the father. Parental education was collapsed into the following categories: less than high school, high school diploma, some college or an Associate's degree, and college degree or higher.

An adolescent's physical activity levels were defined using a previous categorization based on Add Health data. Physical activity was determined from how many times per week (during Wave I) the participant engaged in the following three groups of activities: (a) rollerblading, skating, skate-boarding, and bicycling; (b) baseball, football, basketball, and soccer; and (c) jogging, walking, jumping rope, dancing, and karate. Adolescents' assessed their average weekly participation in these three groups of activities as (1) not at all, (2) 1 or 2 times, (3) 3 or 4 times, or (4) 5 or more times. We then assigned the following numerical values:

Not at all = 0
1 or 2 times = 1.5
3 or 4 times = 3.5
5 or more times = 5

We summed across the three groups of activities and created a physical activity score, which was categorized as: not physically active (< 5 times/week) vs. physically active (≥ 5 times/week)[53].

Statistical Analysis

The predicted prevalence for adolescent obesity was estimated from cluster- and sample-weight adjusted logistic regression models in SUDAAN. Analyses included unadjusted models, as well as models adjusted for covariates in three stages. Stage 1 included logistic regression models adjusted for school-level covariates (size, type, urbanicity) only. Stage 2 included logistic regression models adjusted for school-level covariates and neighborhood-level covariates (poverty, racial/ethnic composition). Stage 3 included the fully-adjusted logistic regression models, which were adjusted for school-level covariates, neighborhood-level covariates, and individual-level covariates (age, gender, parental education, physical activity). All models were then stratified by individual race/ethnicity. Unadjusted models, as well as models from stages 1 & 2, are included in the Appendix.

RESULTS

Socio-Demographic Characteristics of Schools, Neighborhoods, and Participants

Table 1 displays the characteristics of the schools attended by the adolescents who participated in Wave I. Regarding the demographic characteristics of the Add Health schools, Hispanic adolescents were most likely to attend large, urban schools located in the West and South. Non-Hispanic White and non-Hispanic Black adolescents were most likely to attend medium-sized, suburban schools located in the Midwest and South. Asian/Native American/Other adolescents were most likely to attend large, suburban schools located in the West and Northeast.

The racial/ethnic composition of the schools attended by the adolescents in this study population varied by race/ethnicity. Hispanic, non-Hispanic White, and Asian/Native American/Other adolescents were most likely to attend schools that consisted of 0-10% Black students, while non-Hispanic Black adolescents were most likely to attend schools that consisted of 76-100% Black students. However, Hispanic, non-Hispanic Black, and Asian/Native American/Other adolescents were most likely to attend schools that consisted of 76-100% non-White students and schools that had moderate-to-high levels of racial/ethnic diversity. Non-Hispanic White adolescents were most likely to attend schools that consisted of 0-10% non-White students and schools with low levels of racial/ethnic diversity.

The sociodemographic characteristics of the adolescents and their respective neighborhoods in Wave I of Add Health are described in Table 2. Hispanic and non-Hispanic Black adolescents were more likely to have at least one parent with a High School Diploma/GED, while non-Hispanic White and Asian/Native American/Other

adolescents were more likely to have at least one parent with a Bachelor's Degree or higher. Regarding the outcome of interest, approximately 25% of the adolescents in Wave I of Add Health were overweight/obese. However, 29.1% and 31.3% of Hispanic and non-Hispanic Black adolescents, respectively, were overweight/obese; while 23.8% and 19.5% of non-Hispanic White and Asian/Native American/Other adolescents, respectively, were overweight/obese.

Non-Hispanic White and non-Hispanic Black adolescents in Add Health lived in neighborhoods with the lowest and highest proportions of the reported adverse, socioeconomic characteristics. On average, non-Hispanic White adolescents lived in neighborhoods that consisted of 11% of residents living below the federal poverty level, and with 6% and 9% of residents who were Black and non-White, respectively. On average, non-Hispanic Black adolescents lived in neighborhoods that consisted of 26% of residents living below the federal poverty level, and with 54% and 58% of residents who were Black and non-White, respectively.

Proportion Black Students and Overweight/Obesity

Tables 3a-c present the adjusted prevalence ratios for overweight/obesity by school racial/ethnic composition and school entropy (multi-group), stratified by individual race/ethnicity. In Table 3a, there were null associations between the proportion of Black students in a school and the prevalence of overweight/obesity, among Hispanic, non-Hispanic White, and non-Hispanic Black adolescents. Among Asian/Native American/Other adolescents, those who attended schools with 26-50% Black students had a significantly higher likelihood of adolescent overweight/obesity, after adjusting for school-, neighborhood-, and individual-level characteristics.

Compared to those Asian/Native American/Other adolescents who attended schools with 0-10% Black students, those who attended schools with 26-50% Black students were 2.11 times (95% CI: 1.08, 4.15) more likely to be overweight/obese. In Figure 1a, the predicted prevalence of overweight/obesity among Asian/Native American/Other adolescents appeared to be higher as the proportion of Black students within a school was higher. However, there is no meaningful dose-response relationship between the proportion of Black students and overweight/obesity among adolescents of this racial/ethnic sub-group.

Though the overall associations between the proportion of Black students within a school and overweight/obesity, among Hispanic and non-Hispanic Black adolescents, were null, there are two findings from the Appendix that are worth noting. In analyses adjusted for school- and neighborhood-level characteristics, Hispanic adolescents who attended schools with 11-25% Black students had an increased likelihood of overweight/obesity, compared to Hispanic adolescents who attended schools with 0-10% Black students. However, this association among Hispanic adolescents was attenuated after adjusting for individual-level characteristics. In unadjusted analyses, non-Hispanic Black adolescents who attended schools with 76-100% Black students had a higher likelihood of overweight/obesity, compared to non-Hispanic Black adolescents who attended schools with 0-10% Black students. However, this association among non-Hispanic Black adolescents was attenuated after adjusting for school-level characteristics.

Proportion Non-White Students and Overweight/Obesity

In Table 3b, there were null associations between the proportion of non-White students in a school and the prevalence of overweight/obesity, among Hispanic, non-Hispanic White, and Asian/Native American/Other adolescents. Among non-Hispanic Black adolescents, those who attended schools with 11-25% non-White students had a significantly higher likelihood of adolescent overweight/obesity, after adjusting for school-, neighborhood-, and individual-level characteristics. Compared to those non-Hispanic Black adolescents who attended schools with 0-10% non-White students, those who attended schools with 11-25% non-White students were 1.83 times (95% CI: 1.07, 3.11) more likely to be overweight/obese.

Though there was an association between the proportion of non-White students in a school and overweight/obesity among non-Hispanic Black adolescents, the predicted prevalences from Figure 1b demonstrated that this was not a dose-response relationship. Additionally, the predicted prevalence of overweight/obesity among Hispanic adolescents in Figure 1b appeared to be lower as the proportion of non-White students within a school increased. However, as mentioned above, there is no meaningful, overall association among Hispanic adolescents.

School Entropy and Overweight/Obesity

In Table 3c, there were null associations between the levels of racial/ethnic diversity in a school and the prevalence of overweight/obesity among Hispanic, non-Hispanic White, and Asian/Native American/Other adolescents. Among non-Hispanic Black adolescents, those who attended schools that were moderately diverse (i.e., school entropy of 0.11-0.25) had a significantly lower likelihood of adolescent overweight/obesity, after adjusting for school-, neighborhood-, and individual-level

characteristics. Compared to those non-Hispanic Black adolescents who attended schools with the lowest levels of racial/ethnic diversity, those who attended schools that were moderately diverse were 0.81 times (95% CI: 0.66, 0.98) as likely to be overweight/obese.

Although we observed an association among non-Hispanic Black adolescents, the findings from Figure 1c demonstrated that there does not appear to be a meaningful dose-response relationship between school diversity levels and the predicted prevalence of overweight/obesity in this racial/ethnic sub-group. Additionally, the predicted prevalence of overweight/obesity among Hispanic adolescents appeared to be higher as the levels of racial/ethnic diversity within a school increased. However, as mentioned above, there is no meaningful, overall association among Hispanic adolescents.

Though the overall associations between the proportion of Black students within a school and overweight/obesity, among non-Hispanic White adolescents, were null, there is a finding from the Appendix that is worth noting. In analyses adjusted for school- and neighborhood-level characteristics, Non-Hispanic White adolescents who attended schools with higher levels of racial/ethnic diversity had a higher likelihood of overweight/obesity, compared to non-Hispanic White adolescents who attended schools with lower levels of racial/ethnic diversity. However, this association among non-Hispanic White adolescents was attenuated after adjusting for individual-level characteristics.

DISCUSSION

Our study found that there were racial/ethnic differences in the types of schools attended by the adolescents in Add Health. Non-Hispanic White adolescents were most likely to attend predominantly White schools, while non-Hispanic Black adolescents were most likely to attend predominantly Black/non-White schools. Hispanic and Asian/Native American/Other adolescents were most likely to attend schools with moderate-to-high levels of racial/ethnic diversity. Also, the prevalence of overweight/obesity varied by race/ethnicity, with Asian/Native American/Other adolescents with the lowest prevalence, and non-Hispanic Black adolescents with the highest prevalence.

There were also racial/ethnic differences in the associations between school racial/ethnic composition and diversity and adolescent overweight/obesity, after adjusting for school-, neighborhood-, and individual-level characteristics. Among Asian/Native American/Other adolescents, attending schools that consisted of 26-50% Black students was associated with a higher prevalence of adolescent overweight/obesity. Furthermore, the associations between the proportion of Black students within a school and the prevalence of overweight/obesity among Hispanic and non-Hispanic Black students were attenuated after the adjustment of covariates. This attenuation suggests that individual-level characteristics and school-level characteristics could explain these associations among Hispanic and non-Hispanic Black students, respectively.

Among non-Hispanic Black adolescents, attending schools that consisted of 11-25% non-White students was also associated with a higher prevalence of adolescent

overweight/obesity. Additionally, attending moderately diverse schools was associated with a lower prevalence of adolescent overweight/obesity among non-Hispanic Black adolescents. Finally, it is critical to highlight that school racial/ethnic composition and diversity levels do not impact the likelihood of overweight/obesity among non-Hispanic White adolescents; their prevalence was the lowest among the racial/ethnic groups across school-level exposures.

The results from this study are consistent with previous research that established a relationship between school racial composition and adolescent obesity[47]. Although we were unable to account for neighborhood food quality in this study, it is possible that schools that are predominantly Black or non-White are located in areas with poorer dietary options. In addition, our study's findings are similar to the educational studies literature, which has established racial/ethnic differences in the association between school racial/ethnic composition and academic achievement[41-46]. It appears that students of color who attend schools with a higher proportion of Black or non-White students are more likely to have worse academic and chronic health outcomes. The lack of economic resources and social capital could contribute to inadequate educational opportunities and constraints on important dietary behaviors learned in adolescence.

The racial/ethnic structure of schools may also impact adolescent obesity through the harmful effects of negative intergroup contact and perceived discrimination experienced in racially homogenous schools. Black and Hispanic students attending predominantly White schools are more likely to experience discrimination from their teachers and peers, compared to their white counterparts[54-57]. In fact, Black students attending predominantly White schools were found to have worse adult health outcomes compared to their white peers, as a result of the negative consequences of perceived

racial discrimination they experienced being in the minority[58]. Though this association was only found using adult health outcomes, it is plausible that these experiences of racial discrimination impact adolescent health through the negative physiological consequences of stress[34]. Repeated stress can produce higher levels of cortisol, causing an increased risk of abdominal obesity[59-60]. In addition, stress resulting from discrimination could lead to poor dietary behaviors and a reduction in physical activity, ultimately resulting in adolescent obesity[61-62]. However, very little is known about the adverse health consequences for Black students who attend predominantly Black schools.

Given that we only examined the association between the racial/ethnic structure of schools and obesity during adolescence, this study was limited by the cross-sectional nature of the design. Though reverse causality is likely impossible, the design of this study makes it difficult to infer causality. Despite this limitation, our findings suggest that there are potential racial/ethnic differences in this association. It is also worth noting that height and weight data were self-reported during Wave I of Add Health. Therefore, there was some potential for measurement error of body mass index. Overweight and obese adolescents may underestimate their weight, resulting in the underestimation of the associations between school racial/ethnic composition and diversity and adolescent overweight/obesity. However, a previous study found that self-reported height and weight data obtained from Add Health was very reliable (ICCs ≥ 0.92)[63]. In addition, Add Health is an observational study and there are likely unmeasured confounders for which we were unable to adjust. However, we attempted to remedy this limitation by adjusting for relevant confounding factors at three distinct levels: the school, the neighborhood, and the individual. The findings from this study

suggest that the racial/ethnic structure of schools are an important social determinant of adolescent health, even above and beyond the impact of residential characteristics.

This study also had four important strengths that are worth highlighting. First, Add Health was a nationally-representative cohort study, which used a complex stratification and sampling method. This design increased the external validity of our findings, pertaining to adolescents attending public and private schools in the U.S. between 1994 and 1995. Second, Wave I of Add Health consisted of a large study population (N=20,745), which increased the precision of our estimates. Third, to our knowledge, this is one of the first studies that investigated the association between both school racial/ethnic composition, as well as diversity, and adolescent obesity. Characterizing the racial/ethnic composition as the proportion of students who were black and the proportion of students who were non-white allowed us to assess the absolute racial/ethnic make-up within a school. On the other hand, school entropy allowed us to assess how diverse a school was and attempted to measure the degree to which students of various racial/ethnic groups were represented equitably within a school. By measuring the exposure in these different ways, we were able to provide a more comprehensive investigation of the impact of the racial/ethnic structure of school environments on adolescent obesity. We were able to differentiate the relationship between absolute measures vs. diversity measures of racial/ethnic structure of schools and adolescent health. Our study's findings suggest that the absolute measures (i.e., the proportion of black and non-white students within a school) are associated with an increased prevalence of adolescent obesity. Lastly, we were able to control for potential confounding at the three levels described above. Given the complex processes that contribute to the racial/ethnic structure of schools and school systems, it was important

to establish a more comprehensive understanding of how school racial composition and diversity impact adolescent obesity, even after adjusting for potential confounders at these three levels.

Future research should investigate the potential mediating factors that play a role in the relationship between the racial/ethnic structure of schools and adolescent obesity. It is possible that the racial differences that we found could be contributed to differences in experiences of racial/ethnic discrimination or feeling of school connectedness/isolation. Furthermore, the findings from this study provide evidence that the racial/ethnic structure of school environments is important for the health of students of color, particularly non-Hispanic Black adolescents. For these adolescents, the proportion of non-White students may be harmful, while the levels of racial/ethnic diversity may be protective for their likelihood of overweight/obesity. Additional research is warranted that explores how attending racially/ethnically diverse schools could be beneficial for the chronic health of non-Hispanic Black adolescents.

Given the constraints of the data, our analyses were not able to capture the effects of a larger construct – the racial segregation of schools within districts – on adolescent obesity. Since schools operate within school districts across the U.S., it is critical to investigate the spatial and geographic processes that contribute to the increasing burden of obesity among our country's youth. School racial/ethnic composition and diversity are plausible and relevant, social determinants of adolescent obesity that need further investigation. Finally, given the racial/ethnic shifts in the composition of U.S. public schools since the late 20th century the racial/ethnic structure of schools has also become a timely and relevant exposure. Our findings can help contribute to the contemporary educational policy debate surrounding whether attending segregated and racially-

homogenous schools are harmful or beneficial for the health of children and adolescents.

TABLE 1. School Characteristics by Race/Ethnicity among Adolescents who Participated in Wave I of Add Health, 1994-1995*.

	Hispanic	Non-Hispanic White	Non-Hispanic Black	Asian/Native American/ Other
School Region				
West	886; 31.2 (5.5)	1,095; 11.4 (1.6)	474; 6.0 (1.9)	767; 48.1 (9.2)
Midwest	160; 10.9 (3.6)	2,430; 33.9 (3.9)	502; 19.6 (6.1)	162; 16.3 (4.5)
South	906; 39.0 (7.3)	2,714; 38.4 (3.2)	1,913; 68.6 (6.3)	136; 15.3 (3.7)
Northeast	419; 18.9 (5.6)	1,445; 16.4 (1.9)	223; 5.7 (1.7)	169; 20.4 (7.1)
School Size				
Small (1-400 students)	141; 13.4 (4.6)	1,394; 19.9 (4.2)	452; 17.4 (6.6)	71; 12.0 (5.5)
Medium (401-1,000 students)	447; 31.3 (7.0)	3,399; 48.6 (5.8)	1,330; 53.1 (8.7)	294; 40.8 (9.9)
Large (1,001-4,000 students)	1,783; 55.3 (8.1)	2,891; 31.5 (6.1)	1,330; 29.6 (7.6)	869; 47.2 (10.7)
School Type				
Public	2,284; 93.5 (3.0)	6,940; 92.1 (2.6)	2,962; 96.3 (1.6)	1,103; 83.6 (7.0)
Private	87; 6.5 (3.0)	744; 7.9 (2.6)	150; 3.7 (1.6)	131; 16.4 (7.0)
School Urbanicity				
Urban	1,289; 56.1 (7.9)	1,632; 18.1 (3.5)	908; 25.9 (6.3)	292; 28.3 (7.6)
Suburban	1,000; 39.8 (7.7)	4,109; 60.3 (6.0)	1,791; 58.3 (8.5)	883; 65.9 (8.6)
Rural	82; 4.1 (2.7)	1,943; 21.6 (6.0)	413; 15.8 (7.6)	59; 5.8 (3.8)
Proportion Black Students				
0.00 – 0.10	1,244; 58.0 (8.5)	5,712; 76.1 (4.1)	323; 9.9 (2.4)	516; 55.4 (10.8)
0.11 – 0.25	750; 21.2 (8.4)	709; 6.9 (2.4)	488; 9.0 (3.1)	499; 23.8 (11.5)
0.26 – 0.50	266; 14.4 (4.7)	994; 14.1 (3.3)	874; 27.1 (6.7)	102; 12.7 (4.7)
0.51 – 0.75	72; 5.7 (2.9)	134; 2.3 (1.0)	402; 16.7 (6.7)	100; 6.1 (3.4)
0.76 – 1.00	20; 0.8 (0.4)	47; 0.6 (0.3)	993; 38.1 (9.3)	15; 2.1 (1.2)
Proportion Non-White Students				
0.00 – 0.10	207; 14.3 (4.2)	4,384; 60.9 (4.9)	119; 4.0 (1.4)	106; 14.2 (4.4)
0.11 – 0.25	155; 8.6 (3.1)	1,399; 16.1 (3.6)	234; 7.7 (2.7)	107; 9.4 (3.6)
0.26 – 0.50	133; 9.4 (3.7)	872; 12.0 (3.1)	498; 17.9 (5.4)	122; 14.3 (7.4)
0.51 – 0.75	483; 29.8 (7.4)	651; 8.9 (2.3)	678; 25.5 (7.1)	195; 28.2 (8.4)
0.76 – 1.00	1,374; 37.9 (9.2)	290; 2.0 (0.7)	1,551; 45.0 (9.1)	702; 33.8 (11.8)
School Entropy (Multi-Group, Standardized)				
0.02 – 0.10	66; 4.9 (2.3)	2,793; 39.3 (6.0)	321; 14.0 (7.3)	39; 7.3 (3.4)
0.11 – 0.25	144; 10.0 (3.8)	1,143; 17.2 (4.3)	635; 24.4 (9.0)	45; 5.4 (2.0)
0.26 – 0.50	742; 23.2 (7.3)	2,272; 27.8 (4.6)	851; 28.6 (6.6)	254; 25.6 (9.0)
0.51 – 0.75	792; 46.5 (8.5)	1,094; 12.7 (2.9)	754; 26.2 (7.3)	322; 36.7 (9.5)
0.76 – 0.94	608; 15.4 (6.2)	294; 2.9 (1.3)	519; 6.8 (3.7)	572; 25.1 (11.4)

** N=20,745 adolescents participated in the In-School Questionnaire and In-Home Interview in Wave I of Add Health. All statistics are reported as: N; Weighted % (SE).*

Table 2. Individual and Neighborhood Characteristics by Race/Ethnicity among Adolescents who Participated in Wave I of Add Health, 1994-1995.

	Hispanic	Non-Hispanic White	Non-Hispanic Black	Asian/Native American/Other
Individual Characteristics				
Age				
10 – 13 years old	347; 27.3 (5.5)	1,780; 29.9 (3.9)	712; 25.5 (5.5)	189; 26.6 (5.6)
14 – 15 years old	841; 35.0 (2.2)	2,817; 34.7 (1.4)	1,136; 33.5 (1.7)	408; 33.7 (2.3)
16 – 19 years old	1,180; 37.7 (4.3)	3,070; 35.3 (2.7)	1,243; 40.9 (5.0)	634; 39.7 (5.4)
Gender				
Male	1,153; 50.4 (2.0)	3,714; 50.3 (1.0)	1,375; 45.1 (1.2)	636; 52.3 (2.5)
Parent's Educational Attainment				
Less than HS	506; 28.0 (3.1)	416; 6.4 (0.8)	167; 8.4 (1.0)	81; 11.8 (2.7)
HS Diploma/GED	588; 33.1 (2.0)	2,279; 34.6 (1.5)	678; 38.2 (2.4)	186; 23.1 (3.0)
Some College or Associate's Degree	290; 12.5 (1.2)	1,247; 18.1 (0.7)	373; 17.8 (1.2)	136; 14.7 (2.1)
Bachelor's Degree or Higher	454; 26.4 (2.2)	2,937; 40.9 (1.9)	1,395; 35.5 (2.8)	551; 50.5 (4.0)
Physical Activity				
Less than 5 times/week	1,216; 36.9 (1.6)	3,536; 34.0 (1.1)	1,403; 38.0 (1.6)	525; 31.8 (2.0)
5 or more times/week	2,010; 63.1 (1.6)	6,660; 66.0 (1.1)	2,530; 62.0 (1.6)	1,015; 68.2 (2.0)
Adolescent Overweight/Obesity	657; 29.1 (1.5)	1,702; 23.8 (0.9)	875; 31.3 (1.5)	223; 19.5 (3.2)
Neighborhood Characteristics				
Proportion Residents Living Below Poverty Level, 1990**	0.18 (0.01)	0.11 (0.01)	0.26 (0.02)	0.13 (0.02)
Proportion Residents who are Black, 1990**	0.10 (0.01)	0.06 (0.01)	0.54 (0.03)	0.11 (0.02)
Proportion Residents who are Non-White, 1990**	0.30 (0.03)	0.09 (0.01)	0.58 (0.03)	0.35 (0.05)

**N=20,745 adolescents participated in the In-School Questionnaire and In-Home Interview in Wave I of Add Health. Statistics are reported as: N; Weighted % (SE), unless otherwise indicated.*

***Statistics are reported as: Weighted Mean (SE).*

Table 3a. Adjusted Prevalence Ratios* for Adolescent Obesity by Proportion Black Students within a School and Individual Race/Ethnicity.

Proportion Black Students	All Adolescents	Hispanic	Non-Hispanic White	Non-Hispanic Black	Asian/Native American/ Other
0.00 – 0.10	1.00	1.00	1.00	1.00	1.00
0.11 – 0.25	1.15 (1.03, 1.29)	1.16 (0.87, 1.56)	1.13 (0.95, 1.35)	1.08 (0.74, 1.56)	1.22 (0.74, 2.00)
0.26 – 0.50	1.07 (0.92, 1.26)	1.22 (0.91, 1.65)	1.04 (0.83, 1.30)	0.96 (0.72, 1.28)	2.11 (1.08, 4.15)
0.51 – 0.75	1.13 (0.94, 1.35)	0.80 (0.38, 1.65)	1.12 (0.91, 1.39)	1.07 (0.78, 1.46)	1.24 (0.45, 3.39)
0.76 – 1.00	1.15 (0.93, 1.42)	1.52 (0.45, 5.06)	0.95 (0.47, 1.92)	1.07 (0.80, 1.42)	0.97 (0.13, 7.52)

Table 3b. Adjusted Prevalence Ratios* for Adolescent Obesity by Proportion Non-White Students within a School and Individual Race/Ethnicity.

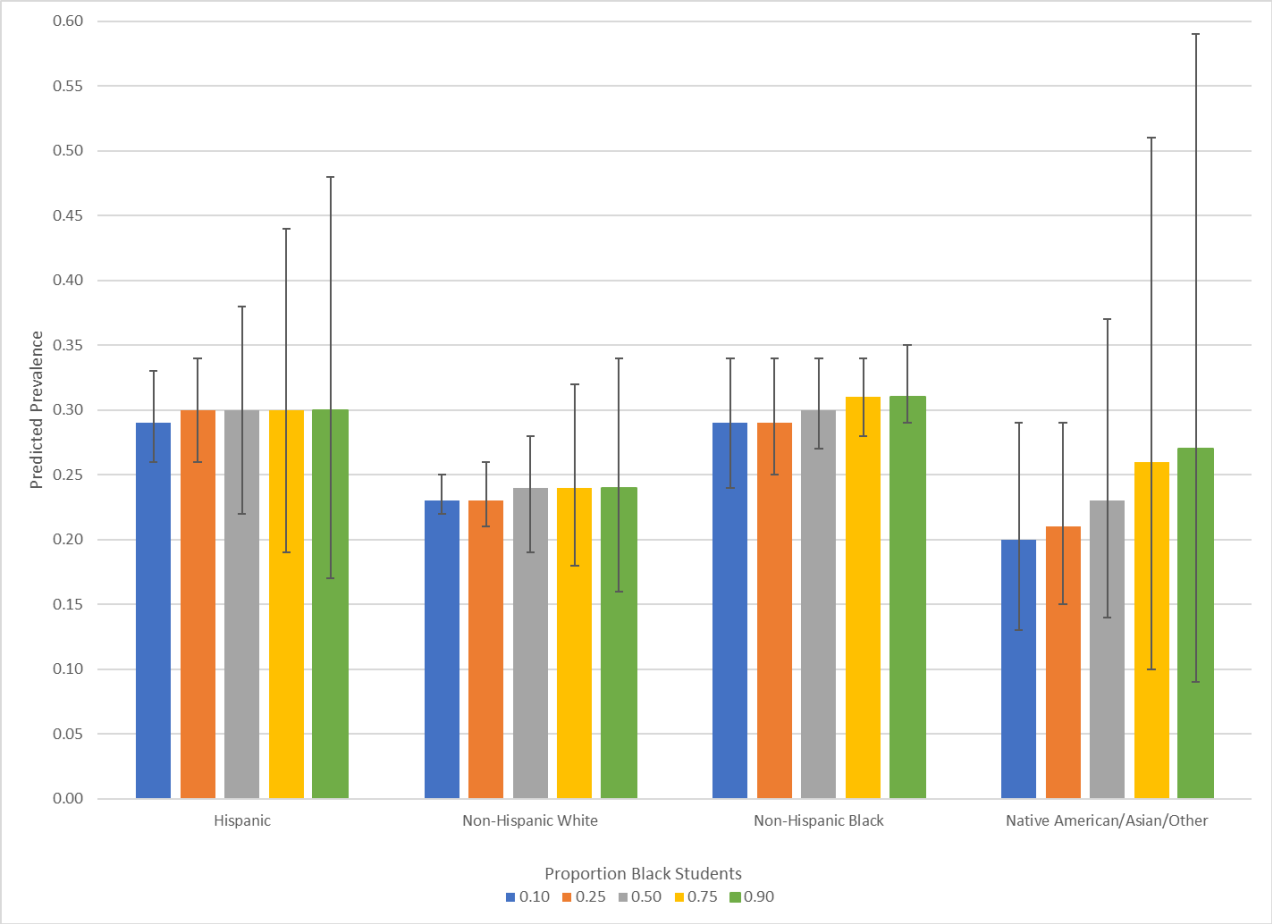
Proportion Non-White Students	All Adolescents	Hispanic	Non-Hispanic White	Non-Hispanic Black	Asian/Native American/ Other
0.00 – 0.10	1.00	1.00	1.00	1.00	1.00
0.11 – 0.25	1.00 (0.88, 1.14)	1.10 (0.71, 1.70)	0.93 (0.78, 1.11)	1.83 (1.07, 3.11)	0.55 (0.15, 2.03)
0.26 – 0.50	0.96 (0.79, 1.16)	0.78 (0.43, 1.41)	0.96 (0.75, 1.23)	1.31 (0.79, 2.17)	0.71 (0.23, 2.19)
0.51 – 0.75	1.15 (0.97, 1.35)	1.11 (0.76, 1.63)	1.09 (0.85, 1.40)	1.54 (0.94, 2.51)	0.93 (0.37, 2.37)
0.76 – 1.00	1.07 (0.88, 1.32)	0.95 (0.60, 1.49)	1.02 (0.70, 1.48)	1.52 (0.93, 2.50)	0.80 (0.21, 3.09)

Table 3c. Adjusted Prevalence Ratios* for Adolescent Obesity by School Entropy (Multi-Group, Standardized) and Individual Race/Ethnicity.

School Entropy	All Adolescents	Hispanic	Non-Hispanic White	Non-Hispanic Black	Asian/Native American/ Other
0.02 – 0.10	1.00	1.00	1.00	1.00	1.00
0.11 – 0.25	0.94 (0.79, 1.11)	1.25 (0.66, 2.36)	0.83 (0.67, 1.03)	0.81 (0.66, 0.98)	0.72 (0.14, 3.56)
0.26 – 0.50	0.95 (0.84, 1.08)	1.11 (0.59, 2.09)	0.90 (0.78, 1.05)	0.87 (0.72, 1.05)	0.86 (0.29, 2.54)
0.51 – 0.75	1.00 (0.85, 1.18)	1.23 (0.69, 2.20)	0.94 (0.77, 1.15)	0.85 (0.68, 1.06)	0.70 (0.23, 2.07)
0.76 – 0.94	1.16 (0.96, 1.39)	1.51 (0.86, 2.66)	1.02 (0.91, 1.56)	0.88 (0.63, 1.22)	0.89 (0.29, 2.77)

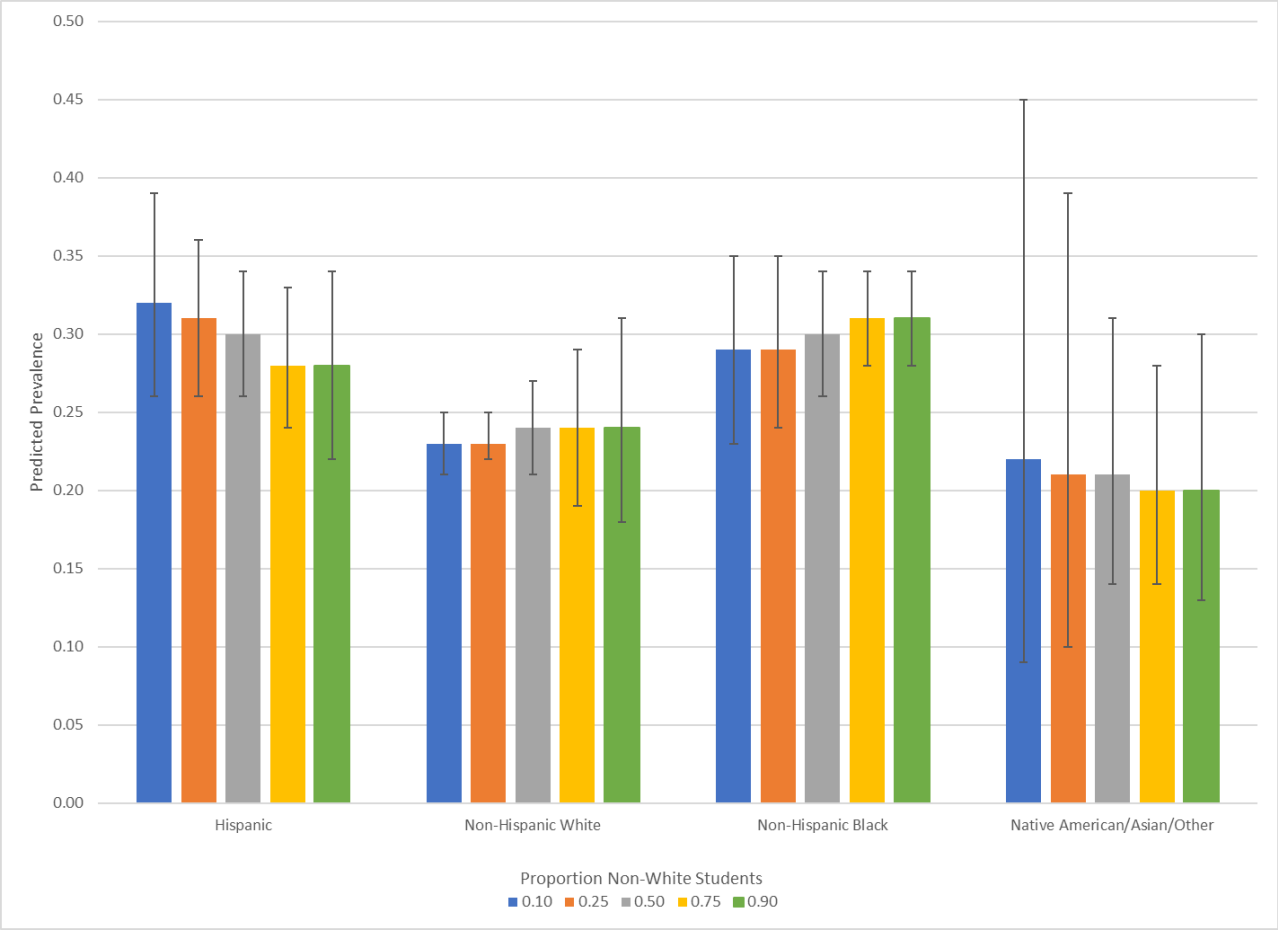
**Adjusted for school-level characteristics (school size, school urbanicity, school type), neighborhood-level characteristics (neighborhood proportion living in poverty, neighborhood proportion who are black), and individual-level characteristics (age, gender, parent's highest education, physical activity).*

Figure 1a. Adjusted* Predicted Prevalence for Adolescent Overweight/Obesity by Proportion Black Students in a School and Individual Race/Ethnicity.



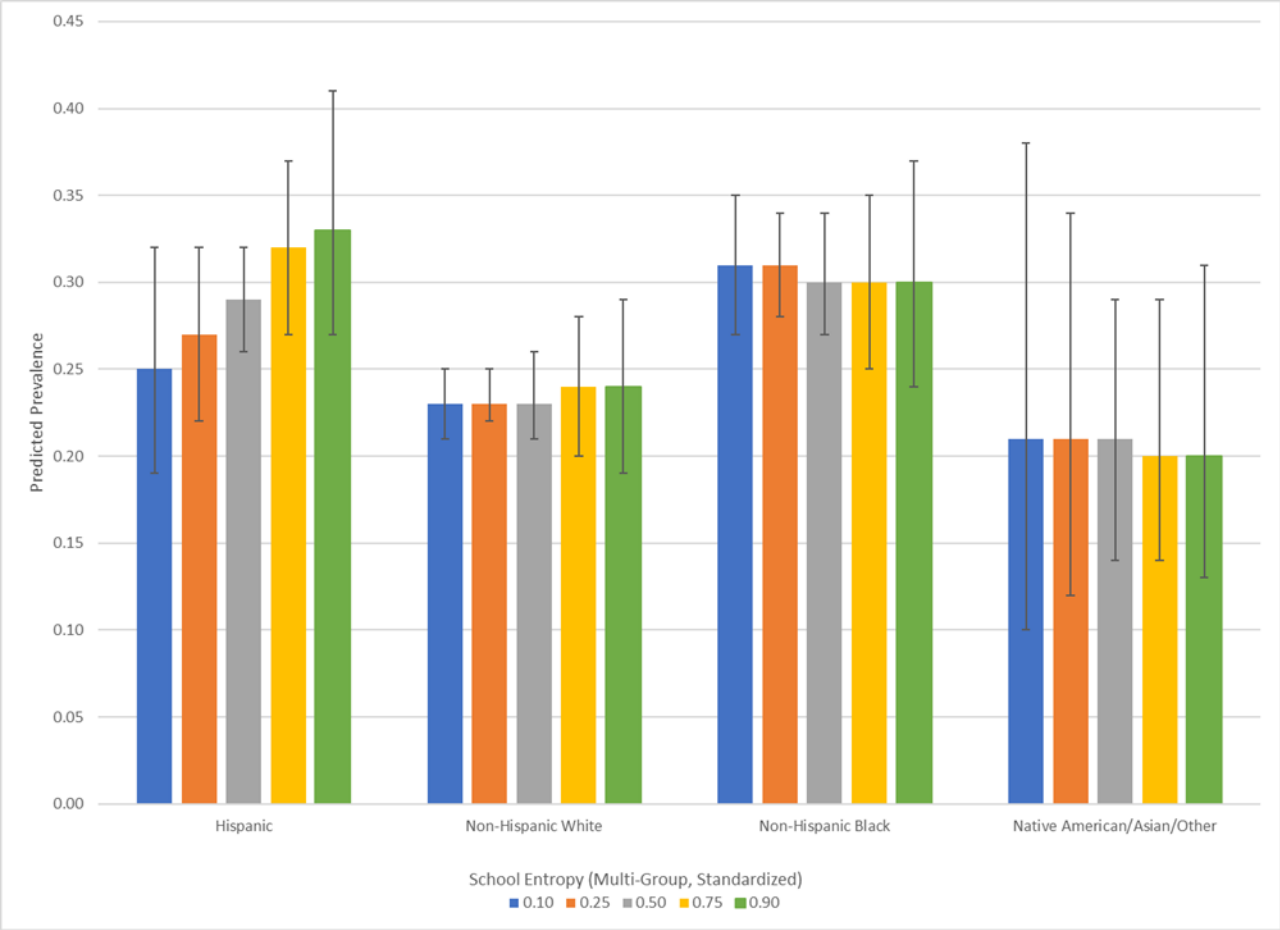
**Adjusted for school-level characteristics (school size, school urbanicity, school type), neighborhood-level characteristics (neighborhood proportion living in poverty, neighborhood proportion who are black), and individual-level characteristics (age, gender, parent's highest education, physical activity).*

Figure 1b. Adjusted* Predicted Prevalence for Adolescent Overweight/Obesity by Proportion Non-White Students in a School and Individual Race/Ethnicity.



**Adjusted for school-level characteristics (school size, school urbanicity, school type), neighborhood-level characteristics (neighborhood proportion living in poverty, neighborhood proportion who are black), and individual-level characteristics (age, gender, parent's highest education, physical activity).*

Figure 1c. Adjusted* Predicted Prevalence for Adolescent Overweight/Obesity by School Entropy (Multi-Group, Standardized) and Individual Race/Ethnicity.



**Adjusted for school-level characteristics (school size, school urbanicity, school type), neighborhood-level characteristics (neighborhood proportion living in poverty, neighborhood proportion who are black), and individual-level characteristics (age, gender, parent's highest education, physical activity).*

References

1. Ogden CL, Carroll MD, Lawman HG, Fryar CD, Kruszon-Moran D, Kit BK, et al. Trends in obesity prevalence among children and adolescents in the United States, 1988-1994 through 2013-2014. *JAMA*. 2016; 315(21):2292-9.
2. Ogden CL, Fryar CD, Hales CM, Carroll MD, Aoki Y, Freedman DS. Differences in obesity prevalence by demographics and urbanization in US children and adolescents, 2013-2016. *JAMA*. 2018; 319(23):2410-8.
3. Benjamin EJ, Blaha MJ, Chiuve SE, Cushman M, Das SR, Deo R, et al. Heart disease and stroke statistics – 2017 update: A report from the American Heart Association. *Circulation*. 2017; 135(10):e146-e603.
4. Barlow SE, Expert Committee. Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity: Summary report. *Pediatrics*. 2007; 120 Suppl 4:S164-92.
5. Liu JH, Jones SJ, Sun H, Probst JC, Merchant AT, Cavicchia P. Diet, physical activity, and sedentary behaviors as risk factors for childhood obesity: An urban and rural comparison. *Child Obes*. 2012; 8(5):440-8.
6. Singh GK, Kogan MD, Van Dyck PC, Siahpush M. Racial/ethnic, socioeconomic, and behavioral determinants of childhood and adolescent obesity in the United States: Analyzing independent and joint associations. *Ann Epidemiol*. 2008; 18(9):682-95.
7. Ogden CL, Carroll MD, Fakhouri TH, Hales CM, Fryar CD, Li X, et al. Prevalence of obesity among youths by household income and education level of head of household – United States 2011-2014. *MMWR Morb Mortal Wkly Rep*. 2018; 67(6):186-9.
8. Frederick CB, Snellman K, Putnam RD. Increasing socioeconomic disparities in adolescent obesity. *Proc Natl Acad Sci U.S.A*. 2014; 111(4):1338-42.
9. Kelly AS, Barlow SE, Rao G, Inge TH, Hayman LL, Steinberger J, et al. Severe obesity in children and adolescents: Identification, associated health risks, and treatment approaches: A scientific statement from the American Heart Association. *Circulation*. 2013; 128(15):1689-712.
10. Miech RA, Kumanyika SK, Stettler N, Link BG, Phelan JC, Chang VW. Trends in the association of poverty with overweight among US adolescents, 1971-2004. *JAMA*. 2006; 295(20):2385-93.
11. Delva J, O'Malley PM, Johnston LD. Racial/ethnic and socioeconomic status differences in overweight and health-related behaviors among American students: National trends 1986-2003. *J Adolesc Health*. 2006; 39(4):536-45.
12. Singh GK, Kogan MD, van Dyck PC. A multilevel analysis of state and regional disparities in childhood and adolescent obesity in the United States. *J Community Health*. 2008; 33(2):90-102.
13. Johnson JA 3rd, Johnson AM. Urban-rural differences in childhood and adolescent obesity in the United States: A systematic review and meta-analysis. *Child Obes*. 2015; 11(3):233-41.
14. Freedman DS, Mei Z, Srinivasan SR, Berenson GS, Dietz WH. Cardiovascular risk factors and excess adiposity among overweight children and adolescents: The Bogalusa Heart Study. *J Pediatr*. 2007; 150(1):12-17.e2.

15. Srinivasan SR, Bao W, Wattigney WA, Berenson GS. Adolescent overweight is associated with adult overweight and related multiple cardiovascular risk factors: The Bogalusa Heart Study. *Metabolism*. 1996; 45(2):235-40.
16. Weiss R, Dziura J, Burgert TS, Tamborlane WV, Taksali SE, Yeckel CW, et al. Obesity and the metabolic syndrome in children and adolescents. *N Engl J Med*. 2004; 350(23):2362-74.
17. Reilly JJ, Kelly J. Long-term impact of overweight and obesity in childhood and adolescence on morbidity and premature mortality in adulthood: Systematic Review. *Int J Obes*. 2011; 35(7):891-8.
18. Norris AL, Steinberger J, Steffen LM, Metzig AM, Schwarzenberg SJ, Kelly AS. Circulating oxidized LDL and inflammation in extreme pediatric obesity. *Obesity*. 2011; 19(7):1415-9.
19. Baker JL, Olsen LW, Sørensen TI. Childhood body-mass index and the risk of coronary heart disease in adulthood. *N Engl J Med*. 2007; 357(23):2329-37.
20. Skinner AC, Perrin EM, Moss LA, Skelton JA. Cardiometabolic risks and severity of obesity in children and young adults. *N Engl J Med*. 2015; 373(14):1307-17.
21. Must A, Strauss RS. Risks and consequences of childhood and adolescent obesity. *Int J Obes Relat Metab Disord*. 1999; 23 Suppl 2:S2-11.
22. Gunnell DJ, Frankel SJ, Nanchahal K, Peters TJ, Davey Smith G. Childhood obesity and adult cardiovascular mortality: A 57-y follow-up study based on the Boyd Orr cohort. *Am J Clin Nutr*. 1998; 67(6):1111-8.
23. Must A, Jacques PF, Dallal GE, Bajema CJ, Dietz WH. Long-term morbidity and mortality of overweight adolescents: A follow-up of the Harvard Growth Study of 1922 to 1935. *N Engl J Med*. 1992; 327(19):1350-5.
24. Lauer RM, Clarke WR. Childhood risk factors for high adult blood pressure: The Muscatine Study. *Pediatrics*. 1989; 84(4):633-41.
25. Plessy v. Ferguson, 163 U.S. 537 (1896).
26. Tischauser LV. *Jim Crow Laws: Landmarks of the American Mosaic*. Greenwood: Santa Barbara, CA; 2012.
27. Clotfelter CT. *After Brown: The Rise and Retreat of School Desegregation*. Princeton University Press: Princeton, NJ; 2004.
28. Brown v. Board of Education of Topeka, 347 U.S. 483 (1954).
29. Milliken v. Bradley, 418 U.S. 717 (1974).
30. Riddick v. School Board of the City of Norfolk, Virginia 784 F.2d 251 (4th Cir. 1986).
31. Board of Education of Oklahoma v. Dowell, 498 U.S. 237 (1991).
32. Freeman v. Pitts, 503 U.S. 567 (1992).
33. Reardon SF, Owens A. 60 years after *Brown*: Trends and Consequences of school segregation. *Annu Rev Sociol*. 2014; 40:199-218.
34. Williams DR. Race, socioeconomic status, and health: The added effects of racism and discrimination. *Ann N Y Acad Sci*. 1999; 896:173-88.
35. Orfield G, Eaton SE, The Harvard Project on School Desegregation. *Dismantling Desegregation: The Quiet Reversal of Brown v. Board of Education*. New Press: New York, NY; 1996.
36. Reardon SF, Yun JT, Mcnulty Eitle T. The changing structure of school segregation: Measurement and evidence of multiracial metropolitan-area school segregation, 1989-1995. *Demography*. 2000; 37(3):351-64.

37. Krieger N. Methods for the scientific study of discrimination and health: An ecosocial approach. *Am J Public Health*. 2012; 102(5):936-44.
38. Krieger N. Theories for social epidemiology in the 21st century: An ecosocial perspective. *Int J Epidemiol*. 2001; 30(4):668-77.
39. Williams DR, Collins C. Racial residential segregation: A fundamental cause of racial disparities in health. *Public Health Rep*. 2001; 116(5):404-16.
40. Smrekar CE, Goldring EB, eds. *From the Courtroom to the Classroom: The Shifting Landscape of School Desegregation*. Harvard Education Press: Cambridge, MA; 2009.
41. Hanusgej EA, Kain JF, Rivkin SG. New evidence about *Brown v. Board of Education*: The complex effects of school racial composition on achievement. *J Labor Economics*. 2009; 27(3):349-83.
42. Lee H. The effects of school racial and ethnic composition on academic achievement during adolescence. *J Negro Education*. 2007; 76(2):154-72.
43. Goldsmith PA. All segregation is not equal: The impact of Latino and Black school composition. *Sociological Perspectives*. 2003; 46(1):83-105.
44. Roscigno VJ. Race and the reproduction of educational disadvantage. *Social Forces*. 1998; 76(3):1033-61.
45. Bankston C III, Caldas SJ. Majority African American schools and social injustice: The influence of de facto segregation on academic achievement. *Social Forces*, 1996
46. Braddock JH II, Dawkins MP. Long-term effects of school desegregation on southern Blacks. *Sociological Spectrum*. 1984; 4(4):365-81.
47. Kwate NO, Loh JM. Separate and unequal: The influence of neighborhood and school characteristics on spatial proximity between fast foods and schools. *Prev Med*. 2010; 51(2):153-6.
48. Wells AS, Crain RL. Perpetuation theory and the long-term effects of school desegregation. *Review of Educational Research*. 1994; 64(4):531-55.
49. O'Malley PM, Johnston LD, Delva J, Bachman JG, Schulenberg JE. Variation in obesity among American secondary school students by school and school characteristics. *Am J Prev Med*. 2007; 33(4 Suppl):S187-94.
50. Harris KM. *The Add Health Study: Design and Accomplishments*. Carolina Population Center, University of North Carolina at Chapel Hill, 2013. Available at http://www.cpc.unc.edu/projects/addhealth/data/guides/DesignPaperWIIV.pdf .
51. Division of Nutrition, Physical Activity, and Obesity, National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention. *About Child and Teen BMI*. https://www.cdc.gov/healthyweight.assessing/bmi/childrens_bmi/about_childrens_bmi.html. Accessed on May 12, 2017.
52. Cunningham SA, Kramer MR, Narayan KM. Incidence of childhood obesity in the United States. *N Engl J Med*. 2014; 370(5):403-11.
53. Bazaco MC, Pereira MA, Wisniewski SR, Zgibor JC, Songer TJ, Burke JD, et al. Is there a relationship between perceived neighborhood contentedness and physical activity in young men and women? *J Urban Health*. 2016; 93(6):940-52.

54. McCabe J. Racial and gender microaggressions on a predominantly-white campus: Experiences of black, Latina/o and white undergraduates. *Race, Gender & Class*. 2009; 16(1/2):133-51.
55. Juvonen J, Nishina A, Graham S. Ethnic diversity and perceptions of safety in urban middle schools. *Psychological Science*. 2006; 17(5):393-400.
56. Feagin JR, Vera H, Imani N. *The Agony of Education: Black Students at a White University* (2nd ed.). Routledge: New York, NY; 1996.
57. Allen WR. The color of success: African-American college student outcomes at predominantly white and historically black public colleges and universities. *Harvard Educational Review*. 1992; 62(1):26-44.
58. Goosby BJ, Walsemann KM. School racial composition and race/ethnic differences in early adulthood health. *Health Place*. 2012; 18(2):296-304.
59. McEwen BS, Seeman T. Protective and damaging effects of mediators of stress: Elaborating and testing the concepts of allostasis and allostatic load. *Ann N Y Acad Sci*. 1999; 896:30-47.
60. McEwen BS. Protective and damaging effects of stress mediators. *N Engl J Med*. 1998; 338(3):171-9.
61. Pervanidou P, Chrousos GP. Stress and obesity/metabolic syndrome in childhood and adolescence. *Int J Pediatr Obes*. 2011; 6 Suppl 1:21-8.
62. De Vriendt T, Moreno LA, De Henauw S. Chronic stress and obesity in adolescents: Scientific evidence and methodological issues for epidemiological research. *Nutr Metab Cardiovasc Dis*. 2009; 19(7):511-9.
63. Hussey J, Nguyen QC, Whitsel EA, Richarson LJ, Halpern CT, Gordon-Larsen P, et al. The reliability of in-home measures of height and weight in large cohort studies: Evidence from Add Health. *Demographic Research*. 2015; 32:1081-98

SPECIFIC AIM 1: APPENDIX

A1.1. Unadjusted Prevalence Ratios for Adolescent Obesity by Proportion Black Students within a School and Individual Race/Ethnicity.

Proportion Black Students	All Adolescents	Hispanic	Non-Hispanic White	Non-Hispanic Black	Asian/Native American/ Other
0.00 – 0.10	1.00	1.00	1.00	1.00	1.00
0.11 – 0.25	1.07 (0.92, 1.25)	1.18 (0.98, 1.42)	0.98 (0.82, 1.16)	1.18 (0.82, 1.69)	0.91 (0.42, 2.00)
0.26 – 0.50	1.15 (0.98, 1.34)	1.19 (0.90, 1.58)	1.11 (0.90, 1.37)	1.14 (0.84, 1.55)	1.21 (0.59, 2.52)
0.51 – 0.75	1.30 (0.96, 1.77)	0.58 (0.28, 1.17)	1.36 (0.88, 2.10)	1.40 (0.98, 2.01)	1.07 (0.45, 2.52)
0.76 – 1.00	1.43 (1.26, 1.62)	1.33 (0.42, 4.21)	1.36 (0.76, 2.44)	1.37 (1.03, 1.81)	2.07 (0.60, 7.22)

A1.2. Prevalence Ratios for Adolescent Obesity by Proportion Black Students within a School and Individual Race/Ethnicity, Adjusted for School-Level Covariates.

Proportion Black Students	All Adolescents	Hispanic	Non-Hispanic White	Non-Hispanic Black	Asian/Native American/Other
0.00 – 0.10	1.00	1.00	1.00	1.00	1.00
0.11 – 0.25	1.20 (1.05, 1.38)	1.22 (1.01, 1.46)	1.18 (0.97, 1.43)	1.11 (0.79, 1.57)	1.38 (0.87, 2.18)
0.26 – 0.50	1.20 (1.05, 1.37)	1.23 (0.94, 1.61)	1.19 (1.00, 1.40)	1.10 (0.84, 1.43)	2.17 (1.23, 3.83)
0.51 – 0.75	1.34 (1.02, 1.77)	0.56 (0.27, 1.17)	1.49 (1.02, 2.17)	1.30 (0.98, 1.70)	1.67 (0.96, 2.91)
0.76 – 1.00	1.35 (1.14, 1.61)	1.23 (0.38, 3.98)	1.26 (0.70, 2.28)	1.18 (0.93, 1.50)	2.47 (0.82, 7.44)
School Size					
1 – 400 students	1.36 (1.15, 1.61)	1.26 (0.93, 1.69)	1.46 (1.21, 1.77)	0.93 (0.77, 1.12)	3.09 (1.51, 6.36)
401 – 1,000 students	1.08 (0.96, 1.20)	1.07 (0.87, 1.32)	1.04 (0.88, 1.21)	1.26 (1.08, 1.47)	0.88 (0.50, 1.53)
1,001 – 4,000 students	1.00	1.00	1.00	1.00	1.00
School Urbanicity					
Urban	0.90 (0.76, 1.07)	1.39 (0.81, 2.40)	0.85 (0.67, 1.08)	0.80 (0.63, 1.00)	0.40 (0.15, 1.09)
Suburban	0.98 (0.86, 1.11)	1.26 (0.73, 2.18)	1.03 (0.88, 1.22)	0.91 (0.80, 1.04)	0.71 (0.28, 1.78)
Rural	1.00	1.00	1.00	1.00	1.00
School Type					
Public	1.41 (1.10, 1.82)	1.46 (0.91, 2.32)	1.20 (0.96, 1.74)	1.26 (0.88, 1.81)	2.37 (1.01, 5.55)
Private	1.00	1.00	1.00	1.00	1.00

A1.3. Prevalence Ratios for Adolescent Obesity by Proportion Black Students within a School and Individual Race/Ethnicity, Adjusted for School- and Neighborhood-Level Covariates.

Proportion Black Students	All Adolescents	Hispanic	Non-Hispanic White	Non-Hispanic Black	Asian/Native American/ Other
0.00 – 0.10	1.00	1.00	1.00	1.00	1.00
0.11 – 0.25	1.14 (1.00, 1.30)	1.23 (1.02, 1.48)	1.12 (0.93, 1.35)	1.06 (0.76, 1.48)	1.24 (0.78, 1.96)
0.26 – 0.50	1.08 (0.92, 1.26)	1.23 (0.93, 1.61)	1.07 (0.87, 1.33)	1.03 (0.79, 1.33)	1.80 (0.97, 3.36)
0.51 – 0.75	1.18 (0.98, 1.44)	0.60 (0.29, 1.22)	1.37 (0.99, 1.89)	1.20 (0.90, 1.59)	1.04 (0.43, 2.54)
0.76 – 1.00	1.12 (0.88, 1.42)	1.38 (0.45, 4.27)	1.15 (0.61, 2.19)	1.09 (0.83, 1.43)	1.40 (0.29, 6.83)
School Size					
1 – 400 students	1.23 (1.05, 1.44)	1.25 (0.91, 1.73)	1.26 (1.06, 1.51)	0.90 (0.75, 1.07)	2.57 (1.23, 5.35)
401 – 1,000 students	1.05 (0.94, 1.18)	1.09 (0.89, 1.34)	0.98 (0.83, 1.15)	1.27 (1.09, 1.47)	0.79 (0.44, 1.40)
1,001 – 4,000 students	1.00	1.00	1.00	1.00	1.00
School Urbanicity					
Urban	0.90 (0.76, 1.07)	1.58 (0.93, 2.69)	0.91 (0.71, 1.16)	0.78 (0.63, 0.98)	0.39 (0.14, 1.07)
Suburban	1.03 (0.90, 1.18)	1.55 (0.90, 2.65)	1.13 (0.95, 1.33)	0.90 (0.80, 1.03)	0.82 (0.32, 2.09)
Rural	1.00	1.00	1.00	1.00	1.00
School Type					
Public	1.31 (1.02, 1.67)	1.34 (0.85, 2.14)	1.18 (0.88, 1.57)	1.21 (0.83, 1.76)	2.23 (0.88, 5.67)
Private	1.00	1.00	1.00	1.00	1.00
Neighborhood Characteristics					
Poverty in 1990	4.68 (2.45, 8.93)	2.82 (0.55, 14.44)	8.08 (2.92, 22.34)	2.13 (0.97, 4.68)	6.54 (0.23, 187.60)
Proportion Black in 1990	1.03 (0.71, 1.48)	0.55 (0.23, 1.36)	0.84 (0.42, 1.70)	1.06 (0.70, 1.61)	3.94 (0.41, 38.03)

A1.4. Unadjusted Prevalence Ratios for Adolescent Obesity by Proportion Non-White Students within a School and Individual Race/Ethnicity.

Proportion Non-White Students	All Adolescents	Hispanic	Non-Hispanic White	Non-Hispanic Black	Asian/Native American/ Other
0.00 – 0.10	1.00	1.00	1.00	1.00	1.00
0.11 – 0.25	0.88 (0.75, 1.04)	1.09 (0.74, 1.60)	0.83 (0.71, 0.97)	1.88 (1.11, 3.16)	0.25 (0.06, 1.08)
0.26 – 0.50	1.05 (0.86, 1.28)	0.92 (0.57, 1.49)	1.05 (0.83, 1.33)	1.51 (0.90, 2.52)	0.90 (0.23, 3.50)
0.51 – 0.75	1.16 (0.96, 1.39)	1.11 (0.83, 1.49)	1.11 (0.87, 1.40)	1.78 (1.04, 3.03)	0.87 (0.27, 2.82)
0.76 – 1.00	1.29 (1.11, 1.50)	1.19 (0.90, 1.56)	1.14 (0.82, 1.59)	1.75 (1.06, 2.91)	1.52 (0.45, 5.13)

A1.5. Prevalence Ratios for Adolescent Obesity by Proportion Non-White Students within a School and Individual Race/Ethnicity, Adjusted for School-Level Covariates.

Proportion Non-White Students	All Adolescents	Hispanic	Non-Hispanic White	Non-Hispanic Black	Asian/Native American/ Other
0.00 – 0.10	1.00	1.00	1.00	1.00	1.00
0.11 – 0.25	1.02 (0.88, 1.17)	1.15 (0.77, 1.72)	0.98 (0.84, 1.15)	1.77 (1.10, 2.81)	0.36 (0.08, 1.62)
0.26 – 0.50	1.11 (0.93, 1.31)	0.88 (0.57, 1.34)	1.12 (0.93, 1.35)	1.40 (0.91, 2.14)	1.13 (0.37, 3.42)
0.51 – 0.75	1.32 (1.11, 1.56)	1.12 (0.81, 1.57)	1.29 (1.03, 1.62)	1.73 (1.13, 2.65)	1.25 (0.51, 3.03)
0.76 – 1.00	1.37 (1.19, 1.57)	1.21 (0.89, 1.64)	1.24 (0.89, 1.72)	1.59 (1.05, 2.40)	1.78 (0.71, 4.44)
School Size					
1 – 400 students	1.38 (1.17, 1.63)	1.27 (0.90, 1.80)	1.47 (1.21, 1.77)	0.98 (0.80, 1.19)	3.06 (1.28, 7.29)
401 – 1,000 students	1.12 (1.00, 1.25)	1.07 (0.84, 1.36)	1.05 (0.90, 1.23)	1.31 (1.12, 1.54)	1.39 (0.74, 2.61)
1,001 – 4,000 students	1.00	1.00	1.00	1.00	1.00
School Urbanicity					
Urban	0.84 (0.71, 1.01)	1.45 (0.84, 2.49)	0.84 (0.65, 1.09)	0.80 (0.66, 0.99)	0.45 (0.16, 1.24)
Suburban	0.95 (0.84, 1.08)	1.43 (0.82, 2.50)	1.01 (0.86, 1.18)	0.96 (0.83, 1.11)	0.65 (0.24, 1.73)
Rural	1.00	1.00	1.00	1.00	1.00
School Type					
Public	1.38 (1.07, 1.78)	1.33 (0.81, 2.18)	1.29 (0.95, 1.75)	1.41 (1.04, 1.92)	1.91 (0.82, 4.41)
Private	1.00	1.00	1.00	1.00	1.00

A1.6. Prevalence Ratios for Adolescent Obesity by Proportion Non-White Students within a School and Individual Race/Ethnicity, Adjusted for School- and Neighborhood-Level Covariates.

Proportion Non-White Students	All Adolescents	Hispanic	Non-Hispanic White	Non-Hispanic Black	Asian/Native American/Other
0.00 – 0.10	1.00	1.00	1.00	1.00	1.00
0.11 – 0.25	0.98 (0.86, 1.13)	1.12 (0.76, 1.66)	0.95 (0.81, 1.12)	1.66 (1.05, 2.63)	0.43 (0.10, 1.78)
0.26 – 0.50	1.00 (0.83, 1.21)	0.84 (0.52, 1.34)	1.02 (0.81, 1.29)	1.30 (0.86, 1.98)	1.03 (0.37, 2.88)
0.51 – 0.75	1.17 (0.99, 1.39)	1.04 (0.71, 1.52)	1.19 (0.94, 1.50)	1.59 (1.04, 2.42)	0.99 (0.38, 2.53)
0.76 – 1.00	1.13 (0.92, 1.39)	1.06 (0.70, 1.60)	1.13 (0.77, 1.66)	1.43 (0.94, 2.20)	1.06 (0.30, 3.77)
School Size					
1 – 400 students	1.24 (1.06, 1.45)	1.23 (0.85, 1.79)	1.25 (1.05, 1.48)	0.94 (0.78, 1.13)	2.60 (1.12, 6.05)
401 – 1,000 students	1.07 (0.95, 1.19)	1.08 (0.86, 1.37)	0.99 (0.84, 1.16)	1.30 (1.10, 1.52)	1.17 (0.60, 2.29)
1,001 – 4,000 students	1.00	1.00	1.00	1.00	1.00
School Urbanicity					
Urban	0.87 (0.73, 1.03)	1.55 (0.91, 2.65)	0.91 (0.71, 1.18)	0.79 (0.64, 0.97)	0.46 (0.16, 1.31)
Suburban	1.01 (0.89, 1.15)	1.59 (0.89, 2.83)	1.11 (0.95, 1.31)	0.94 (0.82, 1.09)	0.74 (0.25, 2.17)
Rural	1.00	1.00	1.00	1.00	1.00
School Type					
Public	1.29 (1.01, 1.67)	1.40 (0.82, 2.37)	1.17 (0.88, 1.56)	1.33 (0.97, 1.83)	2.03 (0.74, 5.55)
Private	1.00	1.00	1.00	1.00	1.00
Neighborhood Characteristics					
Poverty in 1990	4.31 (2.24, 8.32)	1.76 (0.28, 10.95)	8.98 (3.24, 24.87)	2.03 (1.01, 4.10)	6.39 (0.28, 148.35)
Proportion Non-White in 1990	1.10 (0.74, 1.63)	1.33 (0.49, 3.63)	0.84 (0.43, 1.63)	1.12 (0.76, 1.65)	3.59 (0.26, 49.94)

A1.7. Unadjusted Prevalence Ratios for Adolescent Obesity by School Entropy (Multi-Group, Standardized) and Individual Race/Ethnicity.

School Entropy	All Adolescents	Hispanic	Non-Hispanic White	Non-Hispanic Black	Asian/Native American/ Other
0.02 – 0.10	1.00	1.00	1.00	1.00	1.00
0.11 – 0.25	1.01 (0.81, 1.25)	1.25 (0.81, 1.93)	0.90 (0.67, 1.19)	1.22 (0.91, 1.64)	0.45 (0.07, 3.04)
0.26 – 0.50	0.99 (0.85, 1.15)	1.35 (0.85, 2.14)	0.94 (0.80, 1.11)	1.07 (0.80, 1.43)	0.71 (0.16, 3.12)
0.51 – 0.75	1.02 (0.84, 1.23)	1.38 (0.93, 2.06)	0.89 (0.72, 1.11)	1.08 (0.75, 1.57)	0.70 (0.14, 3.61)
0.76 – 0.94	1.10 (0.89, 1.37)	1.46 (0.96, 2.23)	1.24 (0.95, 1.61)	0.85 (0.61, 1.18)	0.78 (0.18, 3.35)

A1.8. Prevalence Ratios for Adolescent Obesity by School Entropy (Multi-Group, Standardized) and Individual Race/Ethnicity, Adjusted for School-Level Covariates.

School Entropy	All Adolescents	Hispanic	Non-Hispanic White	Non-Hispanic Black	Asian/Native American/ Other
0.02 – 0.10	1.00	1.00	1.00	1.00	1.00
0.11 – 0.25	1.05 (0.87, 1.28)	1.26 (0.71, 2.24)	0.90 (0.72, 1.13)	1.08 (0.89, 1.31)	0.51 (0.10, 2.51)
0.26 – 0.50	1.07 (0.94, 1.23)	1.33 (0.76, 2.33)	1.03 (0.89, 1.18)	1.05 (0.87, 1.27)	0.92 (0.33, 2.58)
0.51 – 0.75	1.21 (0.99, 1.47)	1.37 (0.80, 2.34)	1.10 (0.87, 1.38)	1.13 (0.88, 1.45)	0.82 (0.29, 2.28)
0.76 – 0.94	1.32 (1.09, 1.61)	1.53 (0.90, 2.58)	1.41 (1.12, 1.79)	1.05 (0.73, 1.50)	1.12 (0.37, 3.40)
School Size					
1 – 400 students	1.44 (1.23, 1.69)	1.25 (0.90, 1.73)	1.49 (1.22, 1.81)	1.04 (0.81, 1.33)	2.91 (1.26, 6.73)
401 – 1,000 students	1.14 (1.00, 1.30)	1.04 (0.82, 1.34)	1.06 (0.90, 1.23)	1.35 (1.12, 1.62)	1.21 (0.66, 2.21)
1,001 – 4,000 students	1.00	1.00	1.00	1.00	1.00
School Urbanicity					
Urban	0.88 (0.73, 1.08)	1.39 (0.81, 2.36)	0.89 (0.69, 1.14)	0.81 (0.65, 1.01)	0.45 (0.18, 1.16)
Suburban	0.95 (0.82, 1.10)	1.26 (0.72, 2.20)	1.04 (0.89, 1.21)	0.94 (0.81, 1.08)	0.64 (0.25, 1.65)
Rural	1.00	1.00	1.00	1.00	1.00
School Type					
Public	1.54 (1.16, 2.03)	1.27 (0.74, 2.17)	1.41 (1.02, 1.95)	1.42 (0.96, 2.10)	2.86 (1.15, 7.10)
Private	1.00	1.00	1.00	1.00	1.00

A1.9. Prevalence Ratios for Adolescent Obesity by School Entropy (Multi-Group, Standardized) and Individual Race/Ethnicity, Adjusted for School- and Neighborhood-Level Covariates.

School Entropy	All Adolescents	Hispanic	Non-Hispanic White	Non-Hispanic Black	Asian/Native American/ Other
0.00 – 0.10	1.00	1.00	1.00	1.00	1.00
0.11 – 0.25	0.96 (0.81, 1.15)	1.26 (0.69, 2.27)	0.85 (0.70, 1.05)	1.05 (0.86, 1.28)	0.58 (0.11, 3.06)
0.26 – 0.50	0.99 (0.86, 1.14)	1.30 (0.74, 2.29)	0.94 (0.81, 1.10)	1.08 (0.89, 1.31)	0.78 (0.28, 2.19)
0.51 – 0.75	1.08 (0.91, 1.28)	1.30 (0.75, 2.26)	1.02 (0.82, 1.27)	1.16 (0.93, 1.46)	0.72 (0.25, 2.04)
0.76 – 1.00	1.18 (0.96, 1.46)	1.43 (0.82, 2.51)	1.27 (1.01, 1.60)	1.06 (0.75, 1.48)	0.93 (0.32, 2.70)
School Size					
1 – 400 students	1.26 (1.08, 1.47)	1.22 (0.88, 1.70)	1.25 (1.04, 1.50)	0.97 (0.76, 1.25)	2.72 (1.30, 5.66)
401 – 1,000 students	1.08 (0.96, 1.21)	1.07 (0.84, 1.36)	0.98 (0.84, 1.14)	1.31 (1.10, 1.55)	1.28 (0.69, 2.39)
1,001 – 4,000 students	1.00	1.00	1.00	1.00	1.00
School Urbanicity					
Urban	0.88 (0.73, 1.06)	1.51 (0.89, 2.55)	0.96 (0.75, 1.23)	0.76 (0.61, 0.95)	0.52 (0.22, 1.23)
Suburban	1.01 (0.88, 1.16)	1.48 (0.84, 2.61)	1.16 (0.99, 1.36)	0.89 (0.78, 1.03)	0.81 (0.37, 1.79)
Rural	1.00	1.00	1.00	1.00	1.00
School Type					
Public	1.37 (1.05, 1.79)	1.27 (0.74, 2.18)	1.36 (0.94, 1.69)	1.32 (0.88, 1.96)	2.76 (0.98, 7.72)
Private	1.00	1.00	1.00	1.00	1.00
Neighborhood Characteristics					
Poverty in 1990	4.66 (2.43, 8.91)	2.04 (0.29, 14.59)	9.61 (3.57, 25.88)	1.84 (0.78, 4.34)	7.59 (0.41, 139.32)
Proportion Non-White in 1990	1.23 (0.89, 1.69)	1.19 (0.47, 2.99)	0.94 (0.56, 1.59)	1.30 (0.88, 1.93)	4.65 (0.95, 22.86)

Not So Black and White: The Racial/Ethnic Structure of School Environments and Cardiometabolic and Reproductive Health across the Life Course

Chapter 3: Inflammation

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SPECIFIC AIM 2

Investigate the multilevel associations between the racial/ethnic structure of school environments experienced in adolescence and inflammation in early adulthood.

Hypothesis 2a: Adolescents who attend schools with a higher proportion of black students, higher proportion of non-white students, or with less racial/ethnic diversity will have higher levels of C-reactive protein (CRP), after adjusting for individual-, school-, and neighborhood-level characteristics. These associations will differ by an adolescent's race/ethnicity.

Hypothesis 2b: Adolescents who attend schools within districts that have higher levels of racial/ethnic segregation will have higher levels of CRP, after adjusting for individual-, school-, and neighborhood-level characteristics. These associations will differ by an adolescent's race/ethnicity.

ABSTRACT

Given the critical biological and social development that occurs during adolescence and the life course implications of harmful exposures that occur during this critical period, it is necessary to investigate the health consequences of racial/ethnic structure of their school environments. This study will examine the racial/ethnic differences in the associations between school racial/ethnic composition, diversity, and district-level segregation and inflammation in early adulthood. We used data from Waves I (1994-5) and IV (2008) of the National Longitudinal Adolescent to Adult Health Study (N=15,701). Inflammation was classified into the following categories of high-sensitivity C-reactive protein (hsCRP) concentrations: (1) *low/average*: ≤ 3 mg/L, and (2) *high*: 3-10 mg/L. The predicted marginal prevalence of inflammation, by school racial/ethnic composition (i.e., proportion Black and non-White students), school racial/ethnic diversity (i.e., school entropy), and district-level segregation (i.e., Black-White dissimilarity and exposure indices) were estimated from cluster- and sample-weight adjusted logistic regression models in SUDAAN. Models stratified by race/ethnicity were adjusted for school-level (size, type, urbanicity), neighborhood-level (poverty, racial composition), and individual-level (age, gender, parent's education, physical activity) characteristics. There were no meaningful associations between the various measures of the racial/ethnic structure of school environments and inflammation in early adulthood, even across the different racial/ethnic groups.

SIGNIFICANCE

Though there have been considerable declines in mortality due to advances in medical treatments and public health interventions[1], cardiovascular disease is still the leading cause of death in the United States (U.S.)[2]. Furthermore, having higher levels of inflammatory biomarkers are associated with a higher cardiometabolic risk[3-9]. C-reactive protein (CRP) is one biomarker of systemic inflammation that can bind to low density lipoprotein and deposit in the walls of the arteries creating a buildup of plaque, thus playing a direct role in the development of atherosclerosis[10-14]. Though the national prevalence of inflammation has decreased in recent years[7,15], there still remains a large burden among young adults. In fact, among adults ages 20-29 and 30-39 years old, the prevalence of elevated CRP levels (> 3.0 mg/L) is 26.7% and 30.2%, respectively, in the U.S.[16].

Early-life social factors, particularly those experienced in childhood, can contribute to increased cardiometabolic risk in adulthood. Long-term external stressors, such as socio-contextual exposures, can have negative consequences for one's sympathetic nervous system and hypothalamic-pituitary-adrenal axis. Repeated stress on the body may lead to biological "wear and tear" (i.e., allostatic load) and hyperactivity of adaptive responses, resulting in increases in cortisol levels and the disruption of other inflammatory processes. Constant wear and tear on the body, particularly from socioeconomic stressors, can accumulate across one's life course and produce harmful cardiometabolic outcomes during adulthood[17-20].

Racial/ethnic and socioeconomic disparities in inflammation also exist in the U.S. adult population. Compared to Whites and adults with a high socioeconomic status (SES), Blacks and those with a lower SES are more likely to have elevated levels of

inflammatory biomarkers[16,21-24]. Adults with lower educational attainment are also more likely to have higher levels of CRP[25]. Given these persistent disparities, it is important to investigate the socio-contextual risk factors for inflammation that occur during childhood or adolescence. In fact, in a previous study among Add Health participants, Black students who attended predominantly white schools were likely to have worse health outcomes in early adulthood[26]. Thus, the racial/ethnic structure of school environments experienced during adolescence could be associated with cardiometabolic outcomes occurring in adulthood.

In order to fully understand the relationship between the racial/ethnic composition of schools and systemic inflammation in early adulthood, one must consider the historical context and evolution of school segregation. Public schools in the U.S. were segregated based on race due to the legal guidelines set out by *Plessy v. Ferguson* (1896). This Supreme Court decision resulted in unequal educational opportunities for black students in this country[27-29]. *Brown v. Board of Education of Topeka* (1954) dismantled the "separate, but equal" doctrine from *Plessy* and southern U.S. schools were under mandates to desegregate[30]. However, over the coming years, residential segregation persisted and other Supreme Court decisions relinquished school systems from their court-ordered mandates[31-34], ultimately resulting in the resegregation of U.S. public schools beginning in the late 1970s[29,35-37]. Given the evolution from segregation to desegregation, and back to resegregation, the racial/ethnic structure of schools is a relevant and plausible, social determinant of cardiometabolic risk in early adulthood.

There is an established relationship between individual- and neighborhood-level socioeconomic factors during childhood and adolescence and inflammation in

adulthood. SES is a fundamental cause of health and health inequalities through its important link to critical health-relevant resources, as well as its relationship to multiple diseases[38]. Having a lower SES in childhood or adolescence is associated with increased levels of CRP[39-41] and an increased risk of type 2 diabetes and obesity[42] in adulthood. Additionally, residing in a neighborhood that is more socioeconomically disadvantaged in adolescence is associated with an increased risk of metabolic syndrome in early adulthood[43]. However, to our knowledge, there are no recent studies that have examined the relationship between both the absolute racial/ethnic composition of schools, as well as district-level school segregation, and cardiometabolic risk in early adulthood. In this study, we will investigate the associations between school racial/ethnic composition, school diversity, and district-level school segregation experienced during adolescence and inflammation in early adulthood. Given the historical context of school segregation in the U.S. and the persistent racial/ethnic disparities, we will also examine whether these longitudinal associations differ by race/ethnicity.

METHODS

Study Design and Population

For this study, we used data from Waves I (1994-1995) and Wave IV (2008) of the National Longitudinal Study of Adolescent to Adult Health (Add Health). Beginning in 1994, Add Health is an ongoing, longitudinal study that follows adolescents in grades 7-12 into adulthood. This study utilized a complex sampling design and stratification method in order to derive a nationally representative sample of U.S. high schools in Wave I (1994-1995). Using a sampling frame based on a database from the Quality Education Data, Inc. (QED), Add Health consisted of a school-based sample of adolescents living in the U.S. Systematic sampling methods and implicit stratification were used to ensure that the schools in Add Health were representative of all U.S. schools regarding the following factors: region, urbanicity, size, type, and racial composition[44].

The high schools incorporated into the sample had to include eleventh graders and at least thirty enrolled students. Seventy-nine percent of sampled schools participated in the study, resulting in eighty high schools represented in the Add Health sample. Once high schools were sampled, feeder (i.e., middle) schools were identified to capture students in the seventh and eighth grades from the 80 communities in which the high schools were located. These feeder schools were required to include the seventh grade and had to send at least five graduates to one of the eighty high schools in the Add Health sample. Altogether, there were 132 U.S. schools included in the Add Health Wave I sample: eighty high schools and fifty-two feeder schools. There was not a one to one ratio of high schools to feeder schools because some high schools included

students from grades seven through twelve, and thus, separate feeder schools were not recruited for these high schools[44].

Given that this was a school-based sample, both in-school questionnaires and in-home interviews were administered during Wave I. From the sampled schools, there were 90,118 students in grades seven through twelve who participated in the in-school questionnaires during Wave I. Of these students, 20,745 adolescents were randomly selected, based on grade and sex, to be in the "core sample" to participate in the in-home interviews. Only those students who participated in the in-school questionnaires were eligible to be selected to participate in the in-home interviews. In addition to the random selection by grade and sex, investigators oversampled based on the following criteria: ethnicity, disability status, school saturation, and genetic factors (e.g., twins)[44].

Conducted in 2008, Wave IV only consisted of in-home interviews, as well as the collection of geographic and biological data, for the respondents who were part of the "core sample" from Wave I of Add Health. Researchers were able to locate 92.5% of the original Wave I respondents, and of those located, 80.3% were sampled. Wave IV included 15,701 young adults, aged twenty-four through thirty-four years old. In-home interviews were conducted using 90-minute computer-assisted personal interviews and computer-assisted self-interviews. Upon completion of these interviews, biological and physical measurements were collected[44].

Inflammation

Since higher levels of inflammatory biomarkers are associated with a higher cardiometabolic risk[3-9], inflammation in early adulthood was used as the primary outcome for this study. Inflammation was assessed by the concentration of high-sensitivity C-reactive protein (hsCRP) measured during Wave IV of Add Health. Dried capillary whole blood spots were collected during the Wave IV in-home interviews, and then assayed at the University of Washington Medical Center Immunology Lab, in Seattle, Washington[45]. In accordance to previously established criteria, inflammation was classified into the following categories of hsCRP concentration: (1) *low/average*: ≤ 3 mg/L, and (2) *high*: 3-10 mg/L. We excluded 1,786 participants who had hsCRP concentrations > 10 mg/L because these high levels are often a sign of acute inflammation and/or infection, and may not be related to cardiometabolic risk[46].

Individual Race/Ethnicity

Adolescents reported their race/ethnicity during the in-home interview of Wave I. Individual race/ethnicity consisted of the following categories: Hispanic, Non-Hispanic White, Non-Hispanic Black, Asian/Pacific Islander, American Indian/Alaska Native, and Other. We restricted our analyses to include Hispanic, non-Hispanic White, and non-Hispanic Black participants.

Racial/Ethnic Structure of Schools and School Systems

We evaluated two types of exposures in this study: (1) the racial/ethnic composition of the schools that the participants attended in adolescence, and (2) the average racial/ethnic segregation within the district in which their schools resided. The

measures of composition were utilized to describe the racial/ethnic distribution and levels of diversity within the Add Health schools. The measures of district-level segregation aimed to quantify the degree to which racial/ethnic groups of students are segregated within school districts in Add Health. Thus, we utilized these two types of exposures in order to investigate the health consequences of the absolute racial/ethnic make-up of a school, as well as the spatial separation of racial/ethnic groups within a school district.

School racial/ethnic composition was defined in three ways: (1) the proportion of the total student population within each Wave I Add Health school that was non-Hispanic Black; (2) the proportion of the total student population within each school that was non-White; and (3) the racial/ethnic entropy (i.e., diversity) within each school. School entropy, a measure of racial/ethnic diversity within a school, was calculated using the following formula:

$$E_i = \sum_{r_i=1}^{n_i} Q_{r_i} \ln(\frac{1}{Q_{r_i}})$$

n_i represents the total number of racial/ethnic groups in the ith school. r_i represents the specific racial/ethnic group in the ith school. Q_{r_i} represents the total students in the ith school made up of the specific racial/ethnic. School entropy is an index that theoretically ranges from 0 (minimum diversity) to 1 (maximum diversity), after standardization. For this multi-group measure of entropy, a value of 0 was assigned to schools where a single racial/ethnic group was present. A value of 1 was assigned to schools where all racial/ethnic groups were represented equally in the student population[35].

The average racial/ethnic segregation within a school district was evaluated using two indices: (1) the Black-White dissimilarity index, and (2) the Black-White Exposure Index. Both indices assess the extent to which Black students are segregated from White students within a school district. The Black-White dissimilarity represents the proportion of Black students that would have to change their schools in order to achieve an even racial distribution (among Black and White students) in their respective school district. The Black-White dissimilarity index was calculated using the following formula:

$$\frac{1}{2}\sum_{i=1}^{N}(\frac{b_i}{B} - \frac{w_i}{W})$$

b_i is the number of Black students in the ith school. B represents the total Black student enrollment in the school district. w_i is the number of White students in the ith school. W represents the total White student enrollment in the school district.

The Black-White Exposure index represents the extent to which Black students are exposed to White students within their school district. This exposure index was calculated using the following formula:

$$\sum_{i=1}^{N}(\frac{w_i}{W} * \frac{b_i}{t_i})$$

t_i is the total student enrollment within the school (across all racial/ethnic groups). Ranging from 0 (lowest probability of exposure) to 1 (highest probability of exposure), both school segregation indices were previously derived and calculated by Add Health researchers, using the adolescents' Wave I school-level data and data from the Common Core of Data, National Center for Education Statistics[47].

Covariates: School

School-level covariates included: size, urbanicity, and type. Schools were divided into three categories, based on the definition of school size from the National Center for Education Statistics (NCES). These categories included: (1) *small*: 1-400 students, (2) *medium*: 401 – 1,000 students, and (3) *large*: 1,001 – 4,000 students. A school's urbanicity was determined according to the NCES and QED classification of the geographic areas in which the schools reside. This classification included:

(a) Central city of a Consolidated Metropolitan Statistical Area (CMSA) or Metropolitan Statistical Area (MSA) with population of 250,000 or more
(b) Central city of a CMSA or MSA, but not designated as a large central city
(c) Place within the CMSA or MSA of a large central city
(d) Place within the CMSA or MSA of a mid-size central city
(e) Place not within a CMSA or MSA, but with population of 25,000 or more and defined as urban
(f) Place not within a CMSA or MSA with a population of at least 2,500, but less than 25,000
(g) Place not within a CMSA or MSA and designated as rural
(h) Place within a CMSA or MSA designated as rural

In this study, schools were categorized as: (1) *urban*: included schools located within items *(a)* and *(b)*; (2) *suburban*: included schools located within items *(c) – (f)*; and (3) *rural*: included schools located within items *(g)* and *(h)*. Regarding type, schools were labeled as either public or private.

Covariates: Neighborhood

At Wave III (2001-2002), neighborhood-level data was merged retrospectively using area of residence during Wave I. This neighborhood data was derived from the 1990 U.S. Census[44]. Neighborhood-level covariates included: poverty and racial composition. For neighborhood poverty, we used the proportion of residents in each adolescents' census tract who were living below the federal poverty level. The selection of the neighborhood racial composition was dependent upon the exposure of interest.

For the analyses that investigated the proportion of Black students within a school and the school segregation indices, we used the proportion of residents who were non-Hispanic Black. For the analyses that investigated the proportion of non-White students within a school and school entropy, we used the proportion of residents who were non-White.

Covariates: Individual

Individual-level covariates included: age, gender, parental education, and physical activity. An adolescent's age (10-13, 14-15, and 16-19 years) and gender (male vs. female) was self-reported during the in-home interview of Wave I. The educational attainment levels of an adolescent's parents were self-reported by the parents during the in-home interview. In our study, parental education was determined to be the highest educational attainment of either the mother or father. Parental education was collapsed into the following categories: less than high school, high school diploma, some college or an Associate's degree, and college degree or higher.

An adolescent's physical activity levels were defined using a previous categorization based on Add Health data. Physical activity was determined from how many times per week (during Wave I) the participant engaged in the following three groups of activities: (a) rollerblading, skating, skate-boarding, and bicycling; (b) baseball, football, basketball, and soccer; and (c) jogging, walking, jumping rope, dancing, and karate. Adolescents' assessed their average weekly participation in these three groups of activities as (1) not at all, (2) 1 or 2 times, (3) 3 or 4 times, or (4) 5 or more times. We then assigned the following numerical values:

Not at all = 0
1 or 2 times = 1.5

3 or 4 times = 3.5
5 or more times = 5

We summed across the three groups of activities and created a physical activity score, which was categorized as: *not physically active* (< 5 times/week) vs. *physically active* (5+ times/week)[48].

Statistical Analysis

The predicted prevalence of inflammation were estimated from cluster- and sample-weight adjusted logistic regression models in SUDAAN. School racial/ethnic composition, diversity, and district-level segregation were assessed using the continuous versions of these variables. Analyses included unadjusted models, as well as models adjusted for covariates in three stages. Stage 1 included linear and logistic regression models adjusted for school-level covariates (size, type, urbanicity) only. Stage 2 included linear and logistic regression models adjusted for school-level covariates and neighborhood-level covariates (poverty, racial/ethnic composition). Stage 3 included the fully-adjusted linear and logistic regression models, which were adjusted for school-level covariates, neighborhood-level covariates, and individual-level covariates (age, gender, parental education, physical activity). All models were then stratified by individual race/ethnicity. Unadjusted models, as well as models from stages 1 & 2, are included in the Appendix.

RESULTS

Socio-Demographic Characteristics of Schools, Neighborhoods, and Participants

Table 1 displays the characteristics of the schools attended by the young adults who participated in Wave IV of Add Health. The racial/ethnic composition, diversity, and district-level segregation of the schools attended by the participants in this study population also varied by race/ethnicity. Non-Hispanic Black participants attended schools during adolescence that had a higher proportion of Black students than schools attended by Hispanic and non-Hispanic White participants. On average, Hispanic and non-Hispanic Black participants attended schools during adolescence that had the highest proportion of non-White students (58% and 67%, respectively). Compared to non-Hispanic White and non-Hispanic Black adults, Hispanic adults attended schools during adolescence with the highest average levels of racial/ethnic diversity.

Regarding the Black-White dissimilarity index, in schools attended by Hispanic and non-Hispanic Black participants, 40% and 38% of Black students, respectively, would have to change schools in order to achieve an even racial distribution in the school district. Concerning the Black-White exposure index, in schools attended by non-Hispanic White participants, Black students had a 74% probability of being exposed to a White student. In those schools attended by non-Hispanic Black participants had a 39% probability of being exposed to a White student.

The sociodemographic characteristics of the young adults in Wave IV and the neighborhoods in which they resided during Wave I of Add Health are described in Table 2. Compared to non-Hispanic White adults, Hispanic and non-Hispanic Black adults were more likely to have a lower adolescent SES, as measured by their parent's

highest educational attainment at Wave I. The proportion of Add Health participants with high levels of hsCRP (> 3 and ≤ 10 mg/L) were similar across racial/ethnic groups – with approximately one-third of the population exhibiting inflammation.

Proportion Black Students and Inflammation

Table 3 presents the adjusted prevalence ratios for inflammation by measures of school racial/ethnic composition, diversity, and district-level segregation, stratified by individual race/ethnicity. After adjusting for individual-, school-, and neighborhood-level covariates, there were null associations between the proportion of Black students in a school and the prevalence of inflammation, among Hispanic, non-Hispanic White, and non-Hispanic Black participants. Though the predicted prevalence of inflammation among Hispanic adults appeared to be higher as the proportion of Black students within a school was higher (*Figure 1a*), there was still no overall association among this racial/ethnic sub-group. Non-Hispanic White participants did not experience a change in the predicted prevalence of inflammation across the various levels of the proportion of Black students. Finally, though the predicted prevalence of inflammation among non-Hispanic Black participants appeared to be lower as the proportion of Black students within a school was higher, there was no overall association within this racial/ethnic sub-group.

Proportion Non-White Students within a School and Inflammation

After adjusting for individual-, school-, and neighborhood-level covariates, there were null associations between the proportion of non-White students in a school and the prevalence of inflammation, among Hispanic, non-Hispanic White, and non-Hispanic Black participants (*Table 3*). In Figure 1b, the predicted prevalence of inflammation

among Hispanic adults appeared to be higher as the proportion of non-White students within a school was higher. However, there is no overall association between this measure of school racial composition and inflammation among this racial/ethnic sub-group. Furthermore, there is an important finding from the Appendix that is worth noting. After adjusting for school-level characteristics, there was a significant association between the proportion of non-White students within a school and the prevalence of inflammation in early adulthood among Hispanic participants. However, this association was attenuated after adjusting for neighborhood-level characteristics.

Similar to the findings from the measures of school racial/ethnic composition, non-Hispanic White participants did not experience a change in the predicted prevalence of inflammation across the various levels of the proportion of non-White students. Finally, though the predicted prevalence of inflammation among non-Hispanic Black adults appeared to be lower as the proportion of non-White students within a school was higher, there was no overall association among this racial/ethnic sub-group.

School Entropy and Inflammation

After adjusting for individual-, school-, and neighborhood-level covariates, there were null associations between the levels of school racial/ethnic diversity and the prevalence of inflammation, among Hispanic, non-Hispanic White, and non-Hispanic Black participants (*Table 3*). In Figure 1c, the predicted prevalence of inflammation among non-Hispanic Black adults appeared to be higher as the levels of racial/ethnic diversity within a school were higher. However, there was no overall association observed among this racial/ethnic sub-group. Similar to the findings with the measures

of school racial/ethnic composition, non-Hispanic White participants did not experience a change in the predicted prevalence of inflammation across the various levels of school diversity.

Black-White Dissimilarity Index (District-Level Segregation) and Inflammation

After adjusting for individual-, school-, and neighborhood-level covariates, there were null associations between district-level segregation and the prevalence of inflammation, among Hispanic, non-Hispanic White, and non-Hispanic Black participants (*Table 3*). In Figure 1d, the predicted prevalence of inflammation among Hispanic adults appeared to be higher as the levels of the Black-White dissimilarity index were higher. However, there was no overall association observed between this measure of district-level school segregation and inflammation in this racial/ethnic sub-group. Similar to the findings with school racial/ethnic composition, non-Hispanic White participants did not experience a change in the predicted prevalence of inflammation across the various levels of the Black-White dissimilarity index.

Black-White Exposure Index (District-Level Segregation) and Inflammation

After adjusting for individual-, school-, and neighborhood-level covariates, there were null associations between district-level segregation and the prevalence of inflammation, among Hispanic, non-Hispanic White, and non-Hispanic Black participants (*Table 3*). Similar to the findings from the dissimilarity index, Figure 1e demonstrated that non-Hispanic White participants did not experience a change in the predicted prevalence of inflammation across the various levels of the Black-White exposure index.

In Figure 1e, the predicted prevalence of inflammation among non-Hispanic Black adults appeared to be higher as the levels of the Black-White exposure index were higher. However, there was no overall association observed among this racial/ethnic sub-group. Furthermore, there is an important finding from the Appendix that is worth noting. After adjusting for school-level characteristics, there was a significant association between the exposure index and the prevalence of inflammation in early adulthood among non-Hispanic Black adults. However, this association was attenuated after adjusting for neighborhood-level characteristics.

DISCUSSION

Our study found that there were racial/ethnic differences in the types of schools attended by the young adults who participated in Wave IV of Add Health. Non-Hispanic White participants were most likely to attend predominantly White schools, while non-Hispanic Black participants were most likely to attend predominantly Black/non-White schools. Hispanic participants were most likely to attend schools with higher levels of racial/ethnic diversity. Regarding district-level segregation, non-Hispanic Black participants were more likely to attend schools within racially-segregated districts than their Hispanic and non-Hispanic White peers. Also, the prevalence of high CRP levels among these young adults were similar across racial/ethnic groups.

Neither school racial/ethnic composition nor district-level segregation impacted the likelihood of inflammation for non-Hispanic White participants. Among Hispanic and non-Hispanic Black participants, there appeared to be increasing and decreasing trends in the predicted prevalence of inflammation, depending on the levels of school racial/ethnic composition, diversity, and district-level segregation. However, these associations did not appear to be meaningful or consistent. Additionally, the associations between the proportion of non-White students (among Hispanic adults) and levels of district-level segregation (among non-Hispanic Black adults), and the prevalence of inflammation were attenuated after the adjustment of covariates. This attenuation suggests that neighborhood-level characteristics could explain these associations among Hispanic and non-Hispanic Black students. Further, given that the prevalence of inflammation is relatively high among these young adults in Add Health, it is possible that there are other factors occurring in adolescence that could impact levels of CRP.

The findings from this study are consistent with previous research that established a relationship between childhood socioeconomic disadvantage and an increased cardiometabolic risk in adulthood[39-41]. However, to our knowledge, this is the first study that has investigated the association between the racial/ethnic structure of school environments in adolescence and cardiometabolic risk in early adulthood. Regarding school racial/ethnic composition and diversity, the findings indicate that increasing proportions of Black and non-White students is potentially harmful for Hispanic participants. It is possible that the lack of economic resources and social capital in schools that are predominantly Black or non-White can contribute to inadequate educational opportunities and other health-relevant resources accumulated over the life course, from adolescence into adulthood. Furthermore, increasing proportions of Black and non-White students in a school is potentially protective for non-Hispanic Black participants. Recent studies have demonstrated that Black students attending predominantly White schools are more likely to experience discrimination from their teachers and peers, compared to other White students[49-52]. In addition, Black students attending predominantly White schools have worse adult health outcomes compared to their White peers, as a result of the negative consequences of perceived racial discrimination that they experience while attending these schools[26]. Thus, it is possible that with increasing proportions of Black and non-White students in a school result in fewer experiences of racial discrimination. As a result of Black students not being the "other" during adolescence, they might be experiencing fewer of the physiological consequences of stress. In terms of the disruption of inflammatory processes, it may be beneficial for cardiometabolic health of Black students to attend schools during adolescence with students who look like them.

Theories from the life course framework support the findings from this study. The Pathway Model theorizes that harmful socio-contextual exposures experienced in adolescence can result in poorer health outcomes in adulthood, through its impact on social mobility and socioeconomic capital acquired from adolescence into early adulthood[53]. One consequence of attending a racially segregated school is the unequal access to educational opportunities and resources. For example, predominantly Black/Hispanic and low-income schools are more likely to have fewer advanced courses and more remedial/vocational courses, and less likely to have access to college-preparatory course than predominantly White and high-income schools[54]. This differential access to critical resources can impact graduation rates and college attendance, ultimately resulting in detrimental consequences for a student's health-relevant resources as an adult, such as educational attainment, employment, and income. Lower levels of education achieved in these segregated, and often lower-quality, schools can impact cardiometabolic risk through its impact on social support, social mobility, and one's SES that is accumulated across the life course[55-57]. Therefore, it is possible that as Black-White segregation levels, as measured by the exposure index, increase, the prevalence of inflammation also increases for Black participants in Add Health, as a result of the inequitable resources and opportunities to which they are exposed to in adolescence that carry into adulthood.

Due to the nature of the Add Health data, we were not able to estimate the risk of inflammation since adolescence (i.e., Wave I). Though this was a longitudinal study design, we were only able to estimate the prevalence of elevated CRP levels in early adulthood (i.e., at Wave IV). Additionally, Add Health is an observational study and there are likely unmeasured confounders for which we were unable to adjust. However,

we attempted to remedy this by adjusting for relevant confounders at three distinct levels: the school, the neighborhood, and the individual. Lastly, Add Health only reported Black-White segregation indices, which describe the levels of segregation only between Black and White students within a school district. The construction of these segregation indices could have contributed to the non-significant findings, and it is possible that the Hispanic-White dissimilarity and exposure indices are more relevant for Hispanic adolescents.

Our study had four important methodological strengths that are worth highlighting. First, Add Health was a nationally-representative cohort study, which used a complex stratification and sampling method. This design increased the external validity of our findings, pertaining to adolescents attending U.S. public and private schools between 1994 and 1995. Second, Wave IV of Add Health consisted of a large study population (N=15,701), which increased the precision of our estimates. Third, this is one of the first studies to investigate the association between both school-level measures (e.g., proportion Black students, proportion non-White students, school diversity) and district-level measures (e.g., Black-White dissimilarity index, Black-White exposure index) of the racial/ethnic structure of school environments and risk of inflammation. The school-level exposures investigated in this study allowed us to assess the absolute racial/ethnic make-up within a school, while the district-level exposures allowed us to assess the spatial process of racial/ethnic groups within a particular space. In measuring the exposure in these different ways, we were able to provide a more comprehensive examinations of the impact of the racial/ethnic structure of schools and school systems on cardiometabolic health in adulthood. Lastly, we were able to control for potential confounding at the three levels described above. It was critical to establish

a more comprehensive understanding of these relationships given the complicated social processes that operate within schools and school districts.

The findings from this study provide evidence that school racial/ethnic composition, diversity, and district-level segregation are possibly relevant social determinants of cardiometabolic health in later life. However, given the complex nature of U.S. middle and high schools, future research should investigate the potential mediating role of racial/ethnic discrimination in the relationship between the racial/ethnic structure of school environments and inflammation in early adulthood. Additional research is warranted that also examines other segregation indices and spatial process that are operating within school systems. Given the history of school segregation and the racial/ethnic shifts in the composition of U.S. public schools since the late 20th century, our findings can help contribute to both educational polices and the social epidemiology literature surrounding the health consequences experienced across the life course of attending racially-homogenous or segregated schools during adolescence.

Table 1. School Characteristics by Race/Ethnicity among Young Adults who Participated in Wave IV of Add Health, 2008*.

	Hispanic	Non-Hispanic White	Non-Hispanic Black
School Racial/Ethnic Composition			
Proportion Black Students**	0.16 (0.02)	0.10 (0.01)	0.58 (0.05)
Proportion Non-White Students**	0.58 (0.05)	0.17 (0.02)	0.67 (0.04)
School Entropy (Multi-Group, Standardized)**	0.54 (0.03)	0.24 (0.02)	0.36 (0.04)
District-Level Segregation			
Black-White Dissimilarity Index**	0.40 (0.04)	0.27 (0.01)	0.38 (0.03)
Black-White Exposure Index**	0.53 (0.05)	0.74 (0.03)	0.39 (0.03)

** N=15,701 adolescents participated in the In-School Questionnaire and In-Home Interview in Wave I, as well as the In-Home Interview in Wave IV of Add Health. All statistics are reported as: N; Weighted % (SE).*
***Statistics are reported as: Weighted Mean (SE).*

Table 2. Individual and Neighborhood Characteristics by Race/Ethnicity among Young Adults who Participated in Wave IV of Add Health, 2008*.

	Hispanic	Non-Hispanic White	Non-Hispanic Black
Individual Characteristics			
Age at Wave I			
10 – 13 years old	266; 26.7 (5.6)	1,527; 30.5 (3.9)	578; 24.8 (5.3)
14 – 15 years old	631; 34.8 (2.3)	2,337; 34.7 (1.5)	929; 33.7 (1.7)
16 – 19 years old	852; 38.5 (4.4)	2,455; 34.8 (2.7)	980; 41.5 (5.0)
Gender			
Male	1,119; 50.7 (1.8)	3,938; 50.7 (0.8)	1,334; 49.7 (1.4)
Parent's Educational Attainment at Wave I			
Less than HS	375; 29.1 (3.4)	347; 6.4 (0.8)	151; 9.3 (1.2)
HS Diploma/GED	428; 31.4 (2.1)	1,872; 34.2 (1.6)	546; 37.1 (2.7)
Some College or Associate's Degree	210; 12.1 (1.6)	1,037; 17.9 (0.8)	305; 18.2 (1.2)
Bachelor's Degree or Higher	348; 27.4 (2.4)	2,442; 41.5 (1.9)	1,118; 35.4 (2.9)
Physical Activity at Wave I			
Less than 5 times/week	900; 36.6 (1.8)	2,845; 33.7 (1.1)	1,134; 38.6 (1.8)
5 or more times/week	1,456; 63.4 (1.8)	8,304; 66.3 (1.1)	1,952; 61.4 (1.8)
High Sensitivity CRP at Wave IV			
High: > 3 and ≤ 10 mg/L	662; 35.1 (1.5)	2,065; 31.6 (0.8)	725; 34.5 (1.7)
Neighborhood Characteristics			
Proportion Residents Living Below Poverty Level, 1990**	0.18 (0.01)	0.11 (0.01)	0.26 (0.02)
Proportion Residents who are Black, 1990**	0.10 (0.01)	0.06 (0.01)	0.54 (0.03)
Proportion Residents who are Non-White, 1990**	0.30 (0.03)	0.09 (0.01)	0.58 (0.03)

** N=15,701 adolescents participated in the In-School Questionnaire and In-Home Interview in Wave I, as well as the In-Home Interview in Wave IV of Add Health. All statistics are reported as: N; Weighted % (SE).*

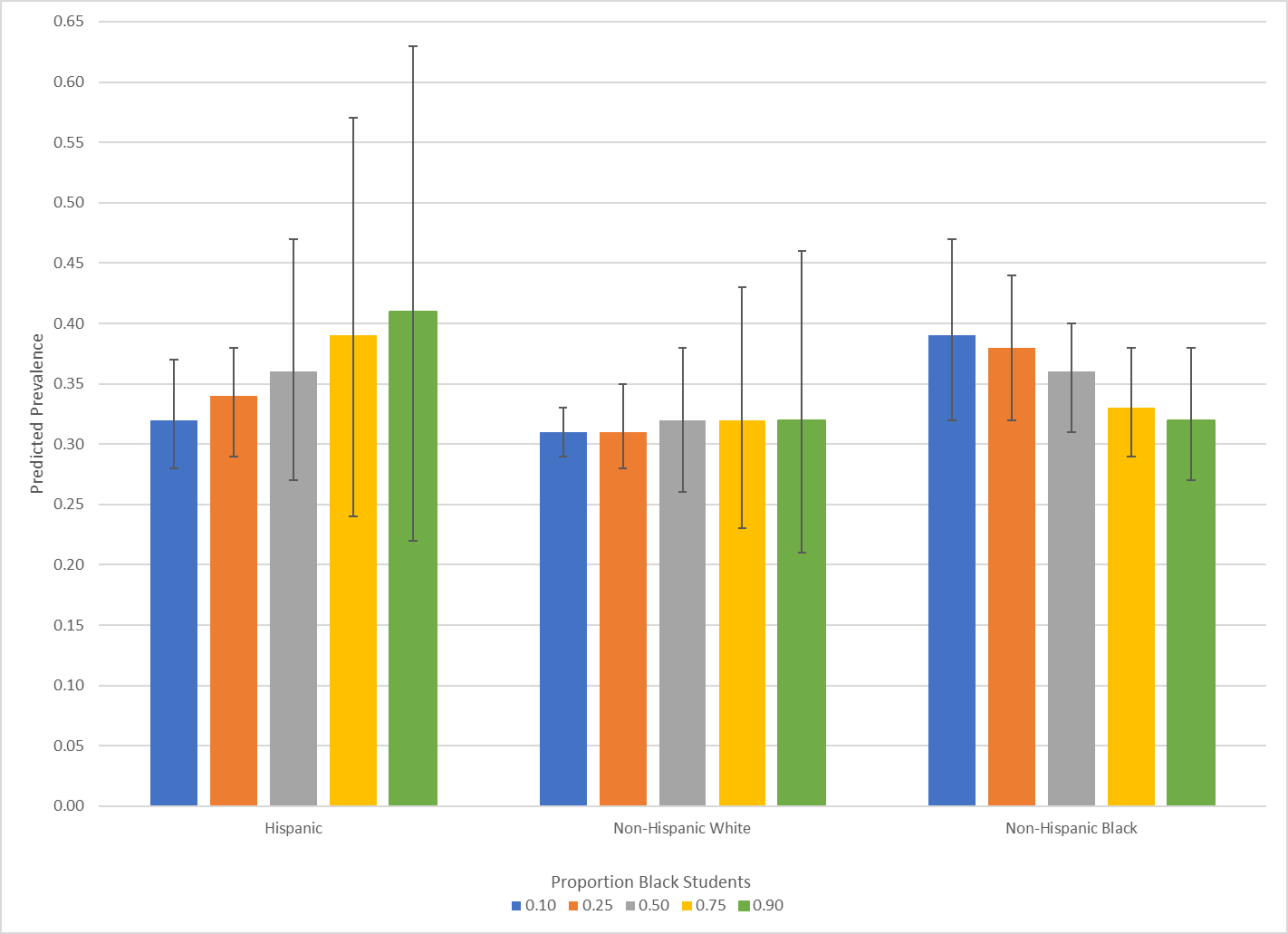
***Statistics are reported as: Weighted Mean (SE).*

Table 3. Adjusted Prevalence Ratios* for Inflammation in Early Adulthood by the Racial/Ethnic Structure of Schools and School Systems during Adolescence and Individual Race/Ethnicity, Add Health Waves I (1994-5) and IV (2008).

	Total Population	Hispanic	Non-Hispanic White	Non-Hispanic Black
Proportion Black Students	1.03 (0.70, 1.52)	1.69 (0.44, 6.45)	1.07 (0.52, 2.20)	0.65 (0.36, 1.16)
Proportion Non-White Students	1.10 (0.78, 1.55)	1.70 (0.63, 4.58)	1.13 (0.64, 2.00)	0.75 (0.41, 1.37)
School Entropy (Multi-Group)	1.14 (0.85, 1.54)	0.95 (0.36, 2.50)	1.06 (0.65, 1.73)	1.67 (0.76, 3.65)
Black-White Dissimilarity Index	0.93 (0.60, 1.45)	2.17 (0.75, 6.31)	1.00 (0.49, 2.05)	1.05 (0.43, 2.55)
Black-White Exposure Index	1.03 (0.77, 1.39)	0.54 (0.24, 1.24)	0.98 (0.68, 1.42)	1.74 (0.82, 3.71)

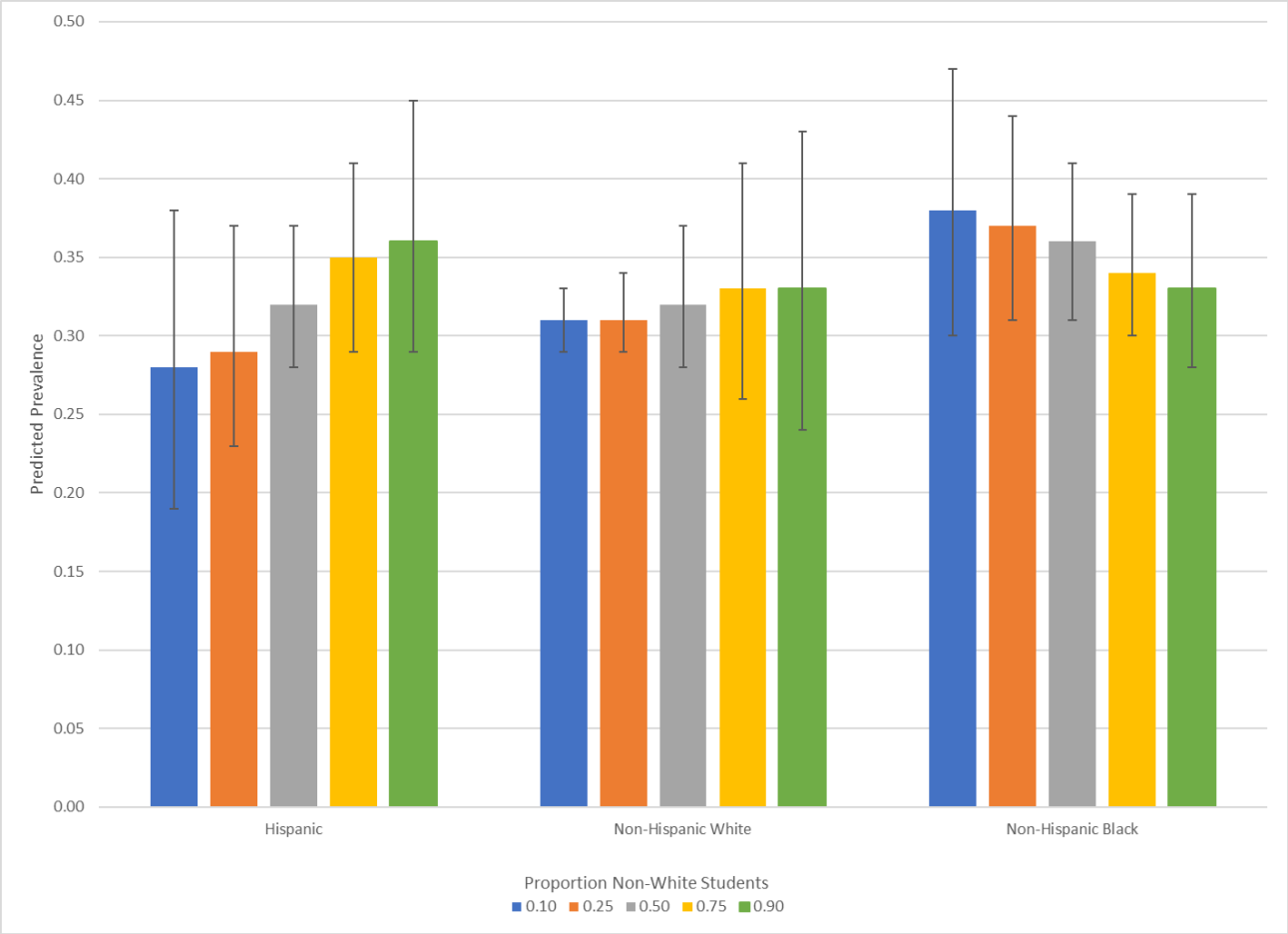
**Adjusted for school-level characteristics (school size, school urbanicity, school type), neighborhood-level characteristics (neighborhood proportion living in poverty, neighborhood proportion who are black), and individual-level characteristics (age, gender, parent's highest education, physical activity).*

Figure 1a. Adjusted* Predicted Prevalence for Inflammation in Early Adulthood by Proportion Black Students in a School and Individual Race/Ethnicity.



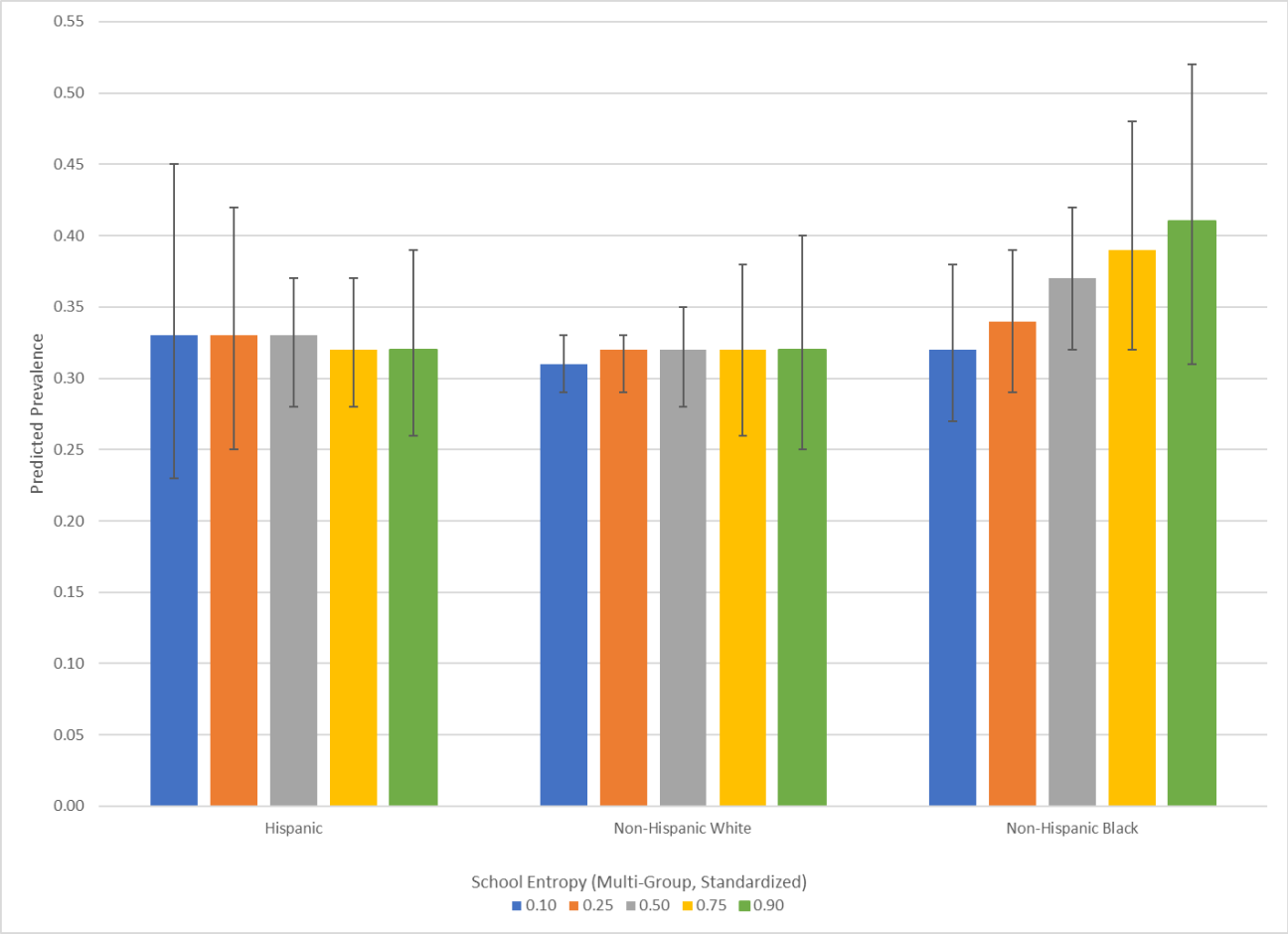
**Adjusted for school-level characteristics (school size, school urbanicity, school type), neighborhood-level characteristics (neighborhood proportion living in poverty, neighborhood proportion who are black), and individual-level characteristics (age, gender, parent's highest education, physical activity).*

Figure 1b. Adjusted* Predicted Prevalence for Inflammation in Early Adulthood by Proportion Non-White Students in a School and Individual Race/Ethnicity.



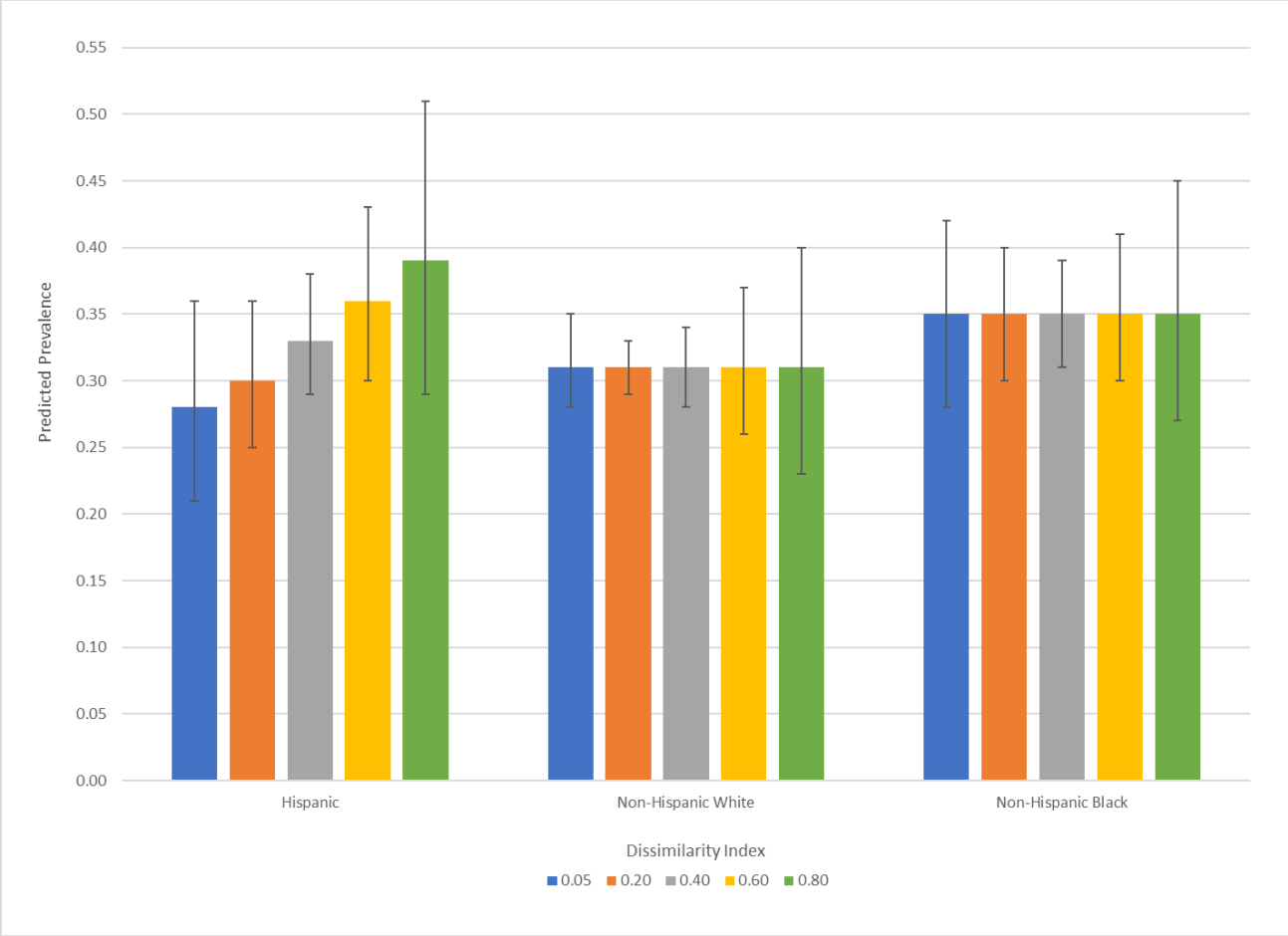
**Adjusted for school-level characteristics (school size, school urbanicity, school type), neighborhood-level characteristics (neighborhood proportion living in poverty, neighborhood proportion who are non-white), and individual-level characteristics (age, gender, parent's highest education, physical activity).*

Figure 1c. Adjusted* Predicted Prevalence for Inflammation in Early Adulthood by School Entropy (Multi-Group, Standardized) and Individual Race/Ethnicity.



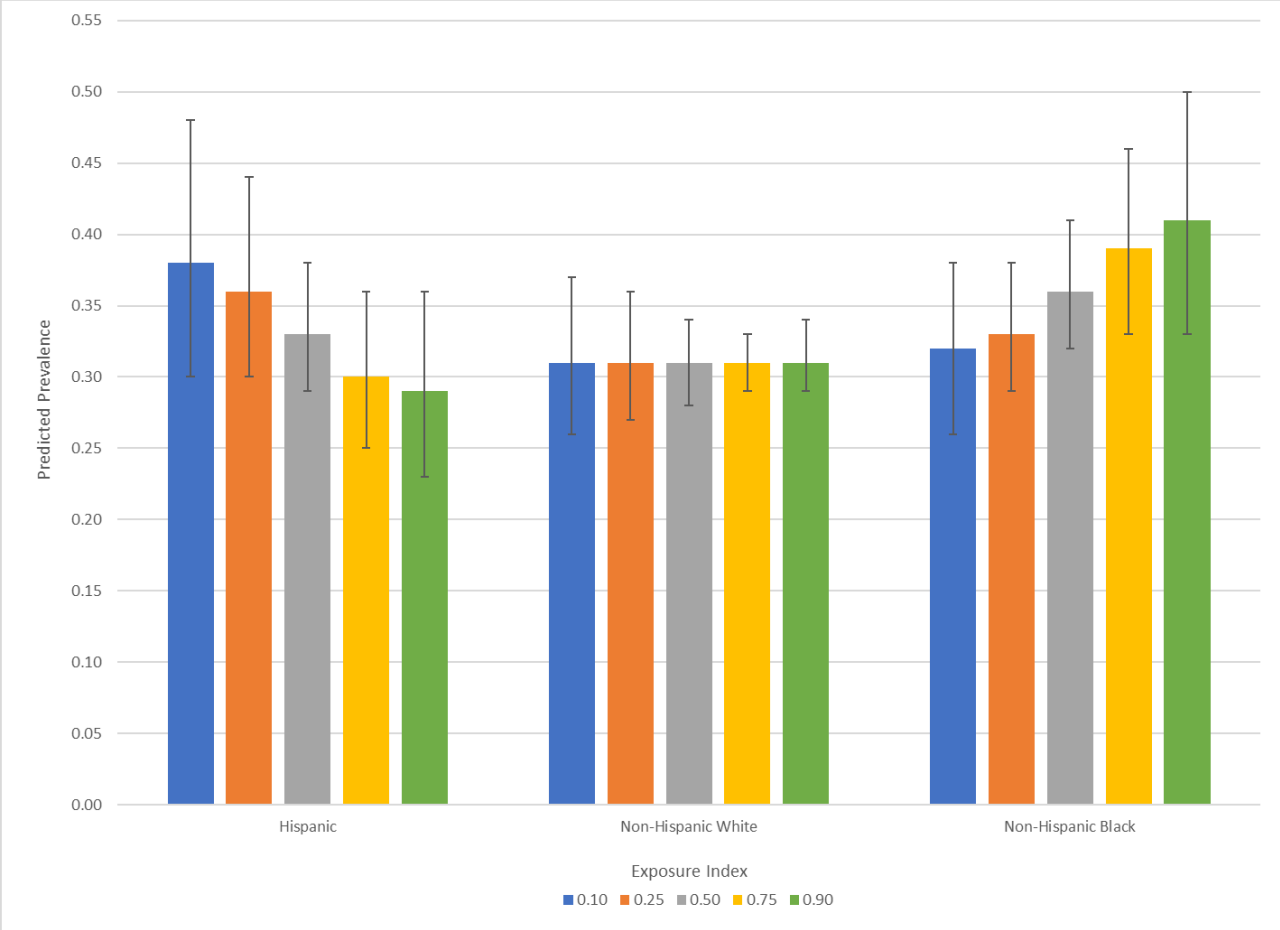
**Adjusted for school-level characteristics (school size, school urbanicity, school type), neighborhood-level characteristics (neighborhood proportion living in poverty, neighborhood proportion who are non-white), and individual-level characteristics (age, gender, parent's highest education, physical activity).*

Figure 1d. Adjusted* Predicted Prevalence for Inflammation in Early Adulthood by Average School Segregation – Black-White Dissimilarity Index – and Individual Race/Ethnicity.



**Adjusted for school-level characteristics (school size, school urbanicity, school type), neighborhood-level characteristics (neighborhood proportion living in poverty, neighborhood proportion who are non-white), and individual-level characteristics (age, gender, parent's highest education, physical activity).*

Figure 1e. Adjusted* Predicted Prevalence for Inflammation in Early Adulthood by Average School Segregation – Black-White Exposure Index – and Individual Race/Ethnicity.



**Adjusted for school-level characteristics (school size, school urbanicity, school type), neighborhood-level characteristics (neighborhood proportion living in poverty, neighborhood proportion who are non-white), and individual-level characteristics (age, gender, parent's highest education, physical activity).*

REFERENCES

1. Ford ES, Ajani UA, Croft JB, Critchley JA, Labarthe DR, Kottke TE, et al. Explaining the decrease in U.S. deaths from coronary disease, 1980-2000. *N Engl J Med*. 2007; 356(23):2388-98.
2. Benjamin EJ, Blaha MJ, Chiuve SE, Cushman M, Das SR, Deo R, et al. Heart disease and stroke statistics – 2017 update: A report from the American Heart Association. *Circulation*. 2017; 135(10):e146-e603.
3. Koenig W. Inflammation and coronary heart disease: An overview. *Cardiol Rev*. 2001; 9(1):31-5.
4. Buckley DI, Fu R, Freeman M, Rogers K, Helfand M. C-reactive protein as a risk factor for coronary heart disease: A systematic review and meta-analyses for the U.S. Preventive Services Task Force. *Ann Intern Med*. 2009; 151(7):483-95.
5. Koenig W, Sund M, Frölich M, Fischer HG, Löwel H, Döring A, et al. C-reactive protein, a sensitive marker of inflammation, predicts future risk of coronary heart disease in initially healthy middle-aged men: Results from the MONICA (Monitoring Trends and Determinants in Cardiovascular Disease) Augsburg Cohort Study, 1984-1992. *Circulation*. 1999; 99(2):237-42.
6. Emerging Risk Factors Collaboration, Kaptoge S, Di Angelantonio E, Lowe G, Pepys MB, Thompson SG, et al. C-reactive protein concentration and risk of coronary heart disease, stroke, and mortality: An individual participant meta-analysis. *Lancet*. 2010; 375(9709):132-40.
7. Rifai N, Ridker PM. High-sensitivity C-reactive protein: A novel and promising marker of coronary heart disease. *Clin Chem*. 2001; 47(3):403-11.
8. Cushman M, Arnold AM, Psaty BM, Manolio TA, Kuller LH, Burke GL, et al. C-reactive protein and the 10-year incidence of coronary heart disease in older men and women: The cardiovascular health study. *Circulation*. 2005; 112(1):25-31.
9. Ridker PM, Buring JE, Shih J, Matias M, Hennekens CH. Prospective study of C-reactive protein and the risk of future cardiovascular events among apparently healthy women. *Circulation*. 1998; 98(8):731-3.
10. Chang MK, Binder CJ, Torzewski M, Witztum JL. C-reactive protein binds to both oxidized LDL and apoptotic cells through recognition of a common ligand: Phosphorylcholine of oxidized phospholipids. *Proc Natl Acad Sci U S A*. 2002; 99(20):13043-8.
11. Zwaka TP, Hombach V, Torzewski J. C-reactive protein-mediated low density lipprotein uptake by macrophages: Implications for atherosclerosis. *Circulation*. 2001; 103(9):1194-7.
12. Black S, Kushner I, Smols D. C-reactive protein. *J Biol Chem*. 2004; 279(47):48487-90.
13. Hirschfield GM, Pepys MB. C-reactive protein and cardiovascular disease: New insights from an old molecule. *QJM*. 2003; 96(11):793-807.

14. Zhang YX, Cliff WJ, Schoefl GI, Higgins G. Coronary C-reactive protein distribution: Its relation to development of atherosclerosis. *Atherosclerosis*. 1999; 145(2):375-9.
15. Ford ES, Giles WH, Mokdad AH, Myers GL. Distribution and correlates of C-reactive protein concentrations among adult US women. *Clin Chem*. 2004; 50(3):574-81.
16. Ong KL, Allison MA, Cheung BM, Wu BJ, Barter PJ, Rye KA. Trends in C-reactive protein levels in US adults from 1999 to 2010. *Am J Epidemiol*. 2013; 177(12):1430-42.
17. Lu MC, Halfon N. Racial and ethnic disparities in birth outcomes: A life-course perspective. *Matern Child Health J*. 2003; 7(1):13-30.
18. McEwen BS. Protective and damaging effects of stress mediators. *N Engl J Med*. 1998; 338(3):171-9.
19. McEwen BS, Seeman T. Protective and damaging effects of mediators of stress: Elaborating and testing the concepts of allostasis and allostatic load. *Ann N Y Acad Sci*. 1999; 896:30-47.
20. Singh-Manoux A, Ferrie JE, Chandola T, Marmot M. Socioeconomic trajectories across the life course and health outcomes in midlife: Evidence for the accumulation hypothesis? *Int J Epidemiol*. 2004; 33(5):1072-9.
21. Nazmi A, Victoria CG. Socioeconomic and racial/ethnic differentials of C-reactive protein levels: A systematic review of population-based studies. *BMC Public Health*. 2007; 7:212.
22. Gruenewald TL, Cohen S, Matthews KA, Tracy R, Seeman TE. Association of socioeconomic status with inflammation markers in black and white men and women in the Coronary Artery Risk Development in Young Adults (CARDIA) study. *Soc Sci Med*. 2009; 69(3):451-9.
23. Dinwiddie GY, Zambrana RE, Doamekpor LA, Lopez L. The impact of educational attainment on observed race/ethnic disparities in inflammatory risk in the 2001-2008 National Health and Nutrition Examination Survey. *Int J Environ Res Public health*. 2015; 13(1):ijerph13010042.
24. Khera A, McGuire DK, Murphy SA, Stanek HD, Das SR, Vongpatanasin W, et al. Race and gender differences in C-reactive protein levels. *J Am Coll Cardiol*. 2005; 46(3):464-9.
25. Stepanikova I, Bateman LB, Oates GR. Systemic inflammation in midlife: Race, socioeconomic status, and perceived discrimination. *Am J Prev Med*. 2017; 52(1S1:S63-S76.
26. Goosby BJ, Walsemann KM. School racial composition and race/ethnic differences in early adulthood health. *Health Place*. 2012; 18(2):296-304.
27. Plessy v. Ferguson, 163 U.S. 537 (1896).
28. Tischauser LV. *Jim Crow Laws: Landmarks of the American Mosaic*. Greenwood: Santa Barbara, CA; 2012.

29. Clotfelter CT. *After Brown: The Rise and Retreat of School Desegregation*. Princeton University Press: Princeton, NJ; 2004.
30. Brown v. Board of Education of Topeka, 347 U.S. 483 (1954).
31. Milliken v. Bradley, 418 U.S. 717 (1974).
32. Riddick v. School Board of the City of Norfolk, Virginia 784 F.2d 251 (4th Cir. 1986).
33. Board of Education of Oklahoma v. Dowell, 498 U.S. 237 (1991).
34. Freeman v. Pitts, 503 U.S. 567 (1992).
35. Reardon SF, Yun JT, Mcnulty Eitle T. The changing structure of school segregation: Measurement and evidence of multiracial metropolitan-area school segregation, 1989-1995. *Demography*. 2000; 37(3):351-64.
36. Williams DR. Race, socioeconomic status, and health: The added effects of racism and discrimination. *Ann N Y Acad Sci*. 1999; 896:173-88.
37. Orfield G, Eaton SE, The Harvard Project on School Desegregation. *Dismantling Desegregation: The Quiet Reversal of Brown v. Board of Education*. New Press: New York, NY; 1996.
38. Link BG, Phelan J. Social conditions as fundamental causes of disease. *J Health Soc Behav*. 1995; (Spec No):80-94.
39. Carmeli C, Steen J, Petrovic D, Lepage B, Delpierre C, Kelly-Irving M, et al. Mechanisms of life-course socioeconomic inequalities in adult systemic inflammation: Findings from two cohort studies. *Soc Sci Med*. 2019 Nov 19; 245:112685 [Epub ahead of print].
40. Yang YC, Gerken K, Schorpp K, Boen C, Harris KM. Early-life socioeconomic status and adult physiological functioning: A life course examination of biosocial mechanisms. *Biodemography Soc Biol*. 2017; 63(2):87-103.
41. Liu RS, Aiello AE, Mensah FK, Gasser CE, Rueb K, Cordell B, et al. Socioeconomic status in childhood and C-reactive protein in adulthood: A systematic review and meta-analysis. *J Epidemiol Community Health*. 2017; 71(8):817-26.
42. Tamayo T, Christian H, Rathmann W. Impact of early psychosocial factors (childhood socioeconomic factors and adversities) on future risk of type 2 diabetes, metabolic disturbances and obesity: A systematic review. *BMC Public Health*. 2010; 10:525.
43. Martin CL, Kane JB, Miles GL, Aiello AE, Harris KM. Neighborhood disadvantage across the transition from adolescence to adulthood and risk of metabolic syndrome. *Health Place*. 2019; 57:131-8.
44. Harris KM. *The Add Health Study: Design and Accomplishments*. Carolina Population Center, University of North Carolina at Chapel Hill, 2013. Available at: http://www.cpc.unc.edu/projects/addhealth/data/guides/DesignPaperWIIV.pdf .

45. Whitsel EA, Cuthbertson CC, Tabor JW, Potter AJ, Wener MH, Killeya-Jones LA, Harris KM. *Add Health Wave IV Documentation: Measures of Inflammation and Immune Function*. Carolina Population Center, University of North Carolina at Chapel Hill, 2012. Available at: https://www.cpc.unc.edu/projects/addhealth/documentation/guides/Wave_IV_hsCRP_EBV_Documentation.pdf.
46. Pearson TA, Mensah GA, Alexander RW, Anderson JL, Cannon RO 3rd, Criqui M, et al. Markers of inflammation and cardiovascular disease. Application to clinical and public health practice. A statement for healthcare professionals from the CDC and AHA. *Circulation*. 2003; 107(3):499-511.
47. Johnson R. *Add Health Wave I School District Data Documentation*. Carolina Population Center, University of North Carolina at Chapel Hill, 2020. Available at: http://www.cpc.unc.edu/addhealth.
48. Bazaco MC, Pereira MA, Wisniewski SR, Zgibor JC, Songer TJ, Burke JD, et al. Is there a relationship between perceived neighborhood contentedness and physical activity in you men and women? *J Urban Health*. 2016; 93(6):940-52.
49. McCabe J. Racial and gender microaggressions on a predominantly-white campus: Experiences of black, Latina/o and white undergraduates. *Race, Gender & Class*. 2009; 16(1/2):133-51.
50. Juvonen J, Nishina A, Graham S. Ethnic diversity and perceptions of safety in urban middle schools. *Psychological Science*. 2006; 17(5):393-400.
51. Feagin JR, Vera H, Imani N. *The Agony of Education: Black Students at a White University* (2nd ed.). Routledge: New York, NY; 1996.
52. Allen WR. The color of success: African-American college student outcomes at predominantly white and historically black public colleges and universities. *Harvard Educational Review*. 1992; 62(1):26-44.
53. Marmot M, Shipley M, Brunner E, Hemingway H. Relative contribution of early life and adult socioeconomic factors to adult morbidity in the Whitehall II study. *J Epidemiol Community Health*. 2001; 55(5):301-7.
54. Smrekar CE, Goldring EB, eds. *From the Courtroom to the Classroom: The Shifting Landscape of School Desegregation*. Harvard Education Press: Cambridge, MA; 2009.
55. Hahn RA, Truman BI. Education improves public health and promotes health equity. *Int J Health Serv*. 2015; 45(4):657-78.
56. Ross CE, Wu CL. The links between education and health. *American Sociological Review*. 1995; 60(5):719-45.
57. Ross CE, Wu CL. Education, age, and the cumulative advantage in health. *J Health Soc Behav*. 1996; 37(1):104-20.

SPECIFIC AIM 2: APPENDIX

A2.1. Unadjusted Prevalence Ratios for Inflammation in Early Adulthood by Proportion of Black Students in a School during Adolescence, Add Health Waves I (1994-5) and IV (2008).

	Total Population	Hispanic	Non-Hispanic White	Non-Hispanic Black
Proportion Black Students	1.19 (0.94, 1.51)	2.74 (0.83, 9.06)	1.04 (0.61, 1.77)	0.86 (0.50, 1.49)
0.10*	0.31 (0.01, 0.30)	0.33 (0.29, 0.37)	0.31 (0.29, 0.33)	0.37 (0.30, 0.45)
0.25*	0.32 (0.30, 0.33)	0.36 (0.32, 0.41)	0.31 (0.29, 0.34)	0.36 (0.31, 0.43)
0.50*	0.33 (0.31, 0.35)	0.42 (0.32, 0.53)	0.32 (0.27, 0.37)	0.36 (0.31, 0.40)
0.75*	0.34 (0.31, 0.37)	0.48 (0.31, 0.66)	0.32 (0.25, 0.40)	0.35 (0.31, 0.39)
0.90*	0.34 (0.31, 0.38)	0.52 (0.31, 0.73)	0.32 (0.23, 0.42)	0.34 (0.29, 0.40)

**Predicted Prevalence (95% CIs).*

A2.2. Adjusted Prevalence Ratios for Inflammation in Early Adulthood by Proportion of Black Students in a School during Adolescence, Add Health Waves I (1994-5) and IV (2008).

	Total Population	Hispanic	Non-Hispanic White	Non-Hispanic Black
Proportion Black Students	1.22 (0.96, 1.53)	2.96 (0.94, 9.28)	1.24 (0.70, 2.22)	0.57 (0.32, 1.02)
0.10*	0.31 (0.30, 0.33)	0.33 (0.29, 0.36)	0.31 (0.29, 0.33)	0.42 (0.33, 0.51)
0.25*	0.32 (0.30, 0.33)	0.36 (0.32, 0.41)	0.32 (0.29, 0.35)	0.40 (0.33, 0.47)
0.50*	0.33 (0.31, 0.35)	0.43 (0.33, 0.53)	0.33 (0.28, 0.39)	0.36 (0.32, 0.41)
0.75*	0.34 (0.31, 0.37)	0.49 (0.33, 0.66)	0.34 (0.26, 0.44)	0.33 (0.29, 0.37)
0.90*	0.35 (0.31, 0.39)	0.53 (0.33, 0.73)	0.35 (0.25, 0.46)	0.31 (0.27, 0.36)
School Size				
1 – 400 students	1.15 (0.99, 1.33)	1.00 (0.71, 1.41)	1.17 (0.98, 1.38)	1.21 (0.85, 1.72)
401 – 1,000 students	1.00 (0.90, 1.11)	1.08 (0.87, 1.34)	0.96 (0.84, 1.10)	1.34 (1.03, 1.74)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.87 (0.76, 0.99)	0.81 (0.53, 1.24)	0.87 (0.72, 1.05)	0.88 (0.67, 1.16)
Suburban	0.92 (0.82, 1.04)	0.88 (0.58, 1.34)	0.94 (0.81, 1.09)	1.05 (0.83, 1.34)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.20 (1.03, 1.40)	1.14 (0.71, 1.85)	1.10 (0.91, 1.33)	2.30 (1.14, 4.64)
Private	1.00	1.00	1.00	1.00

**Predicted Prevalence (95% CIs).*

A2.3. Adjusted Prevalence Ratios for Inflammation in Early Adulthood by Proportion of Black Students in a School during Adolescence, Add Health Waves I (1994-5) and IV (2008).

	Total Population	Hispanic	Non-Hispanic White	Non-Hispanic Black
Proportion Black Students	1.04 (0.77, 1.40)	1.76 (0.61, 5.05)	1.15 (0.60, 2.22)	0.66 (0.34, 1.28)
0.10*	0.32 (0.30, 0.33)	0.34 (0.30, 0.38)	0.31 (0.29, 0.33)	0.40 (0.31, 0.50)
0.25*	0.32 (0.30, 0.33)	0.35 (0.32, 0.39)	0.32 (0.29, 0.35)	0.39 (0.32, 0.46)
0.50*	0.32 (0.30, 0.34)	0.39 (0.30, 0.48)	0.32 (0.27, 0.39)	0.36 (0.32, 0.41)
0.75*	0.32 (0.28, 0.36)	0.42 (0.28, 0.57)	0.33 (0.24, 0.43)	0.34 (0.20, 0.38)
0.90*	0.32 (0.28, 0.37)	0.44 (0.27, 0.63)	0.34 (0.23, 0.46)	0.33 (0.28, 0.38)
School Size				
1 – 400 students	1.11 (0.96, 1.29)	0.98 (0.68, 1.39)	1.12 (0.95, 1.33)	1.13 (0.77, 1.64)
401 – 1,000 students	0.99 (0.89, 1.10)	1.08 (0.87, 1.34)	0.95 (0.82, 1.08)	1.35 (1.05, 1.75)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.86 (0.76, 0.98)	0.76 (0.48, 1.21)	0.88 (0.73, 1.06)	0.90 (0.68, 1.19)
Suburban	0.94 (0.83, 1.05)	0.83 (0.53, 1.30)	0.96 (0.83, 1.11)	1.07 (0.85, 1.35)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.17 (1.01, 1.37)	1.25 (0.72, 2.18)	1.07 (0.89, 1.30)	2.05 (1.03, 4.10)
Private	1.00	1.00	1.00	1.00
Neighborhood Poverty, 1990	1.83 (0.94, 3.56)	0.51 (0.12, 2.14)	2.17 (0.69, 6.85)	2.74 (1.01, 7.45)
Neighborhood Proportion Black, 1990	1.08 (0.79, 1.48)	3.76 (1.33, 10.62)	0.93 (0.44, 2.01)	0.65 (0.38, 1.12)

**Predicted Prevalence (95% CIs).*

A2.4. Adjusted Relative Risks for Inflammation in Early Adulthood by Proportion of Black Students in a School during Adolescence, Add Health Waves I (1994-5) and IV (2008).

	Total Population	Hispanic	Non-Hispanic White	Non-Hispanic Black
Proportion Black Students	1.03 (0.70, 1.52)	1.69 (0.44, 6.45)	1.07 (0.52, 2.20)	0.65 (0.36, 1.16)
0.10*	0.31 (0.29, 0.33)	0.32 (0.28, 0.37)	0.31 (0.29, 0.33)	0.39 (0.32, 0.47)
0.25*	0.31 (0.30, 0.33)	0.34 (0.29, 0.38)	0.31 (0.28, 0.35)	0.38 (0.32, 0.44)
0.50*	0.32 (0.29, 0.35)	0.36 (0.27, 0.47)	0.32 (0.26, 0.38)	0.36 (0.31, 0.40)
0.75*	0.32 (0.27, 0.37)	0.39 (0.24, 0.57)	0.32 (0.23, 0.43)	0.33 (0.29, 0.38)
0.90*	0.32 (0.26, 0.38)	0.41 (0.22, 0.63)	0.32 (0.21, 0.46)	0.32 (0.27, 0.38)
School Size				
1 – 400 students	1.14 (0.97, 1.35)	1.06 (0.74, 1.50)	1.10 (0.90, 1.34)	1.34 (0.97, 1.84)
401 – 1,000 students	1.04 (0.92, 1.17)	1.04 (0.74, 1.45)	0.96 (0.83, 1.11)	1.77 (1.35, 2.34)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.85 (0.73, 0.98)	0.75 (0.42, 1.33)	0.90 (0.74, 1.09)	0.92 (0.64, 1.31)
Suburban	0.92 (0.81, 1.04)	0.94 (0.55, 1.62)	0.95 (0.81, 1.12)	1.01 (0.77, 1.31)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.04 (0.86, 1.27)	1.21 (0.68, 2.15)	0.96 (0.78, 1.20)	2.32 (1.05, 5.14)
Private	1.00	1.00	1.00	1.00
Neighborhood Poverty, 1990	1.51 (0.68, 3.35)	0.42 (0.06, 3.09)	1.55 (0.44, 5.52)	5.43 (1.60, 18.37)
Neighborhood Proportion Black, 1990	1.11 (0.73, 1.70)	5.43 (1.34, 21.90)	1.05 (0.45, 2.44)	0.60 (0.35, 1.04)
Age at Wave I				
10-13 years old	0.94 (0.81, 1.09)	1.06 (0.71, 1.59)	0.97 (0.81, 1.16)	0.71 (0.54, 0.94)
14-15 years old	1.02 (0.92, 1.14)	0.96 (0.71, 1.30)	1.05 (0.91, 1.21)	0.91 (0.72, 1.14)
16-19 years old	1.00	1.00	1.00	1.00
Gender				
Male	1.00	1.00	1.00	1.00
Female	1.58 (1.44, 1.73)	1.95 (1.45, 2.62)	1.55 (1.38, 1.74)	1.73 (1.33, 2.25)
Parental Education at Wave I				
<HS	1.18 (0.97, 1.43)	1.25 (0.86, 1.80)	1.34 (1.05, 1.71)	0.73 (0.53, 1.01)
HS Diploma	1.19 (1.07, 1.34)	1.38 (0.97, 1.96)	1.22 (1.07, 1.40)	0.89 (0.75, 1.06)
Some College	1.07 (0.93, 1.23)	0.96 (0.61, 1.51)	1.08 (0.92, 1.27)	1.04 (0.81, 1.33)
College+	1.00	1.00	1.00	1.00

Physical Activity at Wave I				
<5 times/week	1.05 (0.95, 1.17)	0.77 (0.57, 1.03)	1.11 (0.96, 1.27)	0.97 (0.80, 1.18)
5+ times/week	1.00	1.00	1.00	1.00

**Predicted Prevalence (95% CIs).*

A2.5. Unadjusted Prevalence Ratios for Inflammation in Early Adulthood by Proportion of Non-White Students in a School during Adolescence, Add Health Waves I (1994-5) and IV (2008).

	Total Population	Hispanic	Non-Hispanic White	Non-Hispanic Black
Proportion Non-White Students	1.11 (0.90, 1.37)	1.67 (0.88, 3.20)	1.03 (0.71, 1.50)	0.79 (0.43, 1.44)
0.10*	0.31 (0.29, 0.33)	0.29 (0.22, 0.37)	0.31 (0.29, 0.33)	0.38 (0.29, 0.48)
0.25*	0.31 (0.30, 0.33)	0.30 (0.25, 0.37)	0.31 (0.29, 0.33)	0.38 (0.30, 0.45)
0.50*	0.32 (0.30, 0.34)	0.33 (0.29, 0.37)	0.32 (0.28, 0.35)	0.36 (0.31, 0.41)
0.75*	0.33 (0.30, 0.35)	0.36 (0.32, 0.41)	0.32 (0.27, 0.37)	0.35 (0.31, 0.39)
0.90*	0.33 (0.30, 0.36)	0.38 (0.32, 0.44)	0.32 (0.26, 0.38)	0.34 (0.30, 0.39)

**Predicted Prevalence (95% CIs).*

A2.6. Adjusted Prevalence Ratios for Inflammation in Early Adulthood by Proportion of Non-White Students in a School during Adolescence, Add Health Waves I (1994-5) and IV (2008).

	Total Population	Hispanic	Non-Hispanic White	Non-Hispanic Black
Proportion Non-White Students	1.23 (1.00, 1.51)	2.09 (1.11, 3.94)	1.29 (0.85, 1.97)	0.62 (0.36, 1.07)
0.10*	0.31 (0.29, 0.33)	0.27 (0.20, 0.34)	0.31 (0.29, 0.33)	0.42 (0.33, 0.51)
0.25*	0.31 (0.30, 0.33)	0.29 (0.23, 0.35)	0.32 (0.30, 0.34)	0.40 (0.33, 0.48)
0.50*	0.32 (0.31, 0.34)	0.33 (0.29, 0.37)	0.33 (0.30, 0.37)	0.37 (0.32, 0.42)
0.75*	0.34 (0.31, 0.36)	0.37 (0.33, 0.41)	0.35 (0.29, 0.41)	0.34 (0.31, 0.38)
0.90*	0.34 (0.31, 0.38)	0.39 (0.34, 0.45)	0.35 (0.28, 0.43)	0.33 (0.29, 0.37)
School Size				
1 – 400 students	1.16 (1.00, 1.35)	1.06 (0.77, 1.46)	1.18 (0.99, 1.40)	1.14 (0.81, 1.61)
401 – 1,000 students	1.01 (0.91, 1.13)	1.11 (0.87, 1.40)	0.96 (0.84, 1.10)	1.29 (1.00, 1.66)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.84 (0.74, 0.96)	0.85 (0.60, 1.20)	0.85 (0.71, 1.02)	0.91 (0.69, 1.19)
Suburban	0.91 (0.81, 1.03)	1.02 (0.75, 1.39)	0.93 (0.80, 1.07)	1.06 (0.84, 1.34)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.19 (1.02, 1.39)	1.07 (0.71, 1.61)	1.10 (0.91, 1.33)	2.11 (1.02, 4.38)
Private	1.00	1.00	1.00	1.00

**Predicted Prevalence (95% CIs).*

A2.7. Adjusted Prevalence Ratios for Inflammation in Early Adulthood by Proportion of Non-White Students in a School during Adolescence, Add Health Waves I (1994-5) and IV (2008).

	Total Population	Hispanic	Non-Hispanic White	Non-Hispanic Black
Proportion Non-White Students	1.20 (0.92, 1.56)	1.72 (0.81, 3.64)	1.28 (0.77, 2.14)	0.76 (0.41, 1.42)
0.10*	0.31 (0.29, 0.33)	0.29 (0.21, 0.38)	0.31 (0.29, 0.33)	0.39 (0.30, 0.49)
0.25*	0.31 (0.30, 0.33)	0.30 (0.24, 0.38)	0.32 (0.29, 0.34)	0.38 (0.31, 0.46)
0.50*	0.32 (0.30, 0.34)	0.33 (0.29, 0.38)	0.33 (0.29, 0.37)	0.36 (0.32, 0.42)
0.75*	0.33 (0.30, 0.36)	0.36 (0.32, 0.41)	0.34 (0.28, 0.42)	0.35 (0.31, 0.39)
0.90*	0.34 (0.30, 0.38)	0.38 (0.32, 0.44)	0.35 (0.27, 0.44)	0.34 (0.29, 0.39)
School Size				
1 – 400 students	1.12 (0.97, 1.30)	1.07 (0.78, 1.49)	1.13 (0.95, 1.34)	1.07 (0.73, 1.57)
401 – 1,000 students	1.00 (0.89, 1.11)	1.13 (0.89, 1.43)	0.95 (0.82, 1.09)	1.31 (1.02, 1.68)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.84 (0.75, 0.95)	0.84 (0.60, 1.17)	0.85 (0.71, 1.02)	0.94 (0.71, 1.23)
Suburban	0.93 (0.83, 1.04)	0.94 (0.69, 1.28)	0.95 (0.82, 1.10)	1.08 (0.86, 1.36)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.15 (0.99, 1.35)	1.16 (0.74, 1.82)	1.07 (0.88, 1.29)	1.87 (0.91, 3.81)
Private	1.00	1.00	1.00	1.00
Neighborhood Poverty, 1990	1.95 (0.95, 4.01)	0.34 (0.07, 1.70)	2.18 (0.72, 6.56)	2.44 (0.90, 6.61)
Neighborhood Proportion Non-White, 1990	0.91 (0.64, 1.29)	1.99 (0.90, 4.41)	0.87 (0.40, 1.91)	0.64 (0.37, 1.11)

**Predicted Prevalence (95% CIs).*

A2.8. Adjusted Relative Risks for Inflammation in Early Adulthood by Proportion of Non-White Students in a School during Adolescence, Add Health Waves I (1994-5) and IV (2008).

	Total Population	Hispanic	Non-Hispanic White	Non-Hispanic Black
Proportion Non-White Students	1.10 (0.78, 1.55)	1.70 (0.63, 4.58)	1.13 (0.64, 2.00)	0.75 (0.41, 1.37)
0.10*	0.31 (0.29, 0.33)	0.28 (0.19, 0.38)	0.31 (0.29, 0.33)	0.38 (0.30, 0.47)
0.25*	0.31 (0.30, 0.33)	0.29 (0.23, 0.37)	0.31 (0.29, 0.34)	0.37 (0.31, 0.44)
0.50*	0.32 (0.30, 0.34)	0.32 (0.28, 0.37)	0.32 (0.28, 0.37)	0.36 (0.31, 0.41)
0.75*	0.32 (0.29, 0.36)	0.35 (0.29, 0.41)	0.33 (0.26, 0.41)	0.34 (0.30, 0.39)
0.90*	0.33 (0.28, 0.37)	0.36 (0.29, 0.45)	0.33 (0.24, 0.43)	0.33 (0.28, 0.39)
School Size				
1 – 400 students	1.14 (0.97, 1.34)	1.13 (0.76, 1.68)	1.10 (0.90, 1.34)	1.25 (0.91, 1.73)
401 – 1,000 students	1.04 (0.92, 1.17)	1.06 (0.73, 1.55)	0.96 (0.83, 1.12)	1.71 (1.30, 2.26)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.84 (0.72, 0.98)	0.79 (0.50, 1.27)	0.89 (0.73, 1.08)	0.95 (0.66, 1.37)
Suburban	0.92 (0.81, 1.05)	1.05 (0.67, 1.62)	0.95 (0.81, 1.11)	1.01 (0.77, 1.33)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.03 (0.85, 1.26)	1.13 (0.67, 1.89)	0.96 (0.77, 1.20)	2.11 (0.91, 4.90)
Private	1.00	1.00	1.00	1.00
Neighborhood Poverty, 1990	1.76 (0.77, 4.00)	0.42 (0.05, 3.27)	1.58 (0.47, 5.31)	5.26 (1.53, 18.09)
Neighborhood Proportion Non-White, 1990	0.94 (0.60, 1.48)	1.52 (0.49, 4.76)	0.97 (0.41, 2.30)	0.91 (0.72, 1.14)
Age at Wave I				
10-13 years old	0.94 (0.81, 1.09)	1.08 (0.72, 1.64)	0.97 (0.81, 1.16)	0.72 (0.54, 0.94)
14-15 years old	1.02 (0.92, 1.14)	0.97 (0.71, 1.32)	1.05 (0.91, 1.21)	0.91 (0.72, 1.14)
16-19 years old	1.00	1.00	1.00	1.00
Gender				
Male	1.00	1.00	1.00	1.00
Female	1.58 (1.44, 1.73)	1.31 (1.42, 2.58)	1.55 (1.38, 1.74)	1.74 (1.33, 2.26)
Parental Education at Wave I				
<HS	1.17 (0.96, 1.41)	1.19 (0.83, 1.73)	1.34 (1.05, 1.71)	0.72 (0.52, 1.00)
HS Diploma	1.19 (1.07, 1.33)	1.37 (0.97, 1.93)	1.22 (1.07, 1.40)	0.89 (0.75, 1.06)
Some College	1.07 (0.94, 1.23)	0.99 (0.64, 1.52)	1.08 (0.92, 1.27)	1.04 (0.81, 1.33)
College+	1.00	1.00	1.00	1.00

Physical Activity at Wave I				
<5 times/week	1.05 (0.95, 1.17)	0.79 (0.59, 1.07)	1.11 (0.96, 1.27)	0.97 (0.80, 1.18)
5+ times/week	1.00	1.00	1.00	1.00

**Predicted Prevalence (95% CIs).*

A2.9. Unadjusted Prevalence Ratios for Inflammation in Early Adulthood by School Entropy (Multi-Group, Standardized) during Adolescence, Add Health Waves I (1994-5) and IV (2008).

	Total Population	Hispanic	Non-Hispanic White	Non-Hispanic Black
School Entropy (Multi-Group)	0.96 (0.76, 1.21)	1.61 (0.72, 3.60)	0.92 (0.66, 1.29)	0.92 (0.49, 1.74)
0.10*	0.32 (0.30, 0.34)	0.30 (0.21, 0.40)	0.32 (0.30, 0.34)	0.36 (0.31, 0.41)
0.25*	0.32 (0.30, 0.33)	0.31 (0.24, 0.39)	0.31 (0.29, 0.33)	0.36 (0.31, 0.40)
0.50*	0.31 (0.30, 0.33)	0.34 (0.30, 0.38)	0.31 (0.28, 0.34)	0.35 (0.31, 0.40)
0.75*	0.31 (0.29, 0.34)	0.36 (0.32, 0.41)	0.30 (0.26, 0.35)	0.35 (0.28, 0.42)
0.90*	0.31 (0.28, 0.34)	0.38 (0.32, 0.45)	0.30 (0.25, 0.36)	0.34 (0.26, 0.44)

**Predicted Prevalence (95% CIs).*

A2.10. Adjusted Prevalence Ratios for Inflammation in Early Adulthood by School Entropy (Multi-Group, Standardized) during Adolescence, Add Health Waves I (1994-5) and IV (2008).

	Total Population	Hispanic	Non-Hispanic White	Non-Hispanic Black
School Entropy (Multi-Group)	1.23 (0.92, 1.63)	1.68 (0.78, 3.62)	1.17 (0.79, 1.74)	1.51 (0.56, 4.06)
0.10*	0.31 (0.29, 0.33)	0.29 (0.21, 0.39)	0.31 (0.29, 0.33)	0.33 (0.28, 0.39)
0.25*	0.31 (0.30, 0.33)	0.31 (0.24, 0.38)	0.31 (0.29, 0.33)	0.34 (0.30, 0.38)
0.50*	0.33 (0.31, 0.35)	0.34 (0.30, 0.38)	0.32 (0.29, 0.35)	0.37 (0.31, 0.43)
0.75*	0.34 (0.30, 0.37)	0.37 (0.33, 0.41)	0.33 (0.28, 0.38)	0.39 (0.29, 0.51)
0.90*	0.34 (0.30, 0.39)	0.39 (0.33, 0.45)	0.34 (0.28, 0.40)	0.41 (0.27, 0.55)
School Size				
1 – 400 students	1.18 (1.01, 1.37)	1.04 (0.74, 1.47)	1.18 (0.99, 1.40)	1.17 (0.80, 1.70)
401 – 1,000 students	1.01 (0.91, 1.14)	1.07 (0.85, 1.34)	0.97 (0.84, 1.11)	1.30 (0.99, 1.69)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.84 (0.73, 0.97)	0.87 (0.60, 1.26)	0.86 (0.72, 1.03)	0.87 (0.65, 1.16)
Suburban	0.91 (0.81, 1.03)	0.95 (0.68, 1.32)	0.93 (0.80, 1.08)	1.03 (0.81, 1.31)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.22 (1.04, 1.43)	1.14 (0.71, 1.84)	1.11 (0.92, 1.35)	2.06 (1.10, 3.86)
Private	1.00	1.00	1.00	1.00

**Predicted Prevalence (95% CIs).*

A2.11. Adjusted Prevalence Ratios for Inflammation in Early Adulthood by School Entropy (Multi-Group, Standardized) during Adolescence, Add Health Waves I (1994-5) and IV (2008).

	Total Population	Hispanic	Non-Hispanic White	Non-Hispanic Black
School Entropy (Multi-Group)	1.18 (0.88, 1.57)	1.04 (0.43, 2.52)	1.10 (0.69, 1.74)	1.50 (0.54, 4.20)
0.10*	0.31 (0.29, 0.33)	0.34 (0.24, 0.46)	0.31 (0.29, 0.33)	0.33 (0.27, 0.40)
0.25*	0.31 (0.30, 0.33)	0.34 (0.27, 0.43)	0.31 (0.29, 0.33)	0.35 (0.30, 0.39)
0.50*	0.32 (0.30, 0.34)	0.34 (0.30, 0.39)	0.32 (0.28, 0.35)	0.37 (0.31, 0.43)
0.75*	0.33 (0.30, 0.37)	0.35 (0.31, 0.39)	0.32 (0.27, 0.38)	0.39 (0.29, 0.51)
0.90*	0.34 (0.30, 0.38)	0.35 (0.29, 0.41)	0.32 (0.26, 0.40)	0.41 (0.27, 0.56)
School Size				
1 – 400 students	1.13 (0.97, 1.32)	1.02 (0.72, 1.45)	1.13 (0.95, 1.35)	1.11 (0.75, 1.65)
401 – 1,000 students	1.00 (0.89, 1.12)	1.11 (0.89, 1.39)	0.95 (0.82, 1.09)	1.34 (1.03, 1.73)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.83 (0.73, 0.95)	0.85 (0.59, 1.23)	0.87 (0.72, 1.04)	0.90 (0.68, 1.20)
Suburban	0.93 (0.82, 1.04)	0.91 (0.65, 1.27)	0.95 (0.82, 1.10)	1.07 (0.85, 1.33)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.18 (1.01, 1.37)	1.23 (0.72, 2.09)	1.08 (0.90, 1.30)	1.87 (0.99, 3.56)
Private	1.00	1.00	1.00	1.00
Neighborhood Poverty, 1990	1.96 (0.96, 3.99)	0.37 (0.08, 1.79)	2.10 (0.69, 6.41)	2.95 (1.04, 8.41)
Neighborhood Proportion Non-White, 1990	1.02 (0.75, 1.37)	2.76 (1.22, 6.22)	1.04 (0.47, 2.26)	0.55 (0.32, 0.94)

**Predicted Prevalence (95% CIs).*

A2.12. Adjusted Prevalence Ratios for Inflammation in Early Adulthood by School Entropy (Multi-Group, Standardized) during Adolescence, Add Health Waves I (1994-5) and IV (2008).

	Total Population	Hispanic	Non-Hispanic White	Non-Hispanic Black
School Entropy (Multi-Group)	1.14 (0.85, 1.54)	0.95 (0.36, 2.50)	1.06 (0.65, 1.73)	1.67 (0.76, 3.65)
0.10*	0.31 (0.29, 0.33)	0.33 (0.23, 0.45)	0.31 (0.29, 0.33)	0.32 (0.27, 0.38)
0.25*	0.31 (0.30, 0.33)	0.33 (0.25, 0.42)	0.31 (0.29, 0.33)	0.34 (0.29, 0.39)
0.50*	0.32 (0.30, 0.34)	0.33 (0.28, 0.37)	0.32 (0.28, 0.35)	0.37 (0.32, 0.42)
0.75*	0.33 (0.29, 0.36)	0.32 (0.28, 0.37)	0.32 (0.26, 0.38)	0.39 (0.32, 0.48)
0.90*	0.33 (0.29, 0.37)	0.32 (0.26, 0.39)	0.32 (0.25, 0.40)	0.41 (0.31, 0.52)
School Size				
1 – 400 students	1.15 (0.98, 1.37)	1.07 (0.72, 1.57)	1.10 (0.90, 1.35)	1.32 (0.94, 1.86)
401 – 1,000 students	1.04 (0.92, 1.18)	1.05 (0.73, 1.49)	0.96 (0.83, 1.12)	1.77 (1.33, 2.35)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.83 (0.71, 0.97)	0.82 (0.50, 1.34)	0.89 (0.73, 1.09)	0.91 (0.62, 1.32)
Suburban	0.92 (0.80, 1.04)	1.03 (0.65, 1.63)	0.95 (0.81, 1.11)	1.00 (0.76, 1.32)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.04 (0.86, 1.27)	1.17 (0.65, 2.10)	0.97 (0.78, 1.20)	2.13 (1.01, 4.51)
Private	1.00	1.00	1.00	1.00
Neighborhood Poverty, 1990	1.78 (0.79, 4.00)	0.44 (0.06, 3.42)	1.55 (0.46, 5.24)	6.75 (2.15, 21.13)
Neighborhood Proportion Non-White, 1990	0.98 (0.69, 1.39)	2.31 (0.36, 2.50)	1.05 (0.45, 2.43)	0.46 (0.26, 3.65)
Age at Wave I				
10-13 years old	0.94 (0.81, 1.09)	1.10 (0.73, 1.67)	0.97 (0.81, 1.16)	0.70 (0.53, 0.93)
14-15 years old	1.02 (0.92, 1.14)	0.97 (0.71, 1.33)	1.05 (0.91, 1.21)	0.89 (0.71, 1.12)
16-19 years old	1.00	1.00	1.00	1.00
Gender				
Male	1.00	1.00	1.00	1.00
Female	1.58 (1.44, 1.73)	1.91 (1.42, 2.57)	1.55 (1.38, 1.74)	1.74 (1.34, 2.27)
Parental Education at Wave I				
<HS	1.16 (0.96, 1.41)	1.19 (0.82, 1.72)	1.34 (1.05, 1.71)	0.72 (0.52, 0.99)
HS Diploma	1.19 (1.07, 1.33)	1.35 (0.95, 1.91)	1.22 (1.07, 1.40)	0.89 (0.74, 1.06)
Some College	1.07 (0.93, 1.23)	0.99 (0.65, 1.53)	1.08 (0.92, 1.27)	1.04 (0.82, 1.33)

College+	1.00	1.00	1.00	1.00
Physical Activity at Wave I				
<5 times/week	1.05 (0.95, 1.17)	0.79 (0.58, 1.07)	1.11 (0.96, 1.27)	0.96 (0.79, 1.17)
5+ times/week	1.00	1.00	1.00	1.00

**Predicted Prevalence (95% CIs).*

A2.13. Unadjusted Prevalence Ratios for Inflammation in Early Adulthood by School Segregation – Black-White Dissimilarity Index – in a School during Adolescence, Add Health Waves I (1994-5) and IV (2008).

	Total Population	Hispanic	Non-Hispanic White	Non-Hispanic Black
Dissimilarity Index	0.93 (0.68, 1.26)	0.71 (0.38, 1.31)	1.19 (0.72, 1.96)	0.56 (0.29, 1.10)
0.05*	0.32 (0.30, 0.35)	0.38 (0.32, 0.45)	0.31 (0.28, 0.34)	0.39 (0.33, 0.45)
0.20*	0.32 (0.31, 0.34)	0.37 (0.32, 0.41)	0.31 (0.29, 0.33)	0.37 (0.33, 0.41)
0.40*	0.32 (0.30, 0.33)	0.35 (0.32, 0.38)	0.32 (0.30, 0.34)	0.34 (0.31, 0.38)
0.60*	0.31 (0.29, 0.34)	0.33 (0.30, 0.37)	0.33 (0.29, 0.37)	0.32 (0.27, 0.37)
0.80*	0.31 (0.28, 0.35)	0.32 (0.27, 0.38)	0.33 (0.28, 0.40)	0.29 (0.23, 0.37)

**Predicted Prevalence (95% CIs).*

A2.14. Adjusted Prevalence Ratios for Inflammation in Early Adulthood by School Segregation – Black-White Dissimilarity Index – in a School during Adolescence, Add Health Waves I (1994-5) and IV (2008).

	Total Population	Hispanic	Non-Hispanic White	Non-Hispanic Black
Dissimilarity Index	1.06 (0.75, 1.51)	1.27 (0.65, 2.50)	1.02 (0.55, 1.88)	0.80 (0.40, 1.60)
0.05*	0.31 (0.29, 0.34)	0.33 (0.27, 0.39)	0.31 (0.28, 0.34)	0.37 (0.30, 0.44)
0.20*	0.31 (0.30, 0.33)	0.33 (0.29, 0.38)	0.31 (0.29, 0.33)	0.36 (0.31, 0.42)
0.40*	0.32 (0.30, 0.34)	0.35 (0.31, 0.38)	0.31 (0.29, 0.34)	0.35 (0.32, 0.39)
0.60*	0.32 (0.29, 0.35)	0.36 (0.31, 0.41)	0.31 (0.27, 0.36)	0.34 (0.30, 0.39)
0.80*	0.32 (0.28, 0.37)	0.37 (0.30, 0.45)	0.31 (0.24, 0.39)	0.33 (0.27, 0.41)
School Size				
1 – 400 students	1.14 (0.98, 1.32)	1.10 (0.77, 1.56)	1.13 (0.95, 1.35)	1.08 (0.76, 1.52)
401 – 1,000 students	1.02 (0.91, 1.13)	1.10 (0.88, 1.38)	0.97 (0.82, 1.11)	1.24 (0.97, 1.58)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.88 (0.77, 1.00)	0.85 (0.55, 1.29)	0.89 (0.76, 1.05)	0.91 (0.69, 1.22)
Suburban	0.92 (0.81, 1.04)	0.91 (0.62, 1.35)	0.94 (0.81, 1.09)	1.05 (0.80, 1.36)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.24 (1.07, 1.44)	1.16 (0.65, 2.09)	1.13 (0.95, 1.36)	1.91 (1.01, 3.61)
Private	1.00	1.00	1.00	1.00

**Predicted Prevalence (95% CIs).*

A2.15. Adjusted Prevalence Ratios for Inflammation in Early Adulthood by School Segregation – Black-White Dissimilarity Index – in a School during Adolescence, Add Health Waves I (1994-5) and IV (2008).

	Total Population	Hispanic	Non-Hispanic White	Non-Hispanic Black
Dissimilarity Index	0.96 (0.68, 1.35)	1.27 (0.63, 2.54)	0.96 (0.53, 1.73)	1.21 (0.55, 2.66)
0.05*	0.32 (0.30, 0.34)	0.33 (0.27, 0.39)	0.31 (0.28, 0.35)	0.34 (0.28, 0.41)
0.20*	0.32 (0.30, 0.33)	0.34 (0.29, 0.39)	0.31 (0.29, 0.33)	0.35 (0.30, 0.40)
0.40*	0.32 (0.30, 0.33)	0.35 (0.31, 0.38)	0.31 (0.28, 0.34)	0.36 (0.32, 0.40)
0.60*	0.31 (0.29, 0.34)	0.36 (0.31, 0.41)	0.31 (0.26, 0.36)	0.36 (0.31, 0.42)
0.80*	0.31 (0.27, 0.35)	0.37 (0.30, 0.44)	0.31 (0.24, 0.38)	0.37 (0.29, 0.46)
School Size				
1 – 400 students	1.08 (0.93, 1.25)	1.08 (0.77, 1.51)	1.06 (0.89, 1.25)	1.07 (0.74, 1.55)
401 – 1,000 students	0.99 (0.89, 1.10)	1.11 (0.89, 1.39)	0.94 (0.82, 1.08)	1.33 (1.04, 1.71)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.87 (0.77, 0.98)	0.78 (0.49, 1.25)	0.89 (0.75, 1.05)	0.92 (0.70, 1.21)
Suburban	0.93 (0.83, 1.05)	0.83 (0.53, 1.30)	0.96 (0.82, 1.11)	1.05 (0.83, 1.33)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.18 (1.02, 1.37)	1.31 (0.69, 2.51)	1.08 (0.90, 1.29)	1.82 (0.98, 3.37)
Private	1.00	1.00	1.00	1.00
Neighborhood Poverty, 1990	1.95 (1.01, 3.77)	0.37 (0.08, 1.67)	2.72 (0.88, 8.42)	3.18 (1.14, 8.91)
Neighborhood Proportion Black, 1990	1.08 (0.82, 1.42)	4.68 (1.48, 14.84)	1.02 (0.46, 2.28)	0.46 (0.26, 0.81)

**Predicted Prevalence (95% CIs).*

A2.16. Adjusted Prevalence Ratios for Inflammation in Early Adulthood by School Segregation – Black-White Dissimilarity Index – in a School during Adolescence, Add Health Waves I (1994-5) and IV (2008).

	Total Population	Hispanic	Non-Hispanic White	Non-Hispanic Black
Dissimilarity Index	0.93 (0.60, 1.45)	2.17 (0.75, 6.31)	1.00 (0.49, 2.05)	1.05 (0.43, 2.55)
0.05*	0.32 (0.29, 0.34)	0.28 (0.21, 0.36)	0.31 (0.28, 0.35)	0.35 (0.28, 0.42)
0.20*	0.31 (0.30, 0.33)	0.30 (0.25, 0.36)	0.31 (0.29, 0.33)	0.35 (0.30, 0.40)
0.40*	0.31 (0.29, 0.33)	0.33 (0.29, 0.38)	0.31 (0.28, 0.34)	0.35 (0.31, 0.39)
0.60*	0.31 (0.28, 0.34)	0.36 (0.30, 0.43)	0.31 (0.26, 0.37)	0.35 (0.30, 0.41)
0.80*	0.31 (0.26, 0.36)	0.39 (0.29, 0.51)	0.31 (0.23, 0.40)	0.35 (0.27, 0.45)
School Size				
1 – 400 students	1.11 (0.94, 1.31)	1.19 (0.85, 1.66)	1.04 (0.86, 1.27)	1.24 (0.92, 1.67)
401 – 1,000 students	1.04 (0.92, 1.17)	1.07 (0.76, 1.52)	0.96 (0.83, 1.11)	1.73 (1.30, 2.31)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.85 (0.73, 0.99)	0.68 (0.37, 1.25)	0.91 (0.76, 1.09)	0.94 (0.63, 1.38)
Suburban	0.91 (0.80, 1.04)	0.88 (0.51, 1.53)	0.95 (0.80, 1.12)	0.99 (0.74, 1.33)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.05 (0.87, 1.27)	1.24 (0.67, 2.29)	0.96 (0.78, 1.19)	2.05 (0.96, 4.37)
Private	1.00	1.00	1.00	1.00
Neighborhood Poverty, 1990	1.62 (0.75, 3.51)	0.25 (0.03, 1.75)	1.89 (0.55, 6.53)	5.89 (1.62, 21.43)
Neighborhood Proportion Black, 1990	1.12 (0.79, 1.58)	7.02 (1.62, 30.45)	1.09 (0.45, 2.63)	0.44 (0.23, 0.83)
Age at Wave I				
10-13 years old	0.93 (0.80, 1.08)	1.11 (0.75, 1.65)	0.95 (0.79, 1.14)	0.74 (0.56, 0.97)
14-15 years old	1.01 (0.91, 1.13)	0.96 (0.71, 1.30)	1.04 (0.90, 1.20)	0.91 (0.72, 1.14)
16-19 years old	1.00	1.00	1.00	1.00
Gender				
Male	1.00	1.00	1.00	1.00
Female	1.57 (1.44, 1.73)	1.98 (1.48, 2.65)	1.55 (1.38, 1.73)	1.73 (1.33, 2.24)
Parental Education at Wave I				
<HS	1.20 (0.99, 1.45)	1.24 (0.86, 1.79)	1.35 (1.06, 1.73)	0.79 (0.57, 1.10)
HS Diploma	1.20 (1.07, 1.35)	1.38 (0.97, 1.96)	1.24 (1.08, 1.42)	0.90 (0.75, 1.07)
Some College	1.09 (0.95, 1.25)	0.96 (0.61, 1.52)	1.10 (0.93, 1.30)	1.05 (0.82, 1.35)
College+	1.00	1.00	1.00	1.00

Physical Activity at Wave I				
<5 times/week	1.03 (0.92, 1.14)	0.77 (0.57, 1.03)	1.07 (0.93, 1.23)	0.97 (0.80, 1.19)
5+ times/week	1.00	1.00	1.00	1.00

**Predicted Prevalence (95% CIs).*

A2.17. Unadjusted Prevalence Ratios for Inflammation in Early Adulthood by School Segregation – Black-White Exposure Index – in a School during Adolescence, Add Health Waves I (1994-5) and IV (2008).

	Total Population	Hispanic	Non-Hispanic White	Non-Hispanic Black
Exposure Index	1.08 (0.90, 1.31)	1.03 (0.70, 1.51)	1.17 (0.89, 1.54)	1.94 (1.07, 3.51)
0.10*	0.31 (0.29, 0.33)	0.35 (0.30, 0.39)	0.29 (0.26, 0.33)	0.30 (0.25, 0.36)
0.25*	0.31 (0.29, 0.33)	0.35 (0.31, 0.39)	0.30 (0.27, 0.33)	0.32 (0.29, 0.36)
0.50*	0.32 (0.30, 0.33)	0.35 (0.32, 0.38)	0.31 (0.29, 0.33)	0.36 (0.33, 0.40)
0.75*	0.32 (0.31, 0.34)	0.35 (0.31, 0.39)	0.32 (0.30, 0.33)	0.40 (0.34, 0.46)
0.90*	0.32 (0.30, 0.34)	0.35 (0.31, 0.40)	0.32 (0.30, 0.34)	0.42 (0.35, 0.50)

**Predicted Prevalence (95% CIs).*

A2.18. Adjusted Prevalence Ratios for Inflammation in Early Adulthood by School Segregation – Black-White Exposure Index – in a School during Adolescence, Add Health Waves I (1994-5) and IV (2008).

	Total Population	Hispanic	Non-Hispanic White	Non-Hispanic Black
Exposure Index	0.94 (0.76, 1.17)	0.72 (0.45, 1.15)	1.02 (0.74, 1.39)	2.19 (1.16, 4.16)
0.10*	0.32 (0.29, 0.35)	0.38 (0.32, 0.44)	0.31 (0.26, 0.36)	0.30 (0.26, 0.36)
0.25*	0.32 (0.30, 0.35)	0.37 (0.32, 0.42)	0.31 (0.27, 0.35)	0.33 (0.29, 0.37)
0.50*	0.32 (0.30, 0.33)	0.35 (0.31, 0.39)	0.31 (0.29, 0.34)	0.37 (0.33, 0.42)
0.75*	0.31 (0.30, 0.33)	0.33 (0.29, 0.37)	0.31 (0.29, 0.33)	0.42 (0.35, 0.49)
0.90*	0.31 (0.29, 0.33)	0.32 (0.27, 0.37)	0.31 (0.29, 0.33)	0.45 (0.36, 0.54)
School Size				
1 – 400 students	1.13 (0.98, 1.31)	1.09 (0.76, 1.56)	1.13 (0.96, 1.34)	1.07 (0.76, 1.51)
401 – 1,000 students	1.01 (0.91, 1.13)	1.12 (0.89, 1.41)	0.97 (0.85, 1.11)	1.29 (1.02, 1.63)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.88 (0.77, 0.99)	0.84 (0.56, 1.25)	0.90 (0.77, 1.05)	0.89 (0.68, 1.15)
Suburban	0.92 (0.82, 1.04)	0.94 (0.65, 1.36)	0.94 (0.81, 1.09)	1.02 (0.82, 1.27)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.24 (1.07, 1.44)	1.20 (0.68, 2.14)	1.13 (0.95, 1.36)	1.81 (1.01, 3.24)
Private	1.00	1.00	1.00	1.00

**Predicted Prevalence (95% CIs).*

A2.19. Adjusted Prevalence Ratios for Inflammation in Early Adulthood by School Segregation –Black-White Exposure Index – in a School during Adolescence, Add Health Waves I (1994-5) and IV (2008).

	Total Population	Hispanic	Non-Hispanic White	Non-Hispanic Black
Exposure Index	1.08 (0.85, 1.39)	0.80 (0.49, 1.32)	1.08 (0.78, 1.50)	1.89 (0.95, 3.78)
0.10*	0.31 (0.27, 0.34)	0.37 (0.31, 0.43)	0.30 (0.26, 0.35)	0.31 (0.26, 0.38)
0.25*	0.31 (0.28, 0.34)	0.36 (0.32, 0.41)	0.30 (0.27, 0.34)	0.33 (0.29, 0.38)
0.50*	0.31 (0.30, 0.33)	0.35 (0.31, 0.39)	0.31 (0.28, 0.33)	0.37 (0.33, 0.41)
0.75*	0.32 (0.30, 0.33)	0.34 (0.29, 0.38)	0.31 (0.29, 0.33)	0.41 (0.35, 0.47)
0.90*	0.32 (0.30, 0.34)	0.33 (0.27, 0.39)	0.31 (0.29, 0.34)	0.43 (0.35, 0.42)
School Size				
1 – 400 students	1.09 (0.94, 1.25)	1.07 (0.76, 1.51)	1.06 (0.90, 1.25)	1.03 (0.72, 1.47)
401 – 1,000 students	0.99 (0.90, 1.10)	1.12 (0.89, 1.40)	0.94 (0.82, 1.08)	1.31 (1.03, 1.67)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.87 (0.77, 0.98)	0.79 (0.51, 1.22)	0.89 (0.76, 1.05)	0.91 (0.69, 1.18)
Suburban	0.93 (0.83, 1.05)	0.84 (0.5, 1.30)	0.96 (0.82, 1.11)	1.04 (0.83, 1.31)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.18 (1.02, 1.37)	1.34 (0.70, 2.57)	1.08 (0.90, 1.29)	1.72 (0.95, 3.13)
Private	1.00	1.00	1.00	1.00
Neighborhood Poverty, 1990	1.96 (1.01, 3.82)	0.35 (0.07, 1.76)	2.69 (0.86, 8.42)	2.42 (0.87, 6.72)
Neighborhood Proportion Black, 1990	1.13 (0.83, 1.53)	4.40 (1.42, 13.66)	1.09 (0.48, 2.50)	0.71 (0.42, 1.20)

**Predicted Prevalence (95% CIs).*

A2.20. Adjusted Prevalence Ratios for Inflammation in Early Adulthood by School Segregation – Black-White Exposure Index – in a School during Adolescence, Add Health Waves I (1994-5) and IV (2008).

	Total Population	Hispanic	Non-Hispanic White	Non-Hispanic Black
Exposure Index	1.03 (0.77, 1.39)	0.54 (0.24, 1.24)	0.98 (0.68, 1.42)	1.74 (0.82, 3.71)
0.10*	0.31 (0.27, 0.35)	0.38 (0.30, 0.48)	0.31 (0.26, 0.37)	0.32 (0.26, 0.38)
0.25*	0.31 (0.28, 0.34)	0.36 (0.30, 0.44)	0.31 (0.27, 0.36)	0.33 (0.29, 0.38)
0.50*	0.31 (0.29, 0.33)	0.33 (0.29, 0.38)	0.31 (0.28, 0.34)	0.36 (0.32, 0.41)
0.75*	0.31 (0.30, 0.33)	0.30 (0.25, 0.36)	0.31 (0.29, 0.33)	0.39 (0.33, 0.46)
0.90*	0.31 (0.29, 0.34)	0.29 (0.23, 0.36)	0.31 (0.29, 0.34)	0.41 (0.33, 0.50)
School Size				
1 – 400 students	1.11 (0.95, 1.31)	1.14 (0.81, 1.61)	1.04 (0.86, 1.25)	1.19 (0.89, 1.61)
401 – 1,000 students	1.04 (0.93, 1.18)	1.07 (0.75, 1.52)	0.96 (0.83, 1.11)	1.70 (1.30, 2.23)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.85 (0.74, 0.98)	0.71 (0.40, 1.23)	0.91 (0.76, 1.08)	0.92 (0.64, 1.32)
Suburban	0.91 (0.80, 1.04)	0.92 (0.54, 1.55)	0.95 (0.80, 1.12)	0.98 (0.75, 1.28)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.05 (0.86, 1.27)	1.29 (0.69, 2.41)	0.96 (0.78, 1.19)	1.96 (0.94, 4.09)
Private	1.00	1.00	1.00	1.00
Neighborhood Poverty, 1990	1.60 (0.73, 3.51)	0.22 (0.02, 1.88)	1.89 (0.55, 6.51)	4.77 (1.47, 15.46)
Neighborhood Proportion Black, 1990	1.13 (0.77, 1.66)	5.84 (1.40, 24.30)	1.08 (0.44, 2.67)	0.61 (0.35, 1.05)
Age at Wave I				
10-13 years old	0.93 (0.80, 1.08)	1.11 (0.75, 1.63)	0.95 (0.79, 1.14)	0.75 (0.57, 0.99)
14-15 years old	1.01 (0.91, 1.13)	0.95 (0.70, 1.29)	1.04 (0.90, 1.20)	0.92 (0.73, 1.16)
16-19 years old	1.00	1.00	1.00	1.00
Gender				
Male	1.00	1.00	1.00	1.00
Female	1.57 (1.44, 1.73)	1.98 (1.47, 2.68)	1.55 (1.38, 1.73)	1.7 1(1.31, 2.23)
Parental Education at Wave I				
<HS	1.20 (0.99, 1.45)	1.22 (0.85, 1.76)	1.35 (1.06, 1.73)	0.78 (0.56, 1.09)
HS Diploma	1.20 (1.07, 1.35)	1.39 (0.99, 1.95)	1.24 (1.08, 1.42)	0.89 (0.75, 1.06)
Some College	1.09 (0.95, 1.25)	0.94 (0.60, 1.47)	1.10 (0.94, 1.30)	1.04 (0.82, 1.34)
College+	1.00	1.00	1.00	1.00

Physical Activity at Wave I				
<5 times/week	1.03 (0.92, 1.14)	0.77 (0.57, 1.03)	1.07 (0.93, 1.23)	0.98 (0.80, 1.18)
5+ times/week	1.00	1.00	1.00	1.00

**Predicted Prevalence (95% CIs).*

Not So Black and White: The Racial/Ethnic Structure of School Environments and Cardiometabolic and Reproductive Health across the Life Course

Chapter 4: Experiences of Preterm Birth

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SPECIFIC AIM 3

Investigate the multilevel associations between the racial/ethnic structure of school environments experienced in adolescence and experiences of preterm birth (PTB).

Hypothesis 3a: Female adolescents who attend schools with a higher proportion of Black students, higher proportion of non-White students, or with less racial/ethnic diversity will have a higher prevalence of PTB, after adjusting for individual-, school-, and neighborhood-level characteristics. These associations will differ by an adolescent's race/ethnicity.

Hypothesis 3b: Female adolescents who attend schools within districts that have higher levels of racial/ethnic segregation will have a higher prevalence of PTB, after adjusting for individual-, school-, and neighborhood-level characteristics. These associations will differ by an adolescent's race/ethnicity.

ABSTRACT

Since the causes of the persistent racial/ethnic and socioeconomic disparities in preterm birth (PTB) are still not fully understood, it is necessary to investigate the potential adverse consequence of the racial/ethnic structure of school environments on pregnancy outcomes. This study will examine the racial/ethnic differences in the associations between school racial/ethnic composition, diversity, and district-level segregation and experiences of PTB. Our study population included females who participated in both Waves I (1994-5) and V (2016-18) of the National Longitudinal Adolescent to Adult Health Study, and who had a singleton, first birth by Wave V (N=4,380). Preterm births were defined as any birth that occurred prior to thirty-seven gestational weeks. The predicted marginal prevalence of PTB, by school racial/ethnic composition (i.e., proportion Black and non-White students), school racial/ethnic diversity (i.e., school entropy), and district-level segregation (i.e., Black-White dissimilarity and exposure indices) were estimated from cluster- and sample-weight adjusted logistic regression models in SUDAAN. Models stratified by race/ethnicity were adjusted for school-level (size, type, urbanicity), neighborhood-level (poverty, racial composition), and individual-level (age, gender, parent's education, physical activity) characteristics. Hispanic females were 95% less likely (95% CI: 0.00, 0.56) to experience a PTB as the proportion of Black students in their schools increased from 0 to 100%. There were no meaningful associations between the racial/ethnic structure of school environments and experiences of PTB for non-Hispanic White and Black female participants.

SIGNIFICANCE

Preterm births (PTBs) are defined as an infant born less than thirty-seven gestational weeks[1]. The prevalence of PTBs in the United States (U.S.) has decreased in the last decade due to the reduction of population-wide risk factors (e.g., smoking, etc.) and the development of important medical interventions and policies[2]. However, approximately 11.4% of U.S. infants were still born preterm in 2013[3], which is relatively high compared to other developed nations[1,4]. Though preterm delivery is often thought to be a result of infection or inflammation, hemorrhage, and maternal stress[1,5]; other early-life socio-contextual factors can also contribute to the risk of PTB during the reproductive years.

Increased levels of stress and lower levels of socioeconomic status (SES) during childhood and adolescence are potentially associated with the risk of PTB, through the physiological effects of inflammation. Socioeconomic disadvantage in childhood can have adverse effects on pregnancy outcomes, suggesting that contextual factors in early life may have greater consequences on the reproductive health of women across their life course than social disadvantage experienced during pregnancy[6-8]. Furthermore, women who experience chronic stressors across the life course may not be able to regulate their inflammatory and endocrine responses when confronted with a prenatal stress. This dysregulation, due to psychological and social stress, can result in increased levels of corticotropin-releasing hormone produced by the placenta, increasing the risk of preterm delivery[6,9-11].

Geronimus' "weathering" hypothesis also suggests that Black women have worse birth outcomes than White women due to the declines in reproductive potential that results from social, economic, and political disadvantages and stressors experienced

repeatedly across the life course[12]. In fact, Black infants are more likely to be born preterm than White infants[1,3,6,13-14]. There are also established socioeconomic disparities in PTB[7-8,15-17]. These socioeconomic differences may vary by maternal race/ethnicity, in that having a higher SES may be more beneficial for the reproductive health of White women than Black women[7,17]. It is possible that the pathways for PTB develop during childhood and are exacerbated by stress and inflammation experienced early in life. Given the racial/ethnic and socioeconomic disparities, it is important to investigate the socio-contextual risk factors for PTB that occur during childhood or adolescence.

The racial/ethnic structure of school environments and systems experienced during early life could be a risk factor for PTB during the reproductive years. In another study using Add Health data, researchers found that Black students who attended White schools were more likely to have worse health outcomes in early adulthood[18]. However, there are no recent studies that have investigated the impact of both school racial composition and district-level segregation on pregnancy outcomes, such as PTB. In order to fully understand the relationship between the racial/ethnic structure of school environments and experiences of PTB, one must consider the historical context and evolution of school segregation in the U.S. Public schools in the U.S. were segregated based on race due to the legal guidelines set out by *Plessy v. Ferguson* (1896). This Supreme Court decision resulted in unequal educational opportunities for Black students in this country[19-21]. *Brown v. Board of Education of Topeka* (1954) dismantled the "separate, but equal" doctrine from *Plessy* and southern U.S. schools were under mandates to desegregate[22]. However, over the coming years, residential segregation persisted and other Supreme Court decisions relinquished school systems from their

court-ordered mandates[23-26], ultimately resulting in the resegregation of U.S. public schools beginning in the late 1970s[21,27-29]. Furthermore, the 1980s and 1990s saw significant decreases in the White student enrollment in urban schools, as well as increases in the minority student enrollment[27]. Given this unique historical context and the racial demographics of U.S. schools in the 1990s, the racial/ethnic structure of school environments is a plausible social determinant of health that is relevant for contemporary students.

According to ecosocial theory, humans can biologically embody the social, economic, and political aspects of the environments in which they live and interact[30]. Therefore, it is possible that mothers and children can internalize the adverse health consequences of contextual factors, such as school segregation, over time. Given that adolescents spend a considerable amount of time in school, it is necessary to examine the health consequences of attending racially-homogenous or segregated schools during adolescence. Having lower levels of maternal childhood SES is associated with an increased likelihood of low birth weight among Add Health participants[31]. However, little is known about the consequences of school environments on a mother's risk of PTB.

In addition, the Accumulation of Risks Model suggests that the adverse health effects of harmful socio-contextual exposures experienced in early life, particularly adolescence can accumulate over time[32]. It is possible that the stress that Black and Hispanic students experience in predominantly white schools, due to racial/ethnic discrimination from their teachers and/or peers, can accumulate over time. It is also plausible that Black and Hispanic students attending schools in racially-segregated districts may have less access to critical health-relevant, social and economic resources.

These psychosocial and socioeconomic stressors arising from attending racially homogenous and segregated schools could have negative consequences for one's sympathetic nervous system and hypothalamic-pituitary-adrenal axis. In fact, it has been established that repeated stress, especially during the critical period of adolescence, is associated with increased endocrine levels and the disruptions of inflammatory processes[33-35]. Thus, inadequate educational opportunities experienced in racially homogenous or segregated schools could result in health inequities across the life course and even from mother to child.

To our knowledge, this is the first study that has investigated the impact of school racial/ethnic composition and segregation on experiences of PTB. Given the relevance of this socio-contextual exposure, especially since U.S. schools have resegregated in recent years, it is important to understand whether the racial/ethnic structure of school environments can have adverse effects on pregnancy outcomes. Therefore, this seeks to examine whether the associations between school racial/ethnic composition, diversity, and district-level segregation and experiences of PTB among the participants in Add Health differ by individual race/ethnicity.

METHODS

Study Design and Population

We used data from Waves I (1994-1995) and Wave V (2016-2018) of the National Longitudinal Study of Adolescent to Adult Health (Add Health). Beginning in 1994, Add Health is an ongoing, longitudinal study that follows adolescents in grades 7-12 into adulthood. This study utilized a complex sampling design and stratification method in order to derive a nationally representative sample of U.S. high schools in Wave I (1994-1995). Using a sampling frame based on a database from the Quality Education Data, Inc. (QED), Add Health consisted of a school-based sample of adolescents living in the U.S. Systematic sampling methods and implicit stratification were used to ensure that the schools in Add Health were representative of all U.S. schools regarding the following factors: region, urbanicity, size, type, and racial composition[36].

The high schools incorporated into the sample had to include eleventh graders and at least thirty enrolled students. Seventy-nine percent of sampled schools participated in the study, resulting in eighty high schools represented in the Add Health sample. Once high schools were sampled, feeder (i.e., middle) schools were identified to capture students in the seventh and eighth grades from the 80 communities in which the high schools were located. These feeder schools were required to include the seventh grade and had to send at least five graduates to one of the eighty high schools in the Add Health sample. Altogether, there were 132 U.S. schools included in the Add Health Wave I sample: eighty high schools and fifty-two feeder schools. There was not a one to one ratio of high schools to feeder schools because some high schools included

students from grades seven through twelve, and thus, separate feeder schools were not recruited for these high schools[36].

Given that this was a school-based sample, both in-school questionnaires and in-home interviews were administered during Wave I. From the sampled schools, there were 90,118 students in grades seven through twelve who participated in the in-school questionnaires during Wave I. Of these students, 20,745 adolescents were randomly selected, based on grade and sex, to be in the "core sample" to participate in the in-home interviews. Only those students who participated in the in-school questionnaires were eligible to be selected to participate in the in-home interviews. In addition to the random selection by grade and sex, investigators oversampled based on the following criteria: ethnicity, disability status, school saturation, and twin sets (e.g., genetic factors)[36].

Conducted between 2016 and 2018, Wave V included the collection of biological, behavioral, social, and geographic data for the living Add Health participants who were part of the "core sample" from Wave I. The 19,828 eligible Add Health participants were between 31 and 43 years old. Wave V was the first wave of Add Health to utilize a mixed-mode survey design for data collection. Add Health participants were allowed to choose between a web- or mail-based questionnaires, with in-person and/or phone interviews to follow-up with participants who did not respond. There was a 69.3% response rate in Wave V, resulting in a total sample population of 12,300 participants[37].

Experiences of Preterm Birth (PTB)

The Wave V questionnaires asked Add Health participants about their complete reproductive and fertility histories across the life course. Participants reported all relationships, pregnancies, and live births across their lifetime. We restricted our study population to only include the female participants who had a singleton, first birth by Wave V. There were 8,168 singleton, first births at Wave V. Our final analysis population included the 4,380 of these births corresponded to female participants. Self-reported experiences of preterm birth were assessed using the following question: "*A preterm delivery is one that occurs before 37 weeks in pregnancy (more than 3 weeks early). Was the baby born preterm?*" Participants responded either yes or no.

Individual Race/Ethnicity

Adolescents reported their race/ethnicity during the in-home interview of Wave I. Individual race/ethnicity consisted of the following categories: Hispanic, non-Hispanic White, non-Hispanic Black, Asian/Pacific Islander, American Indian/Alaska Native, and other. We restricted our analyses to include Hispanic, non-Hispanic White, and non-Hispanic Black participants.

Racial/Ethnic Structure of School Environments and School Districts

We evaluated two types of exposures in this study: (1) the racial/ethnic composition of the schools that the participants attended in adolescence, and (2) the average racial/ethnic segregation within the district in which their schools resided. The measures of composition were utilized to describe the racial/ethnic distribution and levels of diversity within the Add Health schools. The measures of segregation aimed to quantify the degree to which racial/ethnic groups of students are segregated within

school districts in Add Health. Thus, we utilized these two types of exposures in order to investigate the health consequences of the absolute racial/ethnic make-up of a school, as well as the spatial separation of racial/ethnic groups within a school district.

School racial/ethnic composition was assessed using three measures: (1) the proportion of the total student population within each Wave I Add Health school that was non-Hispanic Black; (2) the proportion of the total student population within each school that was non-White; and (3) the racial/ethnic entropy (i.e., diversity) within each school. School entropy, a measure of racial/ethnic diversity within a school, was calculated using the following formula:

$$E_i = \sum_{r_i=1}^{n_i} Q_{r_i} \ln(\frac{1}{Q_{r_i}})$$

n_i represents the total number of racial/ethnic groups in the ith school. r_i represents the specific racial/ethnic group in the ith school. Q_{r_i} represents the total students in the ith school made up of the specific racial/ethnic. School entropy is an index that theoretically ranges from 0 (minimum diversity) to 1 (maximum diversity), after standardization. For this multi-group measure of entropy, a value of 0 was assigned to schools where a single racial/ethnic group was present. A value of 1 was assigned to schools where all racial/ethnic groups were represented equally in the student population[27].

The average racial/ethnic segregation within a school district was evaluated using two indices: (1) the Black-White dissimilarity index, and (2) the Black-White Exposure Index. Both indices assess the extent to which Black students are segregated from White students within a school district. The Black-White dissimilarity represents the proportion of Black students that would have to change their schools in order to achieve

an even racial distribution (among Black and White students) in their respective school district. The Black-White dissimilarity index was calculated using the following formula:

$$\frac{1}{2}\sum_{i=1}^{N}(\frac{b_i}{B} - \frac{w_i}{W})$$

b_i is the number of Black students in the ith school. B represents the total Black student enrollment in the school district. w_i is the number of White students in the ith school. W represents the total White student enrollment in the school district.

The Black-White Exposure index represents the extent to which Black students are exposed to White students within their school district. This exposure index was calculated using the following formula:

$$\sum_{i=1}^{N}(\frac{w_i}{W} * \frac{b_i}{t_i})$$

t_i is the total student enrollment within the school (across all racial/ethnic groups). Ranging from 0 (lowest probability of exposure) to 1 (highest probability of exposure), both school segregation indices were previously derived and calculated by Add Health researchers, using the adolescents' Wave I school-level data and data from the Common Core of Data, National Center for Education Statistics[38].

Covariates: School

School-level covariates included: size, urbanicity, and type. Schools were divided into three categories, based on the definition of school size from the National Center for Education Statistics (NCES). These categories included: (1) *small*: 1-400 students, (2) *medium*: 401 – 1,000 students, and (3) *large*: 1,001 – 4,000 students. A school's

urbanicity was determined according to the NCES and QED classification of the geographic areas in which the schools reside. This classification included:

(b) Central city of a Consolidated Metropolitan Statistical Area (CMSA) or Metropolitan Statistical Area (MSA) with population of 250,000 or more
(b) Central city of a CMSA or MSA, but not designated as a large central city
(c) Place within the CMSA or MSA of a large central city
(d) Place within the CMSA or MSA of a mid-size central city
(e) Place not within a CMSA or MSA, but with population of 25,000 or more and defined as urban
(f) Place not within a CMSA or MSA with a population of at least 2,500, but less than 25,000
(g) Place not within a CMSA or MSA and designated as rural
(h) Place within a CMSA or MSA designated as rural

In this study, schools were categorized as: (1) *urban*: included schools located within items *(a)* and *(b)*; (2) *suburban*: included schools located within items *(c)* – *(f)*; and (3) *rural*: included schools located within items *(g)* and *(h)*. Regarding type, schools were labeled as either public or private.

Covariates: Neighborhood

At Wave III (2001-2002), neighborhood-level data was merged retrospectively using area of residence during Wave I. This neighborhood data was derived from the 1990 U.S. Census[36]. Neighborhood-level covariates included: poverty and racial composition. For neighborhood poverty, we used the proportion of residents in each adolescents' census tract who were living below the federal poverty level. The selection of the neighborhood racial composition was dependent upon the exposure of interest. For the analyses that investigated the proportion of Black students within a school and the school segregation indices, we used the proportion of residents who were non-Hispanic Black. For the analyses that investigated the proportion of non-White students

within a school and school entropy, we used the proportion of residents who were non-White.

Covariates: Individual

Individual-level covariates included maternal age at first birth and parent's educational attainment. Using self-reported data from the reproductive and fertility histories at Wave V, maternal age at first birth was organized into three categories: 14-19, 20-29, and 30-41 years old. The educational attainment levels of an adolescent's parents, which served as a proxy for adolescent SES, were self-reported by the parents during the in-home interview at Wave I. In our study, parental education was determined to be the highest educational attainment of either the mother or father. Parental education was collapsed into the following categories: less than high school, high school diploma, some college or an Associate's degree, and college degree or higher.

Statistical Analysis

Using a complete-case analysis, the predicted prevalence of PTB were estimated from cluster- and sample-weight adjusted logistic regression models in SUDAAN. School racial/ethnic composition, diversity, and district-level segregation were assessed using the continuous versions of these variables. Analyses included unadjusted models, as well as models adjusted for covariates in three stages. Stage 1 included linear and logistic regression models adjusted for school-level covariates (size, type, urbanicity) only. Stage 2 included linear and logistic regression models adjusted for school-level covariates and neighborhood-level covariates (poverty, racial/ethnic composition). Stage 3 included the fully-adjusted linear and logistic regression models, which were

adjusted for school-level covariates, neighborhood-level covariates, and individual-level covariates (age, gender, parental education, physical activity). All models were then stratified by individual race/ethnicity. Unadjusted models, as well as models from stages 1 & 2, are included in the Appendix.

RESULTS

Socio-demographic Characteristics of Schools, Neighborhoods, and Participants

Table 1 displays the characteristics of the schools attended by the female participants who experienced a first birth by Wave V of Add Health. The racial/ethnic composition, diversity, and district-level segregation of the schools attended by the female participants in this study population also varied by race/ethnicity. Non-Hispanic Black mothers attended schools during adolescents that had a higher proportion of Black students, compared to the schools attended by Hispanic and non-Hispanic White mothers. On average, Hispanic and non-Hispanic Black mothers attended schools during adolescence that had the highest proportions of non-White students (58% and 65%, respectively). Compared to non-Hispanic White and non-Hispanic Black mothers, Hispanic mothers attended schools during adolescence with the highest levels of racial/ethnic diversity.

Regarding the Black-White dissimilarity index, in schools attended by Hispanic and non-Hispanic Black mothers, 39% and 37% of Black students, respectively, would have to change schools in order to achieve an even racial distribution in the school district. Concerning the Black-White exposure index, in schools attended by non-Hispanic White mothers, Black students had a 75% probability of being exposed to a White student. In those schools attended by non-Hispanic Black mothers, Black students had a 40% probability of being exposed to a White student.

The sociodemographic characteristics of the female participants with a first birth by Wave V and the neighborhoods in which they resided during Wave I of Add Health are described in Table 2. Hispanic and non-Hispanic Black mothers were most likely to

experience their first birth between 14 and 19 years old, while non-Hispanic White mothers were most likely to experience their first birth between 30 and 41 years old. Hispanic and non-Hispanic Black mothers were also more likely to have a lower adolescent SES, as measured by their parents' educational attainment in adolescence, compared to that of non-Hispanic White mothers. Also, the prevalence of preterm births ranged form 12.5% among non-Hispanic White mothers to almost 16% among non-Hispanic Black mothers.

Proportion Black Students and Experiences of PTB

Table 3 presents the adjusted prevalence ratios for experiences of PTB by measures of school racial/ethnic composition and segregation, stratified by individual race/ethnicity. Hispanic mothers were 0.05 times (95% CI: 0.00, 0.56) as likely to experience a PTB for every one-unit change in the proportion of Black students within a school – from 0 to 100%. In fact, Figure 1a demonstrates that the predicted prevalence of PTB was 16% among Hispanic mothers who attended schools during adolescence with 10% Black students, while the predicted prevalence of PTB was 2% among Hispanic mothers who attended schools during adolescence with 90% Black students.

After adjusting for individual-, school-, and neighborhood-level covariates, there were null associations between the proportion of Black students in a school and the prevalence of PTB among non-Hispanic White and non-Hispanic Black mothers. In Figure 1a, the predicted prevalence of PTB among non-Hispanic White mothers appeared to be lower as the proportion of Black students within a school was higher. However, there was no overall association observed among this racial/ethnic sub-group.

Proportion Non-White Students and Experiences of PTB

After adjusting for individual-, school-, and neighborhood-level covariates, there were null associations between the proportion of non-White students in a school and the prevalence of PTB, among Hispanic, non-Hispanic White, and non-Hispanic Black female participants who had experiences a first birth by Wave V (*Table 3*). In Figure 1b, the predicted prevalence of PTB among Hispanic and non-Hispanic White mothers appeared to be lower as the proportion of non-White students within a school was higher. However, there were no overall associations observed in these racial/ethnic sub-groups. In addition, the predicted prevalence of PTB among non-Hispanic Black mothers remained constant – around 17 or 18% – as the proportion of non-White students within a school during adolescence increased from 10% to 90%.

School Entropy and Experiences of PTB

After adjusting for individual-, school-, and neighborhood-level covariates, there were null associations between the levels of school racial/ethnic diversity and the prevalence of PTB, among Hispanic, non-Hispanic White, and non-Hispanic Black female participants who had experiences a first birth by Wave V (*Table 3*). In Figure 1b, the predicted prevalence of PTB among non-Hispanic Black mothers was lower as the levels of racial diversity within a school were higher. However, there was no overall association in this racial/ethnic group. Additionally, the predicted prevalence of PTB among Hispanic and non-Hispanic mothers remained constant – around 14 or 15% – as the levels of racial diversity within a school during adolescence increased from low to high.

Black-White Dissimilarity Index (District-Level Segregation) and Experiences of PTB

After adjusting for individual-, school-, and neighborhood-level covariates, there were null associations between district-level segregation and the prevalence of PTB, among Hispanic, non-Hispanic White, and non-Hispanic Black female participants with a first birth by Wave V of Add Health (*Table 3*). In Figure 1d, the predicted prevalence of PTB across all racial/ethnic groups of mothers appeared to be lower as the levels of the Black-White dissimilarity index. However, there were no overall associations between the Black-White dissimilarity index and the prevalence of PTB in this study population.

Black-White Exposure Index (District-Level Segregation) and Experiences of PTB

After adjusting for individual-, school-, and neighborhood-level covariates, there were null associations between district-level segregation and the prevalence of PTB, among Hispanic, non-Hispanic White, and non-Hispanic Black female participants with a first birth by Wave V of Add Health (*Table 3*). In Figure 1e, the predicted prevalence of PTB among Hispanic mothers appeared to be lower as the levels of the Black-White exposure index were higher. However, there was no observed association in this racial/ethnic sub-group.

Similar to non-Hispanic Whites, there appears to be an increasing trend in the predicted prevalence of PTB among non-Hispanic Black mothers by this measure of district-level segregation. However, there was no meaningful relationship in this racial/ethnic group. Furthermore, though there was a null association observed for non-Hispanic Black mothers, there is an important finding form the Appendix that is worth noting. After adjusting for school-level characteristics, there was a significant association between the Black-White exposure index and the prevalence of PTB among

non-Hispanic Black motehrs. Nevertheless, this association was attenuated after adjusting for neighborhood-level characteristics.

DISCUSSION

Our study found that there were racial/ethnic differences in the types of schools attended by the female participants who experienced a first birth by Wave V of Add Health. Non-Hispanic White mothers were more likely to attend predominantly White schools, while non-Hispanic Black mothers were more likely to attend predominantly Black/non-White schools. Hispanic mothers were most likely to attend schools during adolescence with higher levels of racial/ethnic diversity. Regarding district-level segregation, non-Hispanic Black mothers were more likely to attend racially-segregated schools than Hispanic and non-Hispanic White mothers. Also, the prevalence of PTB in this study population varied by race/ethnicity, with non-Hispanic mothers having the lowest prevalence (12%) and non-Hispanic Black mothers having the highest prevalence (16%). It is important to note that the prevalence of PTB was higher in Add Health than the national prevalence of 11%.

There were also racial/ethnic differences in the association between the racial structure of school environments and experiences of PTB, after adjusting for characteristics at the three levels mentioned above. Among Hispanic mothers, a one-unit change in the proportion of Black students within a school was associated with a lower prevalence of PTB. Neither school racial/ethnic composition nor district-level segregation impacted the likelihood of experiencing a PTB for non-Hispanic White mothers. Additionally, the association between the Black-White exposure index and the prevalence of PTB, among non-Hispanic Black mothers, was attenuated after the adjustment of relevant covariates. This attenuation suggests that neighborhood-level

characteristics could potentially explain this relationship between district-level segregation and the likelihood of PTB among non-Hispanic Black mothers.

It is possible that Hispanic participants who attended schools with a lower proportion of Black students had a higher prevalence of PTB, as a result of the potentially detrimental experiences of racial/ethnic discrimination in these schools that are a majority White. Previous literature has suggested that attending predominantly White schools may have a negative effect on the health of Black students due to the harmful experiences of discrimination. One Add Health study found that, due to experiences of racial discrimination, Black students attending majority White schools were more likely to have worse adult health outcomes than their white peers[18]. Therefore, it is possible that these experiences of racial discrimination impact health through the negative physiological consequences of stress[28]. However, it is important to note that these studies only documented the experiences of discrimination and stress among Black students. Little is known about the experiences of Hispanic students at these types of schools, but it is possible that the significant association between the proportion Black students within a school and prevalence of PTB can be attributed to similar discriminatory experiences.

Regarding the Black-White exposure index, non-Hispanic Black participants who attended schools within more racially segregated districts had a higher prevalence of PTB. Though the findings from this study were imprecise, they are consistent with previous research that established a relationship between childhood socioeconomic disadvantage and adverse pregnancy outcomes. Socioeconomic disadvantage in childhood or adolescence can impact experiences of PTB, through its potential impact on stress and inflammatory processes associated with the disadvantage experienced

during such a critical period of development[6-8]. Thus, it is possible that non-Hispanic Black students had worse pregnancy outcomes due to the lack of access to important health-relevant socioeconomic resources in these segregated environments. To our knowledge, this is the first study that has investigated the association between district-level segregation in adolescence and experiences of PTB. After adjusting for neighborhood-level socioeconomic and demographic characteristics, the association between the exposure index and prevalence of PTB was no longer significant, suggesting that these neighborhood contextual factors could explain this relationship among non-Hispanic Black participants.

This study had a few methodological limitations worth noting. First, due to the nature of the Add Health data, we were only able to utilize information about female participants' first births. Thus, since we restricted our analyses to participants with first births, this limits the external validity of our findings to non-first births, particularly among women who have had multiple births by Wave V. Second, given that the experiences of PTB were self-reported retrospectively, it is possible that there could be some misclassification of the outcome. First births occurred between 14 and 41 years old, so there could be some discrepancies in memory at Wave V for the first births that occurred earlier in life. However, it is important to note that Add Health researchers did not require participants to report the specific gestational week at the time of the first birth, but rather participants only had to recall whether it was born preterm (before 37 weeks) or not. Third, Add Health is an observational study and there are likely unmeasured confounders for which we were unable to adjust. However, we attempted to remedy this by adjusting for the most relevant (measured) confounders at three distinct levels: the school, the neighborhood, and the individual. Lastly, Add Health

only reported Black-White segregation indices, which describe the levels of segregation only between Black and White students within a school district. The construction of these segregation indices could have contributed to the null findings among Hispanic participants. It is possible that the Hispanic-White dissimilarity and exposure indices are more relevant for Hispanics.

Our study also had four important methodological strengths worth highlighting. First, Add Health was a nationally-representative longitudinal study, which used a complex stratification and sampling method. This design increased the external validity of our findings. Second, though we restricted our analyses to female participants with a first birth at Wave V, our analyses still consisted of a relatively large study population (N=4,380), which increased the precision of our effect estimates. Thirst, this is one of the first studies to investigate the association between both school-level measures (e.g., proportion Black students, proportion non-White students, racial/ethnic diversity) and district-level measures (e.g., Black-White dissimilarity index, Black-White exposure index). The school-level exposures assessed the absolute racial/ethnic make-up within a school, while the district-level exposures assessed the social and spatial processes of racial/ethnic groups across schools within a district. In measuring the racial structure of school environments in these various ways, we were able to provide a more extensive examination of the impact of adverse pregnancy outcomes. Lastly, we were able to control for potential confounding at the three levels described above. It was critical to explore a more comprehensive understanding of these relationships given the complicated social processes that operate within schools and school districts.

The findings from this study provide evidence that the racial/ethnic environments of schools and school districts are potentially relevant social determinants

of PTB. Due to the complex structure of U.S. middle and high schools, future research should investigate the potential mediating role of racial/ethnic discrimination in this relationship. Additional research is also warranted that examines other segregation indices and spatial processes that are operating within school systems. Since Hispanic mothers displayed a lower prevalence of PTB in schools with higher proportions of Black students, it is critical that we explore the impact of Hispanic-White and Hispanic-Black segregation measures in order to fully understand the racial/ethnic dynamics and interactions within schools and districts. Given the history of school segregation and the racial/ethnic shifts in the composition of U.S. public schools since the late 20th century, our findings can help contribute to the social epidemiology literature surrounding the health consequences of attending racially-homogenous or segregated schools during adolescence, as well as those passed across generations, from mother to child through experiences of PTB.

Table 1. School Characteristics by Race/Ethnicity among Female Participants who had a First Birth by Wave V of Add Health, 2016-2018*.

	Hispanic	Non-Hispanic White	Non-Hispanic Black
School Racial/Ethnic Composition			
Proportion Black Students**	0.16 (0.02)	0.10 (0.01)	0.57 (0.05)
Proportion Non-White Students**	0.58 (0.05)	0.16 (0.02)	0.65 (0.05)
School Entropy (Multi-Group, Standardized)**	0.54 (0.03)	0.23 (0.02)	0.36 (0.04)
District-Level Segregation			
Black-White Dissimilarity Index**	0.39 (0.04)	0.27 (0.01)	0.37 (0.03)
Black-White Exposure Index**	0.57 (0.06)	0.75 (0.03)	0.40 (0.03)

** N=4,380 female participants who: (1) participated in the In-School Questionnaire and In-Home Interview in Wave I, (2) participated in the mixed-mode survey in Wave V, and (3) had a first birth by Wave V of Add Health. All statistics are reported as: N; Weighted % (SE).*
***Statistics are reported as: Weighted Mean (SE).*

Table 2. Individual and Neighborhood Characteristics by Race/Ethnicity among Female Participants who had a First Birth by Wave V of Add Health, 2016-2018*.

	Hispanic	Non-Hispanic White	Non-Hispanic Black
Individual Characteristics			
Maternal Age at First Birth			
14 – 19 years old	122; 25.5 (3.5)	349; 15.4 (1.3)	240; 32.0 (2.9)
20 – 29 years old	151; 21.4 (2.8)	704; 26.5 (1.8)	137; 12.6 (1.7)
30 – 41 years old	328; 53.1 (2.5)	1,467; 58.1 (1.6)	469; 55.4 (2.5)
Parent's Educational Attainment at Wave I			
Less than HS	111; 28.6 (4.0)	110; 6.1 (1.0)	58; 13.8 (2.5)
HS Diploma/GED	116; 32.4 (3.2)	615; 37.1 (2.0)	178; 38.4 (3.7)
Some College or Associate's Degree	72; 13.7 (2.8)	346; 19.0 (1.4)	105; 20.1 (2.6)
Bachelor's Degree or Higher	93; 25.3 (3.8)	740; 37.8 (2.4)	298; 27.8 (2.7)
Experiences of Preterm Birth by Wave V			
Preterm: < 37 weeks gestation	86; 14.9 (2.2)	337; 12.5 (0.8)	155; 15.9 (1.7)
Neighborhood Characteristics			
Proportion Residents Living Below Poverty Level, 1990**	0.17 (0.01)	0.12 (0.01)	0.27 (0.02)
Proportion Residents who are Black, 1990**	0.10 (0.01)	0.05 (0.01)	0.54 (0.04)
Proportion Residents who are Non-White, 1990**	0.29 (0.03)	0.08 (0.01)	0.57 (0.03)

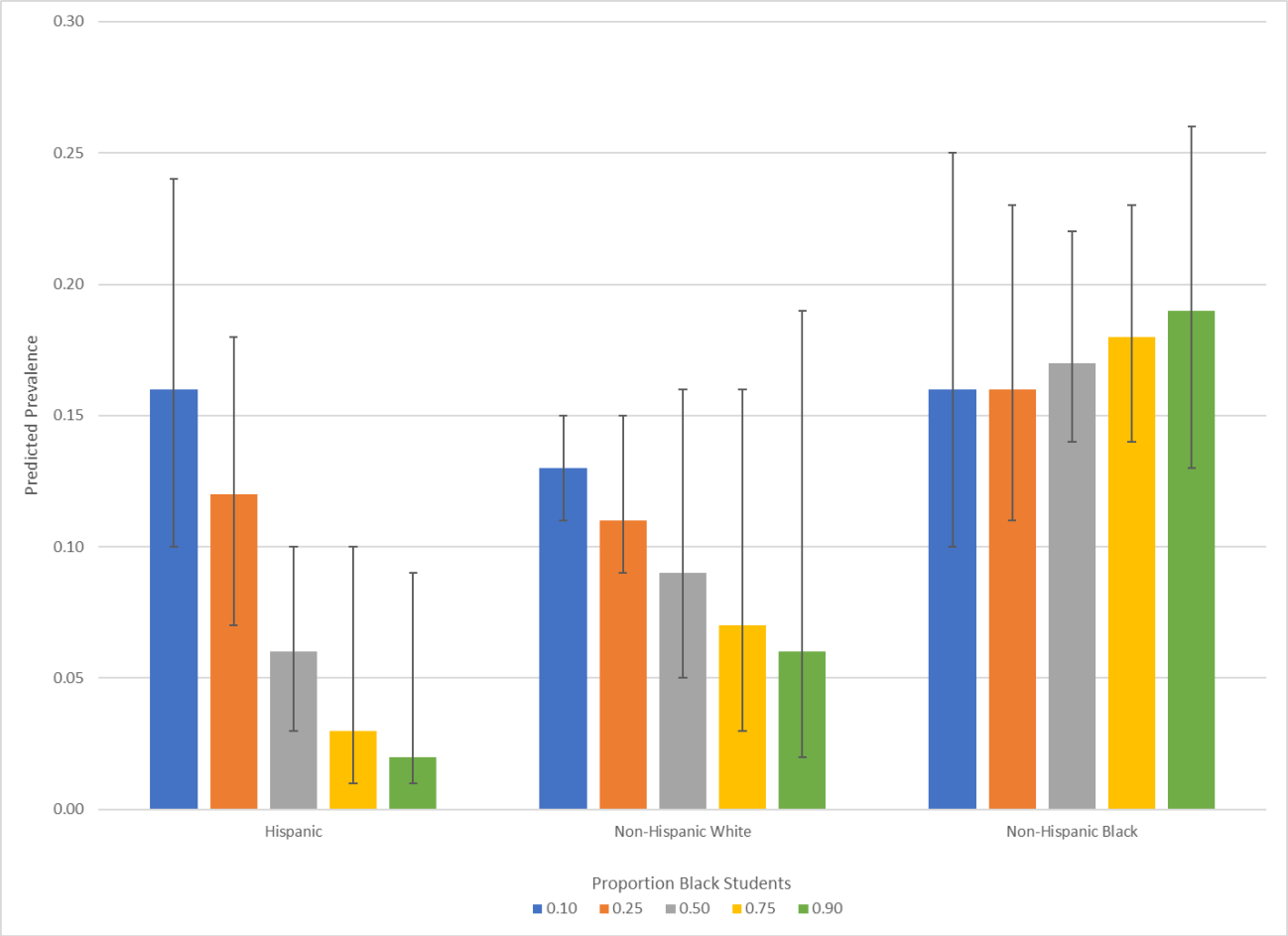
** N=4,380 female participants who: (1) participated in the In-School Questionnaire and In-Home Interview in Wave I, (2) participated in the mixed-mode survey in Wave V, and (3) had a first birth by Wave V of Add Health. All statistics are reported as: N; Weighted % (SE).*
***Statistics are reported as: Weighted Mean (SE).*

Table 3. Adjusted Prevalence Ratios* for Experiences of Preterm Birth by the Racial/Ethnic Structure of Schools and School Systems during Adolescence (Wave I, 1994-1995) and Individual Race/Ethnicity, among Add Health Participants who had a First Birth by Wave V (2016-2018).

	All Females	Hispanic	Non-Hispanic White	Non-Hispanic Black
Proportion Black Students	0.79 (0.35, 1.76)	0.05 (0.00, 0.56)	0.37 (0.09, 1.63)	1.28 (0.45, 3.64)
Proportion Non-White Students	0.76 (0.38, 1.53)	0.26 (0.04, 1.71)	0.62 (0.19, 2.00)	1.06 (0.33, 3.39)
School Entropy (Multi-Group)	0.90 (0.48, 1.67)	1.06 (0.08, 14.05)	1.28 (0.45, 3.61)	0.52 (0.17, 1.59)
Black-White Dissimilarity Index	0.72 (0.33, 1.55)	0.54 (0.06, 5.37)	0.73 (0.21, 2.54)	0.62 (0.12, 3.12)
Black-White Exposure Index	1.10 (0.69, 1.75)	0.30 (0.06, 1.49)	1.31 (0.73, 2.36)	1.47 (0.30, 7.16)

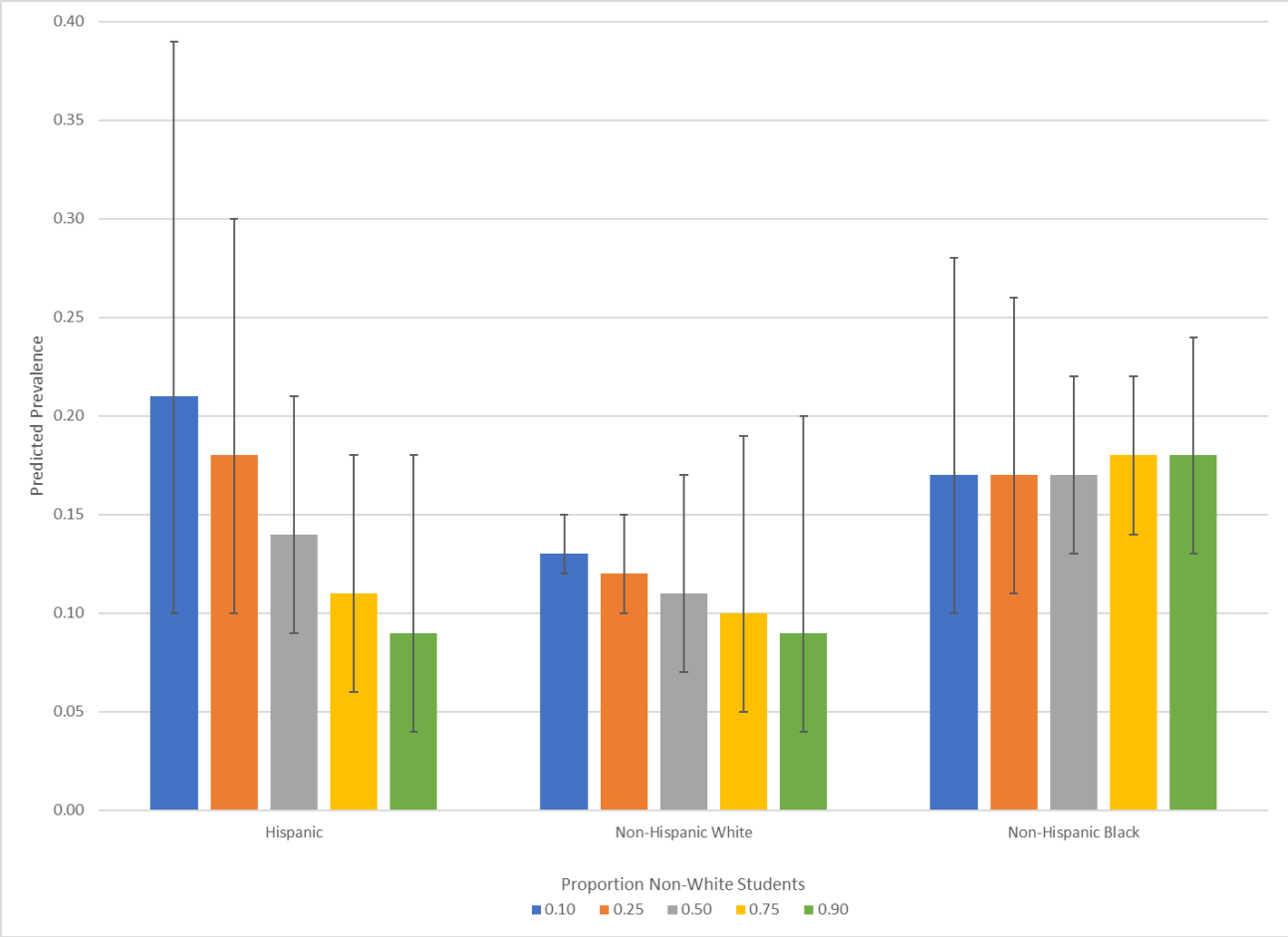
**Adjusted for school-level characteristics (school size, school urbanicity, school type), neighborhood-level characteristics (neighborhood proportion living in poverty, neighborhood proportion who are black), and individual-level characteristics (maternal age at first birth, parent's highest education).*

Figure 1a. Adjusted* Predicted Prevalence for Experiences of Preterm Birth by Proportion Black Students in a School and Individual Race/Ethnicity.



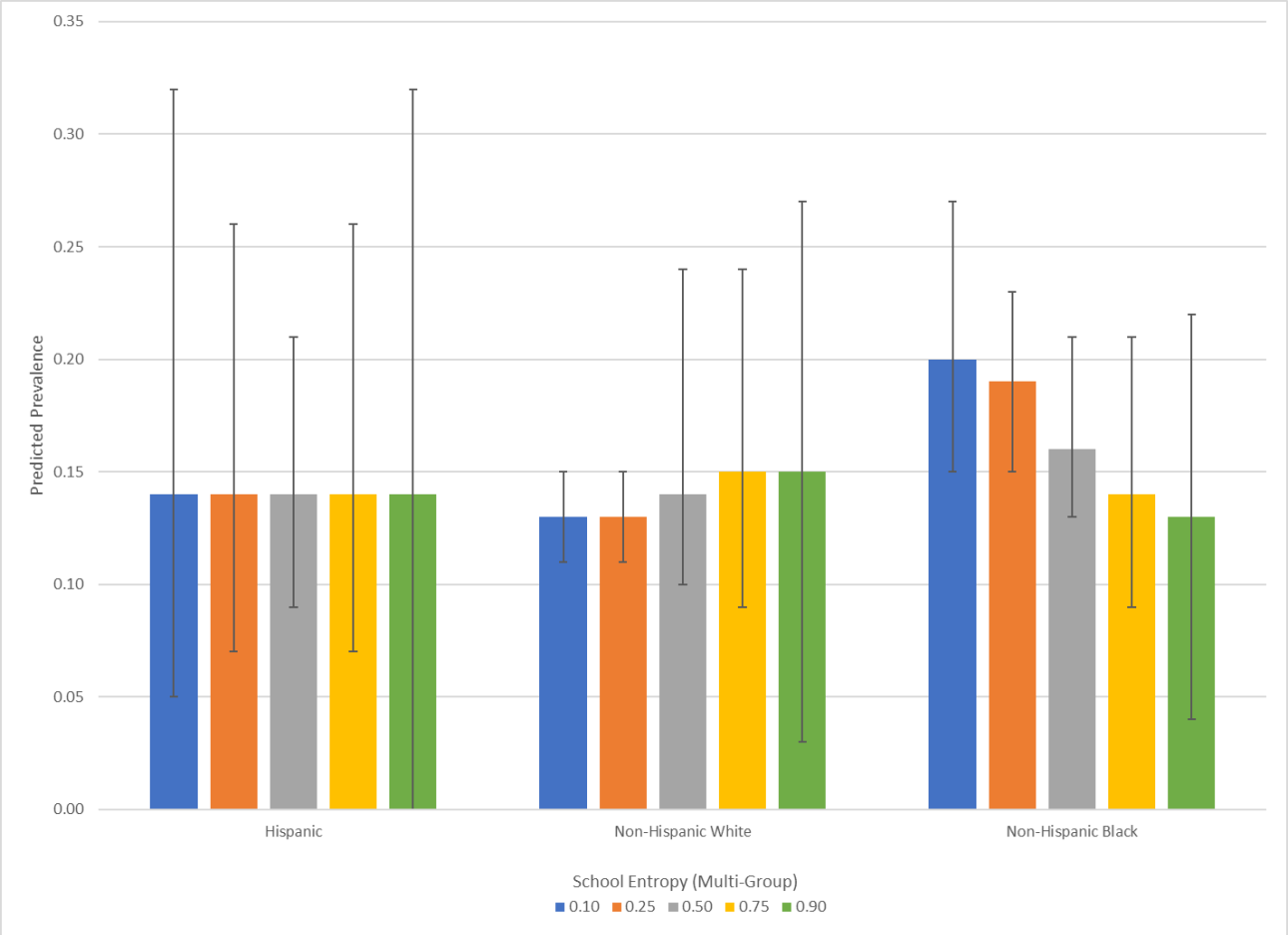
**Adjusted for school-level characteristics (school size, school urbanicity, school type), neighborhood-level characteristics (neighborhood proportion living in poverty, neighborhood proportion who are black), and individual-level characteristics (maternal age at first birth, parent's highest education).*

Figure 1b. Adjusted* Predicted Prevalence for Experiences of Preterm Birth by Proportion Non-White Students in a School and Individual Race/Ethnicity.



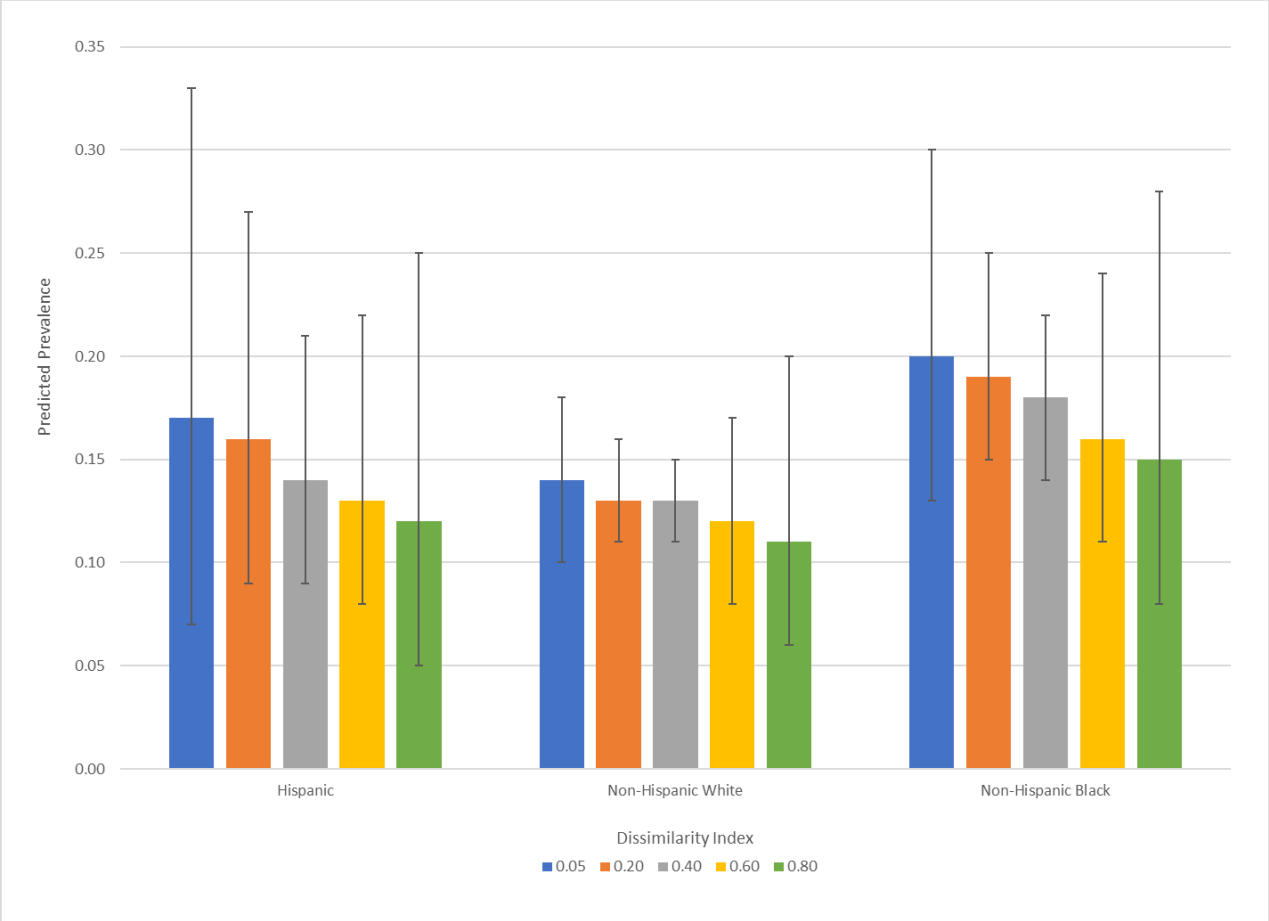
**Adjusted for school-level characteristics (school size, school urbanicity, school type), neighborhood-level characteristics (neighborhood proportion living in poverty, neighborhood proportion who are non-white), and individual-level characteristics (maternal age at first birth, parent's highest education).*

Figure 1c. Adjusted* Predicted Prevalence for Experiences of Preterm Birth by School Entropy (Multi-Group, Standardized) and Individual Race/Ethnicity.



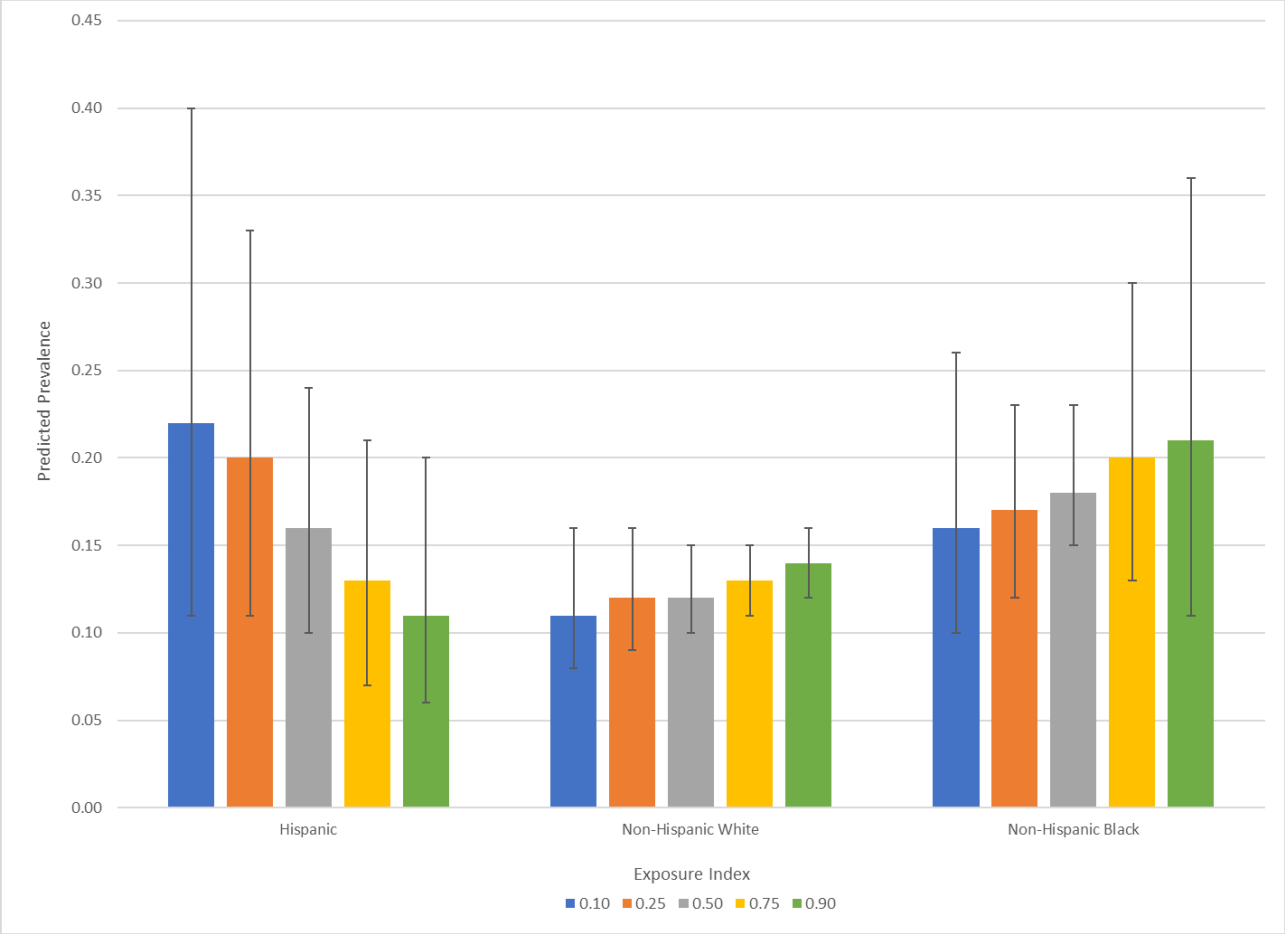
**Adjusted for school-level characteristics (school size, school urbanicity, school type), neighborhood-level characteristics (neighborhood proportion living in poverty, neighborhood proportion who are non-white), and individual-level characteristics (maternal age at first birth, parent's highest education).*

Figure 1d. Adjusted* Predicted Prevalence for Experiences of Preterm Birth by Average School Segregation – Black-White Dissimilarity Index – and Individual Race/Ethnicity.



**Adjusted for school-level characteristics (school size, school urbanicity, school type), neighborhood-level characteristics (neighborhood proportion living in poverty, neighborhood proportion who are non-white), and individual-level characteristics (maternal age at first birth, parent's highest education).*

Figure 1e. Adjusted* Predicted Prevalence for Experiences of Preterm Birth by Average School Segregation – Black-White Exposure Index – and Individual Race/Ethnicity.



**Adjusted for school-level characteristics (school size, school urbanicity, school type), neighborhood-level characteristics (neighborhood proportion living in poverty, neighborhood proportion who are non-white), and individual-level characteristics (maternal age at first birth, parent's highest education).*

REFERENCES

1. Goldenberg RL, Culhane JF, Iams JD, Romero R. Epidemiology and causes of preterm birth. *Lancet*. 2008; 371(9606):75-84.
2. Schoen CN, Tabbah S, Iams JD, Caughey AB, Berghella V. Why the United States preterm birth rate is declining. *Am J Obstet Gynecol*. 2015; 213(2):175-80.
3. Martin JA, Hamilton BE, Osterman MJ, Driscoll AK, Mathews TJ. Births: Final Data for 2015. *Natl Vital Stat Rep*. 2017; 66(1):1.
4. Beck S, Wojdyla D, Say L, Betran AP, Merialdi M, Requejo JH, et al. The worldwide incidence of preterm birth: A systematic review of maternal mortality and morbidity. *Bull World Health Organ*. 2010; 88(1):31-8.
5. Wadhwa PD, Culhane JF, Rauh V, Barve SS, Hogan V, Sandman CA, et al. Stress, infection and preterm birth: A biobehavioural perspective. *Paediatr Perinat Epidemiol*. 2001; 15 Suppl 2:17-29.
6. Lu MC, Halfon N. Racial and ethnic disparities in birth outcomes: A life-course perspective. *Matern Child Health J*. 2003; 7(1):13-30.
7. Blumenshine P, Egerter S, Barclay CJ, Cubbin C, Braveman PA. Socioeconomic disparities in adverse birth outcomes: A systematic review. *Am J Prev Med*. 2010; 39(3):263-72.
8. Kramer MS, Goulet L, Lydon J, Séguin L, McNamara H, Dassa C, et al. Socio-economic disparities in preterm birth: Causal pathways and mechanisms. *Paediatr Perinat Epidemiol*. 2001; 15 Suppl 2: 104-23.
9. Wadhwa PD, Culhane JF, Rauh V, Barve SS. Stress and preterm birth: Neuroendocrine, immune/inflammatory, and vascular mechanisms. *Matern Child Health J*. 2001; 5(2):119-25.
10. Hobel CJ, Dunkel-Schetter C, Roesch SC, Castro LC, Arora CP. Maternal plasma corticotropin-releasing hormone associated with stress at 20 weeks' gestation in pregnancies ending in preterm delivery. *Am J Obstet Gynecol*. 1999; 180(1 Pt 3):S257-63.
11. Lockwood CJ. Stress-associated preterm delivery: The role of corticotropin-releasing hormone. *Am J Obstet Gynecol*. 1999; 180(1 Pt 3):S264-6.
12. Geronimus AT. The weathering hypothesis and the health of African-American women and infants: Evidence and speculations. *Ethn Dis*. 1992; 2(3):207-21.
13. Culhane JF, Goldenberg RL. Racial disparities in preterm birth. *Semin Perinatol*. 2011; 35(4):234-9.
14. MacDorman MF. Race and ethnic disparities in fetal mortality, preterm birth, and infant mortality in the United States: An overview. *Semin Perinatol*. 2011; 35(4):200-8.
15. Kramer MS, Séguin L, Lydon J, Goulet L. Socio-economic disparities in pregnancy outcome: Why do the poor fare so poorly? *Paediatr Perinat Epidemiol*. 2000; 14(3):194-210.

16. Peacock JL, Bland JM, Anderson HR. Preterm delivery: Effects of socioeconomic factors, psychological stress, smoking, alcohol, and caffeine. *BMJ*. 1995; 311(7004):531-5.
17. Braveman PA, Heck K, Egerter S, Marchi KS, Dominguez TP, Cubbin C, et al. The role of socioeconomic factors in Black-White disparities in preterm birth. *Am J Public Health*. 2015; 105(4):694-702.
18. Goosby BJ, Walsemann KM. School racial composition and race/ethnic differences in early adulthood health. *Health Place*. 2012; 18(2):296-304.
19. Plessy v. Ferguson, 163 U.S. 537 (1896).
20. Tischauser LV. *Jim Crow Laws: Landmarks of the American Mosaic*. Greenwood: Santa Barbara, CA; 2012.
21. Clotfelter CT. *After Brown: The Rise and Retreat of School Desegregation*. Princeton University Press: Princeton, NJ; 2004.
22. Brown v. Board of Education of Topeka, 347 U.S. 483 (1954).
23. Milliken v. Bradley, 418 U.S. 717 (1974).
24. Riddick v. School Board of the City of Norfolk, Virginia 784 F.2d 251 (4th Cir. 1986).
25. Board of Education of Oklahoma v. Dowell, 498 U.S. 237 (1991).
26. Freeman v. Pitts, 503 U.S. 567 (1992).
27. Reardon SF, Yun JT, Mcnulty Eitle T. The changing structure of school segregation: Measurement and evidence of multiracial metropolitan-area school segregation, 1989-1995. *Demography*. 2000; 37(3):351-64.
28. Williams DR. Race, socioeconomic status, and health: The added effects of racism and discrimination. *Ann N Y Acad Sci*. 1999; 896:173-88.
29. Orfield G, Eaton SE, The Harvard Project on School Desegregation. *Dismantling Desegregation: The Quiet Reversal of Brown v. Board of Education*. New Press: New York, NY; 1996.
30. Krieger N. Theories for social epidemiology in the 21st century: An ecosocial perspective. *Int J Epidemiol*. 2001; 30(4):668-77.
31. Gavin AR, Thompson E, Rue T, Guo Y. Maternal early life risk factors for offspring birth weight: Findings from the Add Health study. *Prev Sci*. 2012; 13(2):162-72.
32. Wilson AE, Shuey KM, Elder GH, Jr. Cumulative advantage processes as mechanisms of inequality in life course health. *Am J Sociology*. 2007; 112(6):1886-1924.
33. McEwen BS. Protective and damaging effects of stress mediators. *N Engl J Med*. 1998; 338(3):171-9.
34. McEwen BS, Seeman T. Protective and damaging effects of mediators and stress: Elaborating and testing the concepts of allostasis and allostatic load. *Ann N Y Acad Sci*. 1999; 896:30-47.

35. Singh-Manoux A, Ferrie JE, Chandola T, Marmot M. Socioeconomic trajectories across the life course and health outcomes in midlife: Evidence for the accumulation hypothesis? *Int J Epidemiol*. 2004; 33(5):1072-9.
36. Harris KM. *The Add Health Study: Design and Accomplishments*. Carolina Population Center, University of North Carolina at Chapel Hill, 2013. Available at: http://www.cpc.unc.edu/projects/addhealth/data/guides/DesignPaperWIIV.pdf .
37. Harris KM, Halpern CT, Biemer P, Liao D, Dean SC. *Add Health Wave V Documentation: Sampling and Mixed-Mode Survey Design*. Carolina Population Center, University of North Carolina at Chapel Hill, 2019. Available at: http://www.cpc.unc.edu/projects/addhealth/documentation/guides/.
38. Johnson R. *Add Health Wave I School District Data Documentation*. Carolina Population Center, University of North Carolina at Chapel Hill, 2020. Available at: http://www.cpc.unc.edu/addhealth.

SPECIFIC AIM 3: APPENDIX

A3.1. Unadjusted Prevalence Ratios for Experiences of Preterm Birth by the Proportion of Black Students within a School during Adolescence (Wave I, 1994-1995) and Individual Race/Ethnicity, among Add Health Participants who had a First Birth by Wave V (2016-2018).

	All Females	Hispanic	Non-Hispanic White	Non-Hispanic Black
Proportion Black Students	0.91 (0.61, 1.36)	0.11 (0.01, 1.84)	0.68 (0.30, 1.54)	0.67 (0.32, 1.41)
0.10*	0.15 (0.14, 0.17)	0.17 (0.13, 0.23)	0.14 (0.13, 0.16)	0.20 (0.13, 0.31)
0.25*	0.15 (0.13, 0.16)	0.13 (0.08, 0.20)	0.14 (0.12, 0.16)	0.19 (0.14, 0.26)
0.50*	0.15 (0.13, 0.17)	0.08 (0.03, 0.21)	0.13 (0.09, 0.17)	0.17 (0.14, 0.22)
0.75*	0.14 (0.12, 0.17)	0.05 (0.01, 0.23)	0.12 (0.07, 0.18)	0.16 (0.13, 0.20)
0.90*	0.14 (0.11, 0.18)	0.03 (0.00, 0.25)	0.11 (0.06, 0.19)	0.15 (0.11, 0.20)

**Predicted Prevalence (95% CIs).*

A3.2. Adjusted Prevalence Ratios for Experiences of Preterm Birth by the Proportion of Black Students within a School during Adolescence (Wave I, 1994-1995) and Individual Race/Ethnicity, among Add Health Participants who had a First Birth by Wave V (2016-2018).

	All Females	Hispanic	Non-Hispanic White	Non-Hispanic Black
Proportion Black Students	0.94 (0.63, 1.39)	0.09 (0.01, 1.09)	0.79 (0.34, 1.85)	0.73 (0.35, 1.51)
0.10*	0.15 (0.13, 0.17)	0.17 (0.13, 0.23)	0.14 (0.13, 0.16)	0.19 (0.13, 0.28)
0.25*	0.15 (0.13, 0.16)	0.13 (0.09, 0.19)	0.14 (0.12, 0.16)	0.19 (0.13, 0.25)
0.50*	0.15 (0.13, 0.17)	0.08 (0.03, 0.18)	0.13 (0.10, 0.18)	0.17 (0.14, 0.22)
0.75*	0.14 (0.12, 0.18)	0.05 (0.01, 0.17)	0.13 (0.08, 0.20)	0.16 (0.13, 0.20)
0.90*	0.14 (0.11, 0.18)	0.03 (0.01, 0.17)	0.12 (0.07, 0.22)	0.16 (0.12, 0.20)
School Size				
1 – 400 students	1.23 (0.91, 1.67)	3.32 (1.70, 6.50)	1.33 (0.93, 1.90)	0.98 (0.57, 1.69)
401 – 1,000 students	0.95 (0.74, 1.21)	0.80 (0.37, 1.71)	1.13 (0.84, 1.50)	0.72 (0.42, 1.23)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.91 (0.70, 1.18)	6.01 (1.27, 28.53)	0.83 (0.57, 1.20)	1.19 (0.59, 2.42)
Suburban	0.92 (0.73, 1.15)	6.36 (1.38, 29.27)	0.78 (0.60, 1.02)	1.41 (0.85, 2.34)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.23 (0.91, 1.67)	0.48 (0.26, 0.90)	1.61 (0.99, 2.61)	0.97 (0.39, 2.42)
Private	1.00	1.00	1.00	1.00

**Predicted Prevalence (95% CIs).*

A3.3. Adjusted Prevalence Ratios for Experiences of Preterm Birth by the Proportion of Black Students within a School during Adolescence (Wave I, 1994-1995) and Individual Race/Ethnicity, among Add Health Participants who had a First Birth by Wave V (2016-2018).

	All Females	Hispanic	Non-Hispanic White	Non-Hispanic Black
Proportion Black Students	0.62 (0.33, 1.19)	0.04 (0.00, 0.44)	0.57 (0.19, 1.72)	0.73 (0.32, 1.63)
0.10*	0.16 (0.14, 0.17)	0.18 (0.14, 0.24)	0.14 (0.13, 0.16)	0.19 (0.13, 0.28)
0.25*	0.15 (0.13, 0.16)	0.13 (0.08, 0.18)	0.13 (0.11, 0.16)	0.19 (0.13, 0.25)
0.50*	0.13 (0.11, 0.16)	0.06 (0.03, 0.14)	0.12, 0.08, 0.18)	0.17 (0.14, 0.22)
0.75*	0.12 (0.09, 0.17)	0.03 (0.01, 0.11)	0.11 (0.05, 0.19)	0.16 (0.13, 0.20)
0.90*	0.11 (0.07, 0.17)	0.02 (0.00, 0.10)	0.10 (0.04, 0.21)	0.16 (0.12, 0.21)
School Size				
1 – 400 students	1.24 (0.90, 1.72)	3.40 (1.75, 6.60)	1.32 (0.89, 1.96)	1.08 (0.60, 1.95)
401 – 1,000 students	0.95 (0.74, 1.22)	0.83 (0.40, 1.75)	1.13 (0.84, 1.53)	0.70 (0.41, 1.22)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.89 (0.69, 1.16)	6.62 (1.58, 27.74)	0.82 (0.57, 1.19)	1.17 (0.58, 2.38)
Suburban	0.90 (0.71, 1.14)	8.14 (1.86, 35.53)	0.77 (0.59, 1.02)	1.41 (0.87, 2.29)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.30 (0.94, 1.79)	0.47 (0.26, 0.87)	1.67 (0.99, 2.81)	0.12 (0.43, 2.87)
Private	1.00	1.00	1.00	1.00
Neighborhood Poverty, 1990	0.69 (0.20, 2.33)	4.34 (0.19, 98.66)	0.87 (0.13, 5.66)	0.29 (0.05, 1.79)
Neighborhood Proportion Black, 1990	1.87 (0.94, 3.71)	3.60 (0.30, 42.85)	1.95 (0.28, 13.80)	1.23 (0.53, 2.83)

**Predicted Prevalence (95% CIs).*

A3.4. Adjusted Prevalence Ratios for Experiences of Preterm Birth by the Proportion of Black Students within a School during Adolescence (Wave I, 1994-1995) and Individual Race/Ethnicity, among Add Health Participants who had a First Birth by Wave V (2016-2018).

	All Females	Hispanic	Non-Hispanic White	Non-Hispanic Black
Proportion Black Students	0.79 (0.35, 1.76)	0.05 (0.00, 0.56)	0.37 (0.09, 1.63)	1.28 (0.45, 3.64)
0.10*	0.14 (0.13, 0.16)	0.16 (0.10, 0.24)	0.13 (0.11, 0.15)	0.16 (0.10, 0.25)
0.25*	0.14 (0.12, 0.15)	0.12 (0.07, 0.18)	0.11 (0.09, 0.15)	0.16 (0.11, 0.23)
0.50*	0.13 (0.10, 0.17)	0.06 (0.03, 0.13)	0.09 (0.05, 0.16)	0.17 (0.14, 0.22)
0.75*	0.12 (0.08, 0.19)	0.03 (0.01, 0.10)	0.07 (0.03, 0.18)	0.18 (0.14, 0.23)
0.90*	0.12 (0.07, 0.20)	0.02 (0.01, 0.09)	0.06 (0.02, 0.19)	0.19 (0.13, 0.26)
School Size				
1 – 400 students	1.27 (0.91, 1.77)	3.29 (1.23, 8.78)	1.31 (0.84, 2.02)	1.02 (0.53, 1.95)
401 – 1,000 students	1.03 (0.79, 1.33)	0.77 (0.30, 2.00)	1.13 (0.81, 1.58)	1.09 (0.66, 1.79)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.93 (0.67, 1.29)	5.13 (0.85, 30.75)	0.94 (0.60, 1.48)	1.90 (0.89, 4.06)
Suburban	0.92 (0.71, 1.17)	7.22 (1.23, 42.42)	0.80 (0.59, 1.10)	2.00 (1.10, 3.65)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.22 (0.88, 1.69)	0.31 (0.15, 0.61)	1.72 (1.00, 2.96)	1.37 (0.44, 4.21)
Private	1.00	1.00	1.00	1.00
Neighborhood Poverty, 1990	0.47 (0.10, 2.18)	19.84 (0.56, 705.60)	0.46 (0.04, 5.25)	0.51 (0.08, 3.45)
Neighborhood Proportion Black, 1990	2.00 (0.91, 4.43)	2.41 (0.08, 71.94)	3.15 (0.29, 33.64)	0.80 (0.34, 1.86)
Age at First Birth				
14-19 years old	0.83 (0.60, 1.16)	1.55 (0.67, 3.59)	0.76 (0.48, 1.20)	0.61 (0.30, 1.23)
20-29 years old	1.00	1.00	1.00	1.00
30-41 years old	0.87 (0.66, 1.15)	0.91 (0.43, 1.93)	0.85 (0.59, 1.22)	1.22 (0.69, 2.13)
Parental Education at Wave I				
<HS	1.07 (0.69, 1.67)	0.60 (0.24, 1.51)	1.09 (0.57, 2.09)	1.06 (0.40, 2.78)
HS Diploma	1.10 (0.81, 1.48)	0.57 (0.26, 1.23)	1.19 (0.82, 1.71)	0.90 (0.45, 1.80)
Some College	1.00 (0.68, 1.46)	0.79 (0.23, 2.76)	1.04 (0.63, 1.73)	1.06 (0.53, 2.13)
College+	1.00	1.00	1.00	1.00

**Predicted Prevalence (95% CIs).*

A3.5. Unadjusted Prevalence Ratios for Experiences of Preterm Birth by the Proportion of Non-White Students within a School during Adolescence (Wave I, 1994-1995) and Individual Race/Ethnicity, among Add Health Participants who had a First Birth by Wave V (2016-2018).

	All Females	Hispanic	Non-Hispanic White	Non-Hispanic Black
Proportion Non-White Students	0.88 (0.62, 1.25)	0.39 (0.13, 1.20)	0.59 (0.30, 1.16)	0.67 (0.32, 1.41)
0.10*	0.15 (0.13, 0.17)	0.22 (0.13, 0.36)	0.15 (0.13, 0.18)	0.20 (0.13, 0.29)
0.25*	0.15 (0.14, 0.17)	0.20 (0.13, 0.30)	0.14 (0.12, 0.16)	0.19 (0.14, 0.26)
0.50*	0.15 (0.13, 0.16)	0.16 (0.12, 0.22)	0.12 (0.10, 0.15)	0.17 (0.14, 0.22)
0.75*	0.14 (0.12, 0.17)	0.13 (0.10, 0.18)	0.11 (0.07, 0.16)	0.16 (0.13, 0.20)
0.90*	0.14 (0.12, 0.17)	0.12 (0.08, 0.17)	0.10 (0.06, 0.16)	0.15 (0.12, 0.20)

**Predicted Prevalence (95% CIs).*

A3.6. Adjusted Prevalence Ratios for Experiences of Preterm Birth by the Proportion of Non-White Students within a School during Adolescence (Wave I, 1994-1995) and Individual Race/Ethnicity, among Add Health Participants who had a First Birth by Wave V (2016-2018)

	All Females	Hispanic	Non-Hispanic White	Non-Hispanic Black
Proportion Non-White Students	0.93 (0.65, 1.33)	0.49 (0.19, 1.29)	0.72 (0.32, 1.59)	0.73 (0.35, 1.51)
0.10*	0.15 (0.13, 0.17)	0.20 (0.12, 0.32)	0.15 (0.13, 0.17)	0.19 (0.13, 0.28)
0.25*	0.15 (0.14, 0.17)	0.19 (0.12, 0.28)	0.14 (0.12, 0.16)	0.19 (0.13, 0.25)
0.50*	0.15 (0.13, 0.16)	0.16 (0.12, 0.22)	0.13 (0.10, 0.17)	0.17 (0.14, 0.22)
0.75*	0.15 (0.12, 0.17)	0.14 (0.10, 0.19)	0.12 (0.08, 0.18)	0.16 (0.13, 0.20)
0.90*	0.14 (0.12, 0.17)	0.13 (0.09, 0.18)	0.12 (0.07, 0.19)	0.16 (0.12, 0.20)
School Size				
1 – 400 students	1.22 (0.91, 1.65)	2.59 (1.12, 6.01)	1.31 (0.91, 1.87)	0.98 (0.57, 1.69)
401 – 1,000 students	0.84 (0.74, 1.19)	0.78 (0.37, 1.64)	1.12 (0.84, 1.49)	0.72 (0.42, 1.23)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.92 (0.70, 1.21)	4.38 (0.61, 31.25)	0.87 (0.57, 1.31)	1.19 (0.59, 2.42)
Suburban	0.92 (0.73, 1.15)	4.15 (0.59, 29.27)	0.79 (0.60, 1.05)	1.41 (0.85, 2.34)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.23 (0.91, 1.66)	0.48 (0.24, 0.95)	1.62 (0.99, 2.65)	0.97 (0.39, 2.42)
Private	1.00	1.00	1.00	1.00

**Predicted Prevalence (95% CIs).*

A3.7. Adjusted Prevalence Ratios for Experiences of Preterm Birth by the Proportion of Non-White Students within a School during Adolescence (Wave I, 1994-1995) and Individual Race/Ethnicity, among Add Health Participants who had a First Birth by Wave V (2016-2018).

	All Females	Hispanic	Non-Hispanic White	Non-Hispanic Black
Proportion Non-White Students	0.80 (0.45, 1.40)	0.46 (0.16, 1.33)	0.67 (0.25, 1.83)	0.67 (0.27, 1.66)
0.10*	0.16 (0.14, 0.18)	0.21 (0.12, 0.33)	0.15 (0.13, 0.17)	0.20 (0.13, 0.31)
0.25*	0.15 (0.14, 0.17)	0.19 (0.12, 0.28)	0.14 (0.12, 0.16)	0.19 (0.13, 0.28)
0.50*	0.14 (0.13, 0.16)	0.16 (0.12, 0.22)	0.13 (0.09, 0.17)	0.18 (0.14, 0.23)
0.75*	0.14 (0.11, 0.17)	0.14 (0.10, 0.19)	0.12 (0.07, 0.19)	0.17 (0.13, 0.21)
0.90*	0.13 (0.10, 0.18)	0.13 (0.09, 0.18)	0.11 (0.06, 0.21)	0.16 (0.12, 0.21)
School Size				
1 – 400 students	1.21 (0.87, 1.68)	2.66 (1.18, 5.99)	1.28 (0.86, 1.92)	1.07 (0.59, 1.94)
401 – 1,000 students	0.94 (0.74, 1.20)	0.79 (0.35, 1.77)	1.12 (0.83, 1.51)	0.70 (0.40, 1.20)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.91 (0.69, 1.21)	5.18 (0.75, 35.81)	0.86 (0.57, 1.30)	1.20 (0.60, 2.39)
Suburban	0.91 (0.72, 1.15)	5.48 (0.77, 38.78)	0.79 (0.59, 1.05)	1.43 (0.88, 2.32)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.27 (0.91, 1.76)	0.44 (0.22, 0.88)	1.62 (0.96, 2.74)	1.11 (0.42, 2.93)
Private	1.00	1.00	1.00	1.00
Neighborhood Poverty, 1990	0.87 (0.23, 3.30)	8.07 (0.38, 172.59)	1.10 (0.16, 7.64)	0.30 (0.04, 2.12)
Neighborhood Proportion NW, 1990	1.30 (0.62, 2.73)	0.65 (0.06, 6.55)	1.10 (0.16, 7.49)	1.17 (0.44, 3.13)

**Predicted Prevalence (95% CIs)*

A3.8. Adjusted Prevalence Ratios for Experiences of Preterm Birth by the Proportion of Non-White Students within a School during Adolescence (Wave I, 1994-1995) and Individual Race/Ethnicity, among Add Health Participants who had a First Birth by Wave V (2016-2018).

	All Females	Hispanic	Non-Hispanic White	Non-Hispanic Black
Proportion Non-White Students	0.76 (0.38, 1.53)	0.26 (0.04, 1.71)	0.62 (0.19, 2.00)	1.06 (0.33, 3.39)
0.10*	0.14 (0.12, 0.17)	0.21 (0.10, 0.39)	0.13 (0.12, 0.15)	0.17 (0.10, 0.28)
0.25*	0.14 (0.13, 0.15)	0.18 (0.10, 0.30)	0.12 (0.10, 0.15)	0.17 (0.11, 0.26)
0.50*	0.13 (0.11, 0.16)	0.14 (0.09, 0.21)	0.11 (0.07, 0.17)	0.17 (0.13, 0.22)
0.75*	0.12 (0.09, 0.17)	0.11 (0.06, 0.18)	0.10 (0.05, 0.19)	0.18 (0.14, 0.22)
0.90*	0.12 (0.08, 0.18)	0.09 (0.04, 0.18)	0.09 (0.04, 0.20)	0.18 (0.13, 0.24)
School Size				
1 – 400 students	1.25 (0.89, 1.75)	2.33 (0.75, 7.21)	1.29 (0.82, 2.04)	1.06 (0.56, 2.00)
401 – 1,000 students	1.03 (0.80, 1.33)	0.76 (0.29, 1.99)	1.12 (0.80, 1.57)	1.12 (0.69, 1.80)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.96 (0.68, 1.35)	3.79 (0.38, 37.76)	0.94 (0.56, 1.58)	1.88 (0.87, 4.03)
Suburban	0.92 (0.72, 1.19)	4.54 (0.48, 42.99)	0.81 (0.59, 1.11)	2.01 (1.11, 3.64)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.22 (0.88, 1.71)	0.31 (0.14, 0.66)	1.67 (0.98, 2.85)	1.44 (0.48, 4.30)
Private	1.00	1.00	1.00	1.00
Neighborhood Poverty, 1990	0.59 (0.11, 3.06)	16.60 (0.78, 352.85)	0.51 (0.04, 6.27)	0.64 (0.07, 5.52)
Neighborhood Proportion NW, 1990	1.74 (0.73, 4.12)	1.32 (0.07, 26.25)	1.64 (0.19, 14.18)	0.72 (0.25, 2.08)
Age at First Birth				
14-19 years old	0.84 (0.60, 1.17)	1.39 (0.56, 3.44)	0.75 (0.48, 1.19)	0.61 (0.30, 1.24)
20-29 years old	1.00	1.00	1.00	1.00
30-41 years old	0.87 (0.66, 1.15)	1.02 (0.46, 2.28)	0.85 (0.59, 1.23)	1.22 (0.69, 2.14)
Parental Education at Wave I				
<HS	1.06 (0.68, 1.65)	0.76 (0.29, 1.96)	1.07 (0.56, 2.06)	1.04 (0.39, 2.74)
HS Diploma	1.09 (0.81, 1.47)	0.68 (0.29, 1.59)	1.18 (0.82, 1.70)	0.88 (0.44, 1.76)
Some College	0.99 (0.68, 1.44)	0.89 (0.28, 2.88)	1.03 (0.62, 1.72)	1.05 (0.53, 2.09)

College+	1.00	1.00	1.00	1.00

**Predicted Prevalence (95% CIs).*

A3.9. Unadjusted Prevalence Ratios for Experiences of Preterm Birth by School Entropy (Multi-Group, Standardized) during Adolescence (Wave I, 1994-1995) and Individual Race/Ethnicity, among Add Health Participants who had a First Birth by Wave V (2016-2018).

	All Females	Hispanic	Non-Hispanic White	Non-Hispanic Black
School Entropy	0.94 (0.56, 1.57)	0.34 (0.06, 1.87)	0.68 (0.35, 1.34)	2.08 (0.62, 7.02)
0.10*	0.15 (0.13, 0.17)	0.23 (0.12, 0.40)	0.15 (0.13, 0.17)	0.14 (0.10, 0.20)
0.25*	0.15 (0.13, 0.17)	0.20 (0.12, 0.31)	0.14 (0.13, 0.16)	0.16 (0.13, 0.20)
0.50*	0.15 (0.13, 0.17)	0.16 (0.12, 0.21)	0.13 (0.11, 0.16)	0.18 (0.14, 0.24)
0.75*	0.15 (0.12, 0.18)	0.13 (0.08, 0.20)	0.12 (0.09, 0.16)	0.21 (0.13, 0.32)
0.90*	0.14 (0.11, 0.19)	0.11 (0.06, 0.21)	0.11 (0.08, 0.17)	0.23 (0.13, 0.39)

**Predicted Prevalence (95% CIs).*

A3.10. Adjusted Prevalence Ratios for Experiences of Preterm Birth by School Entropy (Multi-Group, Standardized) during Adolescence (Wave I, 1994-1995) and Individual Race/Ethnicity, among Add Health Participants who had a First Birth by Wave V (2016-2018).

	All Females	Hispanic	Non-Hispanic White	Non-Hispanic Black
School Entropy	1.19 (0.65, 2.20)	0.55 (0.16, 1.91)	0.93 (0.42, 2.30)	2.20 (0.67, 7.19)
0.10*	0.15 (0.13, 0.17)	0.19 (0.12, 0.29)	0.14 (0.12, 0.17)	0.14 (0.11, 0.19)
0.25*	0.15 (0.13, 0.16)	0.18 (0.12, 0.25)	0.14 (0.13, 0.16)	0.16 (0.13, 0.20)
0.50*	0.15 (0.13, 0.18)	0.16 (0.12, 0.21)	0.14 (0.11, 0.18)	0.19 (0.14, 0.24)
0.75*	0.16 (0.12, 0.20)	0.14 (0.09, 0.21)	0.14 (0.10, 0.21)	0.22 (0.13, 0.33)
0.90*	0.16 (0.12, 0.22)	0.13 (0.08, 0.22)	0.14 (0.09, 0.23)	0.24 (0.13, 0.39)
School Size				
1 – 400 students	1.26 (0.94, 1.69)	2.39 (0.92, 6.19)	1.33 (0.93, 1.90)	1.06 (0.64, 1.76)
401 – 1,000 students	0.96 (0.76, 1.20)	0.79 (0.39, 1.60)	1.12 (0.84, 1.48)	0.73 (0.44, 1.21)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.86, 0.63, 1.17)	3.97 (0.56, 28.16)	0.80 (0.52, 1.23)	1.08 (0.52, 2.20)
Suburban	0.90 (0.71, 1.14)	4.09 (0.58, 28.92)	0.78 (0.58, 1.05)	1.37 (0.83, 2.28)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.24 (0.92, 1.66)	0.43 (0.21, 0.87)	1.58 (0.99, 2.52)	0.98 (0.44, 2.21)
Private	1.00	1.00	1.00	1.00

**Predicted Prevalence (95% CIs).*

A3.11. Adjusted Prevalence Ratios for Experiences of Preterm Birth by School Entropy (Multi-Group, Standardized) during Adolescence (Wave I, 1994-1995) and Individual Race/Ethnicity, among Add Health Participants who had a First Birth by Wave V (2016-2018).

	All Females	Hispanic	Non-Hispanic White	Non-Hispanic Black
School Entropy	1.20 (0.64, 2.27)	0.80 (0.11, 5.75)	1.11 (0.43, 2.91)	2.08 (0.60, 7.19)
0.10*	0.15 (0.13, 0.17)	0.17 (0.08, 0.32)	0.14 (0.12, 0.17)	0.15 (0.11, 0.20)
0.25*	0.15 0.14, 0.17)	0.17 (0.10, 0.26)	0.15 (0.13, 0.16)	0.16 (0.13, 0.20)
0.50*	0.16 (0.13, 0.18)	0.16 (0.12, 0.21)	0.14 (0.11, 0.19)	0.19 (0.14, 0.24)
0.75*	0.16 (0.12, 0.21)	0.15 (0.09, 0.25)	0.15 (0.10, 0.23)	0.21 (0.13, 0.33)
0.90*	0.17 (0.12, 0.23)	0.15 (0.07, 0.29)	0.15 (0.09, 0.26)	0.23 (01.2, 0.39)
School Size				
1 – 400 students	1.26 (0.91, 1.74)	2.65 (1.10, 6.36)	1.32 (0.88, 1.97)	1.17 (0.69, 1.99)
401 – 1,000 students	0.96 (0.76, 1.21)	0.78 (0.36, 1.72)	1.12 (0.83, 1.51)	0.74 (0.44, 1.23)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.85 (0.63, 1.16)	4.74 (0.68, 33.20)	0.79 (0.51, 1.23)	1.11 (0.55, 2.22)
Suburban	0.89 (0.70, 1.13)	5.36 (0.75, 38.13)	0.77 (0.57, 1.04)	1.41 (0.87, 2.28)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.25 (0.92, 1.70)	0.41 (0.21, 0.80)	1.56 (0.96, 2.54)	1.12 (0.48, 2.58)
Private	1.00	1.00	1.00	1.00
Neighborhood Poverty, 1990	0.92 (0.24, 3.45)	5.29 (0.23, 120.01)	1.15 (0.17, 7.78)	0.40 (0.05, 3.00)
Neighborhood Proportion NW, 1990	0.99 (0.58, 1.70)	0.44 (0.03, 7.51)	0.65 (0.12, 3.48)	0.91 (0.37, 2.22)

**Predicted Prevalence (95% CIs).*

A3.12. Adjusted Prevalence Ratios for Experiences of Preterm Birth by School Entropy (Multi-Group, Standardized) during Adolescence (Wave I, 1994-1995) and Individual Race/Ethnicity, among Add Health Participants who had a First Birth by Wave V (2016-2018).

	All Females	Hispanic	Non-Hispanic White	Non-Hispanic Black
School Entropy	0.90 (0.48, 1.67)	1.06 (0.08, 14.05)	1.28 (0.45, 3.61)	0.52 (0.17, 1.59)
0.10*	0.14 (0.12, 0.16)	0.14 (0.05, 0.32)	0.13 (0.11, 0.15)	0.20 (0.15, 0.27)
0.25*	0.14 (0.12, 0.15)	0.14 (0.07, 0.26)	0.13 (0.11, 0.15)	0.19 (0.15, 0.23)
0.50*	0.14 (0.11, 0.16)	0.14 (0.09, 0.21)	0.14 (0.10, 0.19)	0.16 (0.13, 0.21)
0.75*	0.13 (0.10, 0.17)	0.14 (0.07, 0.26)	0.15 (0.09, 0.24)	0.14 (0.09, 0.21)
0.90*	0.13 (0.09, 0.18)	0.14 (0.06, 0.32)	0.15 (0.08, 0.27)	0.13 (0.07, 0.22)
School Size				
1 – 400 students	1.24 (0.87, 1.78)	2.53 (0.69, 9.28)	1.36 (0.85, 2.15)	0.93 (0.47, 1.87)
401 – 1,000 students	1.03 (0.80, 1.34)	0.73 (0.28, 1.94)	1.13 (0.81, 1.59)	1.09 (0.67, 1.77)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.95 (0.66, 1.36)	3.64 (0.35, 38.04)	0.83 (0.48, 1.44)	2.10 (0.98, 4.49)
Suburban	0.92 (0.71, 1.19)	4.80 (0.50, 46.52)	0.78 (0.57, 1.07)	2.05 (1.12, 3.74)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.18 (0.85, 1.65)	0.28 (0.13, 0.59)	1.61 (0.95, 2.73)	1.35 (0.42, 4.33)
Private	1.00	1.00	1.00	1.00
Neighborhood Poverty, 1990	0.62 (0.13, 3.06)	13.98 (0.41, 475.94)	0.51 (0.04, 6.31)	0.53 (0.08, 3.70)
Neighborhood Proportion NW, 1990	1.40 (0.79, 2.51)	0.39 (0.02, 7.52)	0.80 (0.12, 5.31)	0.78 (0.33, 1.82)
Age at First Birth				
14-19 years old	0.84 (0.60, 1.16)	1.36 (0.52, 3.56)	0.75 (0.48, 1.18)	0.62 (0.30, 1.27)
20-29 years old	1.00	1.00	1.00	1.00
30-41 years old	0.87 (0.66, 1.15)	0.90 (0.41, 1.98)	0.84 (0.58, 1.22)	1.26 (0.70, 2.25)
Parental Education at Wave I				
<HS	1.05 (0.68, 1.64)	0.74 (0.28, 1.95)	1.06 (0.55, 2.04)	1.07 (0.41, 2.78)
HS Diploma	1.09 (0.81, 1.48)	0.72 (0.30, 1.71)	1.18 (0.82, 1.70)	0.89 (0.45, 1.76)
Some College	0.99 (0.68, 1.45)	0.90 (0.29, 2.75)	1.03 (0.62, 1.72)	1.05 (0.52, 2.11)
College+	1.00	1.00	1.00	1.00

**Predicted Prevalence (95% CIs).*

A3.13. Unadjusted Prevalence Ratios for Experiences of Preterm Birth by District-Level School Segregation – Black-White Dissimilarity Index – during Adolescence (Wave I, 1994-1995) and Individual Race/Ethnicity, among Add Health Participants who had a First Birth by Wave V (2016-2018).

	All Females	Hispanic	Non-Hispanic White	Non-Hispanic Black
Dissimilarity Index	0.98 (0.55, 1.75)	0.04 (0.28, 3.13)	0.85 (0.31, 2.30)	0.59 (0.23, 1.50)
0.05*	0.14 (0.12, 0.17)	0.16 (0.09, 0.25)	0.14 (0.11, 0.17)	0.19 (0.13, 0.26)
0.20*	0.14 (0.13, 0.16)	0.16 (0.11, 0.22)	0.14 (0.12, 0.16)	0.18 (0.14, 0.23)
0.40*	0.14 (0.13, 0.16)	0.15 (0.12, 0.20)	0.13 (0.11, 0.16)	0.16 (0.13, 0.20)
0.60*	0.14 (0.12, 0.17)	0.14 (0.11, 0.21)	0.13 (0.09, 0.18)	0.15 (0.11, 0.19)
0.80*	0.14 (0.11, 0.18)	0.15 (0.10, 0.23)	0.13 (0.08, 0.20)	0.14 (0.09, 0.20)

**Predicted Prevalence (95% CIs).*

A3.14. Adjusted Prevalence Ratios for Experiences of Preterm Birth by District-Level School Segregation – Black-White Dissimilarity Index – during Adolescence (Wave I, 1994-1995) and Individual Race/Ethnicity, among Add Health Participants who had a First Birth by Wave V (2016-2018).

	All Females	Hispanic	Non-Hispanic White	Non-Hispanic Black
Dissimilarity Index	0.70 (0.36, 1.38)	0.62 (0.12, 3.27)	0.61 (0.20, 1.87)	0.40 (0.13, 1.25)
0.05*	0.16 (0.14, 0.20)	0.18 (0.10, 0.32)	0.16 (0.12, 0.20)	0.22 (0.15, 0.32)
0.20*	0.16 (0.14, 0.18)	0.17 (0.11, 0.27)	0.15 (0.13, 0.17)	0.20 (0.15, 0.26)
0.40*	0.15 (0.13, 0.16)	0.16 (0.12, 0.21)	0.14 (0.12, 0.16)	0.17 (0.14, 0.21)
0.60*	0.14 (0.12, 0.17)	0.15 (0.11, 0.20)	0.13 (0.09, 0.18)	0.15 (0.11, 0.19)
0.80*	0.13 (0.10, 0.17)	0.14 (0.08, 0.23)	0.12 (0.07, 0.19)	0.13 (0.08, 0.19)
School Size				
1 – 400 students	1.27 (0.93, 1.72)	3.43 (1.52, 7.75)	1.33 (0.92, 1.93)	0.87 (0.50, 1.51)
401 – 1,000 students	0.94 (0.73, 1.20)	0.82 (0.38, 1.76)	1.09 (0.79, 1.49)	0.70 (0.41, 1.17)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.95 (0.73, 1.24)	5.30 (1.13, 24.77)	0.81 (0.58, 1.13)	1.41 (0.71, 2.78)
Suburban	0.94 (0.74, 1.19)	4.84 (1.00, 23.49)	0.79 (0.60, 1.04)	1.66 (0.96, 2.88)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.23 (0.89, 1.70)	0.47 (0.23, 0.94)	1.53 (0.88, 2.64)	0.92 (0.38, 2.21)
Private	1.00	1.00	1.00	1.00

**Predicted Prevalence (95% CIs).*

A3.15. Adjusted Prevalence Ratios for Experiences of Preterm Birth by District-Level School Segregation – Black-White Dissimilarity Index – during Adolescence (Wave I, 1994-1995) and Individual Race/Ethnicity, among Add Health Participants who had a First Birth by Wave V (2016-2018)

	All Females	Hispanic	Non-Hispanic White	Non-Hispanic Black
Dissimilarity Index	0.69 (0.34, 1.40)	0.57 (0.10, 3.26)	0.60 (0.19, 1.96)	0.40 (0.10, 1.56)
0.05*	0.17 (0.14, 0.20)	0.19 (0.10, 0.33)	0.16 (0.12, 0.21)	0.22 (0.14, 0.32)
0.20*	0.16 (0.14, 0.18)	0.18 (0.11, 0.27)	0.15 (0.13, 0.18)	0.20 (0.15, 0.26)
0.40*	0.15 (0.13, 0.16)	0.16 (0.12, 0.21)	0.14 (0.12, 0.17)	0.17 (0.14, 0.21)
0.60*	0.14 (0.12, 0.17)	0.15 (0.10, 0.20)	0.13 (0.09, 0.18)	0.15 (0.11, 0.20)
0.80*	0.13 (0.10, 0.18)	0.14 (0.08, 0.23)	0.12 (0.07, 0.20)	0.13 (0.07, 0.21)
School Size				
1 – 400 students	1.29 (0.93, 1.80)	3.52 (1.61, 7.70)	1.33 (0.88, 2.01)	1.00 (0.55, 1.84)
401 – 1,000 students	0.94 (0.72, 1.21)	0.83 (0.39, 1.77)	1.10 (0.78, 1.53)	0.70 (0.40, 1.20)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.93 (0.71, 1.22)	5.96 (1.30, 27.37)	0.79 (0.55, 1.14)	1.40 (0.73, 2.69)
Suburban	0.91 (0.71, 1.17)	5.68 (1.10, 29.22)	0.77 (0.58, 1.04)	1.67 (1.00, 2.78)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.27 (0.91, 1.77)	0.45 (0.22, 0.92)	1.54 (0.88, 2.69)	1.10 (0.44, 2.75)
Private	1.00	1.00	1.00	1.00
Neighborhood Poverty, 1990	0.62 (0.18, 2.14)	2.26 (0.07, 76.55)	0.98 (0.14, 6.83)	0.22 (0.03, 1.66)
Neighborhood Proportion Black, 1990	1.34 (0.83, 2.16)	0.69 (0.04, 12.63)	1.13 (0.24, 5.23)	1.21 (0.45, 3.26)

**Predicted Prevalence (95% CIs).*

A3.16. Adjusted Prevalence Ratios for Experiences of Preterm Birth by District-Level School Segregation – Black-White Dissimilarity Index – during Adolescence (Wave I, 1994-1995) and Individual Race/Ethnicity, among Add Health Participants who had a First Birth by Wave V (2016-2018).

	All Females	Hispanic	Non-Hispanic White	Non-Hispanic Black
Dissimilarity Index	0.72 (0.33, 1.55)	0.54 (0.06, 5.37)	0.73 (0.21, 2.54)	0.62 (0.12, 3.12)
0.05*	0.15 (0.12, 0.18)	0.17 (0.07, 0.33)	0.14 (0.10, 0.18)	0.20 (0.13, 0.30)
0.20*	0.14 (0.13, 0.16)	0.16 (0.09, 0.27)	0.13 (0.11, 0.16)	0.19 (0.15, 0.25)
0.40*	0.14 (0.12, 0.15)	0.14 (0.09, 0.21)	0.13 (0.11, 0.15)	0.18 (0.14, 0.22)
0.60*	0.13 (0.10, 0.16)	0.13 (0.08, 0.21)	0.12 (0.08, 0.17)	0.16 (0.11, 0.24)
0.80*	0.12 (0.09, 0.17)	0.12 (0.05, 0.25)	0.11 (0.06, 0.20)	0.15 (0.08, 0.28)
School Size				
1 – 400 students	1.31 (0.93, 1.86)	3.79 (1.25, 11.52)	1.29 (0.79, 2.09)	1.07 (0.56, 2.06)
401 – 1,000 students	1.02 (0.78, 1.33)	0.80 (0.30, 2.15)	1.10 (0.75, 1.60)	1.11 (0.69, 1.81)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.095 (0.68, 1.31)	4.82 (0.93, 25.05)	0.82 (0.51, 1.32)	1.97 (0.97, 4.03)
Suburban	0.91 (0.70, 1.19)	5.01 (0.88, 28.49)	0.78 (0.56, 1.09)	2.17 (1.21, 3.89)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.16 (0.85, 1.60)	0.33 (0.15, 0.71)	1.43 (0.78, 2.62)	1.59 (0.56, 4.56)
Private	1.00	1.00	1.00	1.00
Neighborhood Poverty, 1990	0.39 (0.08, 1.86)	6.23 (0.12, 316.92)	0.49 (0.03, 7.41)	0.37 (0.04, 3.42)
Neighborhood Proportion Black, 1990	1.81 (1.07, 3.06)	0.18 (0.00, 22.25)	1.42 (0.25, 7.91)	0.99 (0.34, 2.82)
Age at First Birth				
14-19 years old	0.82 (0.58, 1.15)	1.16 (0.42, 3.22)	0.77 (0.50, 1.19)	0.56 (0.27, 1.16)
20-29 years old	1.00	1.00	1.00	1.00
30-41 years old	0.85 (0.64, 1.13)	0.83 (0.41, 1.65)	0.83 (0.57, 1.21)	1.22 (0.70, 2.12)
Parental Education at Wave I				
<HS	1.08 (0.69, 1.68)	0.69 (0.27, 1.78)	0.99 (0.51, 1.91)	1.18 (0.48, 2.91)
HS Diploma	1.09 (0.81, 1.46)	0.76 (0.36, 1.62)	1.13 (0.79, 1.62)	0.88 (0.45, 1.75)
Some College	0.92 (0.63, 1.35)	0.90 (0.27, 2.94)	0.90 (0.53, 1.52)	1.08 (0.55, 2.11)
College+	1.00	1.00	1.00	1.00

**Predicted Prevalence (95% CIs).*

A3.17. Unadjusted Prevalence Ratios for Experiences of Preterm Birth by District-Level School Segregation – Black-White Exposure Index – during Adolescence (Wave I, 1994-1995) and Individual Race/Ethnicity, among Add Health Participants who had a First Birth by Wave V (2016-2018).

	All Females	Hispanic	Non-Hispanic White	Non-Hispanic Black
Exposure Index	0.94 (0.67, 1.32)	0.59 (0.26, 1.35)	1.14 (0.69, 1.87)	2.20 (1.02, 4.73)
0.10*	0.15 (0.12, 0.18)	0.19 (0.13, 0.27)	0.13 (0.09, 0.17)	0.13 (0.10, 0.17)
0.25*	0.15 (0.12, 0.17)	0.18 (0.13, 0.24)	0.13 (0.10, 0.17)	0.15 (0.12, 0.18)
0.50*	0.14 (0.13, 0.16)	0.16 (0.12, 0.21)	0.13 (0.11, 0.16)	0.17 (0.14, 0.21)
0.75*	0.14 (0.13, 0.16)	0.14 (0.10, 0.20)	0.14 (0.12, 0.15)	0.20 (0.15, 0.27)
0.90*	0.14 (0.12, 0.16)	0.13 (0.09, 0.20)	0.14 (0.12, 0.16)	0.22 (0.15, 0.32)

**Predicted Prevalence (95% CIs).*

A3.18. Adjusted Prevalence Ratios for Experiences of Preterm Birth by District-Level School Segregation – Black-White Exposure Index – during Adolescence (Wave I, 1994-1995) and Individual Race/Ethnicity, among Add Health Participants who had a First Birth by Wave V (2016-2018).

	All Females	Hispanic	Non-Hispanic White	Non-Hispanic Black
Exposure Index	1.07 (0.76, 1.51)	0.87 (0.27, 2.83)	1.30 (0.79, 2.15)	2.36 (1.09, 5.14)
0.10*	0.15 (0.12, 0.18)	0.17 (0.11, 0.26)	0.13 (0.09, 0.17)	0.14 (0.11, 0.18)
0.25*	0.15 (0.13, 0.17)	0.17 (0.11, 0.24)	0.13 (0.10, 0.17)	0.16 (0.13, 0.19)
0.50*	0.15 (0.14, 0.17)	0.16 (0.12, 0.22)	0.14 (0.12, 0.16)	0.19 (0.15, 0.23)
0.75*	0.15 (0.14, 0.17)	0.16 (0.11, 0.23)	0.15 (0.13, 0.17)	0.22 (0.16, 0.30)
0.90*	0.15 (0.14, 0.17)	0.16 (0.09, 0.25)	0.15 (0.13, 0.17)	0.24 (0.16, 0.35)
School Size				
1 – 400 students	1.29 (0.94, 1.76)	3.63 (1.66, 7.93)	1.39 (0.96, 2.01)	0.92 (0.53, 1.60)
401 – 1,000 students	0.96 (0.75, 1.22)	0.88 (0.39, 1.95)	1.13 (0.83, 1.53)	0.76 (0.46, 1.26)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.94 (0.72, 1.22)	4.87 (0.95, 24.95)	0.83 (0.59, 1.17)	1.27 (0.66, 2.43)
Suburban	0.93 (0.74, 1.17)	4.75 (0.94, 23.95)	079 (0.60, 1.04)	1.47 (0.90, 2.42)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.24 (0.90, 1.70)	0.47 (0.23, 0.95)	1.59 (0.95, 2.66)	0.82 (0.38, 1.77)
Private	1.00	1.00	1.00	1.00

**Predicted Prevalence (95% CIs).*

A3.19. Adjusted Prevalence Ratios for Experiences of Preterm Birth by District-Level School Segregation – Black-White Exposure Index – during Adolescence (Wave I, 1994-1995) and Individual Race/Ethnicity, among Add Health Participants who had a First Birth by Wave V (2016-2018).

	All Females	Hispanic	Non-Hispanic White	Non-Hispanic Black
Exposure Index	1.15 (0.75, 1.77)	0.83 (0.27, 2.58)	1.37 (0.81, 2.29)	2.50 (0.78, 7.97)
0.10*	0.14 (0.11, 0.18)	0.17 (0.11, 0.26)	0.12 (0.09, 0.17)	0.14 (0.09, 0.20)
0.25*	0.15 (0.12, 0.17)	0.17 (0.12, 0.24)	0.13 (0.10, 0.17)	0.15 (0.12, 0.20)
0.50*	0.15 (0.13, 0.17)	0.16 (0.12, 0.22)	0.14 (0.12, 0.16)	0.19 (0.15, 0.23)
0.75*	0.15 (0.14, 0.17)	0.16 (0.11, 0.23)	0.15 (0.13, 0.17)	0.22 (0.15, 0.31)
0.90*	0.16 (0.14, 0.18)	0.15 (0.09, 0.24)	0.15 (0.13, 0.18)	0.25 (0.15, 0.38)
School Size				
1 – 400 students	1.33 (0.96, 1.84)	3.73 (1.75, 7.92)	1.41 (0.94, 2.13)	1.02 (0.56, 1.85)
401 – 1,000 students	0.96 (0.75, 1.23)	0.89 (0.40, 1.94)	1.14 (0.83, 1.57)	0.75 (0.45, 1.24)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.92 (0.70, 1.21)	5.08 (0.99, 26.18)	0.80 (0.56, 1.15)	1.24 (0.66, 2.34)
Suburban	0.90 (0.71, 1.15)	5.04 (0.94, 26.98)	0.77 (0.57, 1.03)	1.46 (0.90, 2.36)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.28 (0.92, 1.78)	0.46 (0.22, 0.95)	1.63 (0.96, 2.76)	0.93 (0.41, 2.12)
Private	1.00	1.00	1.00	1.00
Neighborhood Poverty, 1990	0.58 (0.17, 2.00)	1.36 (0.04, 43.97)	0.78 (0.12, 5.07)	0.25 (0.04, 1.68)
Neighborhood Proportion Black, 1990	1.41 (0.85, 2.33)	0.61 (0.04, 8.32)	1.68 (0.37, 7.63)	1.44 (0.50, 4.13)

**Predicted Prevalence (95% CIs).*

A3.20. Adjusted Prevalence Ratios for Experiences of Preterm Birth by District-Level School Segregation – Black-White Exposure Index – during Adolescence (Wave I, 1994-1995) and Individual Race/Ethnicity, among Add Health Participants who had a First Birth by Wave V (2016-2018).

	All Females	Hispanic	Non-Hispanic White	Non-Hispanic Black
Exposure Index	1.10 (0.69, 1.75)	0.30 (0.06, 1.49)	1.31 (0.73, 2.36)	1.47 (0.30, 7.16)
0.10*	0.13 (0.10, 0.17)	0.22 (0.11, 0.40)	0.11 (0.08, 0.16)	0.16 (0.10, 0.26)
0.25*	0.14 (0.11, 0.16)	0.20 (0.11, 0.33)	0.12 (0.09, 0.16)	0.17 (0.12, 0.23)
0.50*	0.14 (0.12, 0.16)	0.16 (0.10, 0.24)	0.12 (0.10, 0.15)	0.18 (0.15, 0.23)
0.75*	0.14 (0.13, 0.16)	0.13 (0.07, 0.21)	0.13 (0.11, 0.15)	0.20 (0.13, 0.30)
0.90*	0.14 (0.12, 0.16)	0.11 (0.06, 0.20)	0.14 (0.12, 0.16)	0.21 (0.11, 0.36)
School Size				
1 – 400 students	1.34 (0.95, 1.89)	4.11 (1.41, 12.03)	1.35 (0.83, 2.19)	1.10 (0.57, 2.12)
401 – 1,000 students	1.05 (0.81, 1.35)	1.00 (0.35, 2.86)	1.14 (0.79, 1.62)	1.16 (0.72, 1.85)
1,001 – 4,000 students	1.00	1.00	1.00	1.00
School Urbanicity				
Urban	0.94 (0.67, 1.31)	2.86 (0.41, 19.83)	0.83 (0.52, 1.35)	1.87 (0.91, 3.85)
Suburban	0.90 (0.69, 1.18)	3.42 (0.56, 20.90)	0.78 (0.55, 1.10)	2.04 (1.12, 3.73)
Rural	1.00	1.00	1.00	1.00
School Type				
Public	1.17 (0.86, 1.61)	0.37 (0.17, 0.81)	1.49 (0.85, 2.62)	1.49 (0.54, 4.14)
Private	1.00	1.00	1.00	1.00
Neighborhood Poverty, 1990	0.37 (0.08, 1.72)	1.30 (0.04, 40.97)	0.43 (0.04, 5.23)	0.38 (0.05, 3.13)
Neighborhood Proportion Black, 1990	1.86 (1.04, 3.34)	0.08 (0.00, 9.32)	1.91 (0.34, 10.76)	1.04 (0.31, 3.50)
Age at First Birth				
14-19 years old	0.82 (0.58, 1.15)	1.28 (0.45, 3.59)	0.77 (0.50, 1.18)	0.57 (0.28, 1.17)
20-29 years old	1.00	1.00	1.00	1.00
30-41 years old	0.85 (0.64, 1.13)	0.78 (0.41, 1.49)	0.83 (0.57, 1.21)	1.21 (0.70, 2.11)
Parental Education at Wave I				
<HS	1.08 (0.69, 1.68)	0.66 (0.25, 1.74)	0.99 (0.52, 1.91)	1.19 (0.50, 2.86)
HS Diploma	1.09 (0.81, 1.46)	0.76 (0.37, 1.55)	1.13 (0.79, 1.61)	0.89 (0.45, 1.77)
Some College	0.92 (0.63, 1.35)	0.91 (0.26, 3.10)	0.90 (0.52, 1.53)	1.06 (0.53, 2.14)
College+	1.00	1.00	1.00	1.00

**Predicted Prevalence (95% CIs).*

Not So Black and White: The Racial/Ethnic Structure of School Environments and Cardiometabolic and Reproductive Health across the Life Course

Chapter 5: Conclusion

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CONCLUSION

This dissertation investigated the impact of the racial/ethnic structure of school environments on three distinct, yet connected, health outcomes that occur across the life course. Across all three Specific Aims, we found that there were racial/ethnic differences in the types of schools attended by Add Health participants during adolescence. Overall, Hispanic and Asian/Native American/Other adolescents were most likely to attend racially diverse schools; non-Hispanic White adolescents were most likely to attend predominantly White schools; and non-Hispanic Black adolescents were most likely to attend predominantly Black/non-White schools and schools within racially-segregated districts. These disparities suggest that each of the racial/ethnic groups investigated in this dissertation were a part of distinct school environments with unique racial structures.

Aim 1 found that students identifying as Asian/Native American/Other who attended schools with moderate levels of Black students were more likely to be overweight/obese, compared to those who attended schools with the lowest levels of Black students. Also, non-Hispanic Black students who attended schools with moderate levels of non-White students were more likely to be overweight/obese, compared to those who attended schools with the lowest levels of non-White students. It is possible that schools with moderate levels of Black and non-White schools consist of predominantly White students. This racial imbalance could contribute to experiences of racial/ethnic discrimination for Asian/Native American/Other and non-Hispanic Black adolescents. Ultimately, the findings from this dissertation suggest that the racial

structure of these school environments could impact the prevalence of adolescent obesity.

Aim 2 found that there were no meaningful differences by race/ethnicity in the associations between the racial/ethnic structure of school environments and inflammation in early adulthood. In additional analyses, we found that there were significant associations between the proportion of non-White students within a school and a higher prevalence of inflammation among Hispanic participants; as well as, between district-level segregation (Black-White exposure index) and a higher prevalence of inflammation among non-Hispanic Black participants. However, it is important to note that these associations were attenuated after adjusting for neighborhood-level characteristics. These findings suggest that adverse socioeconomic characteristics within a neighborhood (i.e., poverty and racial composition) could explain the relationship between the racial structure of school environments and inflammation.

Finally, Aim 3 found that for every one-unit change in the proportion of Black students in a school (from 0 to 100%), Hispanic females had a significantly lower prevalence of PTB. In fact, we observed a significantly lower predicted prevalence of PTB among Hispanic mothers who attended schools with 90% Black students, compared those who attended schools with 10% Black students. This finding was unexpected. It is possible that attending schools with a higher proportion of Black students during adolescence is protective for Hispanic mothers because there is the potential for less racial/ethnic discrimination. In addition, similar to the findings from Aim 2, the association between district-level segregation (Black-White exposure index) and the prevalence of PTB among non-Hispanic Black mothers was attenuated after adjusting for neighborhood-level characteristics. Additional research is warranted that

explores the role of these adverse socioeconomic characteristics in the structure of school environments.

This dissertation proved to be innovative in that we examined three aspects of the racial/ethnic structure of school environments among a nationally-representative cohort: (1) the racial composition of schools, (2) school diversity levels, and (3) district-level segregation. The findings from these studies suggest that the absolute levels of school racial composition, and not district-level segregation, is an important risk factor in the development of cardiometabolic and reproductive outcomes that occur across the life course. It is possible that the individual school environments, and not necessarily the spatial processes operating at the district-level, are critical exposures, which experienced during adolescence can contribute to adverse outcomes in adolescence and adulthood.

This dissertation has important implications for both the construction of educational policies, as well as its applications to social epidemiologic literature. First, there have been large racial gaps in academic achievement that have persisted since 1990[1]. The resegregation of U.S. schools that occurred in the 1990s and the policies that have reversed the *Brown* decision have not been effective in achieving racial equality in education. However, very little is known about the health consequences of these inequalities. This dissertation has attempted to address this gap in the literature, but more work is needed to investigate how the racial/ethnic structure of school environments is a relevant and plausible social determinant of health.

It is important to note that the findings from this dissertation work suggest that the racial/ethnic structure of school environments does not impact the cardiometabolic and reproductive health of non-Hispanic White participants in Add Health. However,

the racial/ethnic composition of schools and district-level segregation proved to important risk factors for the cardiometabolic and reproductive health of persons of color. Thus, more research is needed that incorporates additional school segregation indices that assess the levels of segregation between students from different racial/ethnic backgrounds. This will be critical in the coming years, given that roughly 50% of Americans are persons of color[2]. Finally, future social epidemiologic literature should investigate some of the mediating factors, such as economic segregation (in terms of resources distributed within and across districts) and racial/ethnic discrimination experienced by students, that could explain the relationship between the racial/ethnic structure of school environments and cardiometabolic and reproductive health across the life course.

To our knowledge, this was one of the first extensive studies to examine the health impacts of both the racial/ethnic composition within schools, as well as the spatial processes of race across schools within a district. Many social epidemiologic studies have investigated the cross-sectional effects of contextual exposures, or during one specific period of the life course. This dissertation was novel in that we examined the effects of the distribution of racial groups within school and within districts on three periods of the life course. In doing so, we have begun to understand the long-term impacts of this social determinant of population health. Given the large public health burden of the three outcomes in this dissertation, these findings can help researchers understand the context and potential causes of the persistent disparities associated with obesity, inflammation, and PTB. The recent resegregation of public schools in the U.S. has made the structure of schools and school systems a timely and relevant exposure

that will allow us to continue to investigate the social, economic, and political contexts that impact adolescents across the life course.

REFERENCES

1. Harris D, Herrington C. Accountability, standards, and the growing achievement gap: Lessons from the past half-century. *American Journal of Education*. 2006; 112(2):209-38.
2. Mitchell DE, Crowson RL, Shipps D (eds.). *Shaping Education Policy: Power and Process*. Routledge: New York, NY; 2011.