Equal Protection Under the Law: The Measurement of Structural Racism and Health Disparities

by

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Dedication

To an unconventional mother from an unconventional daughter, thank you for teaching me to value education and how to reinvent yourself at any age. To my husband Jason thank you for your sacrifices and unwavering support as I pursued this degree, this achievement could not have been earned without you—I love you. To Charlotte, you will someday appreciate the sacrifice we made to accomplish this dissertation, as I did with my mom. I will be there for you when you accomplish your goals, as you were as I accomplished mine. To my family all over the country, thank you for believing in me and keeping me true to my goals. I especially want to thank my brother Dan Santefort for teaching me how to fight and to have "thick skin" in the face of adversity. I also want to thank Bob Newman for giving me the chance to be a city kid and exposing me to the great city of Chicago, without that move I am not sure I would be here today. To Mary Allgood-Johnson, without your love and help this would never have been possible. To Dr. Steve Whitman, thank you for teaching me to fight for what is right and to "seek truth" and understand the past to solve problems occurring in the present. I will never forget the lessons you taught me and never stop the pursuit for health equity. To my SUHI family, thank you for nurturing my professional growth and intellectual curiosity. I greatly appreciate your dedication to helping the community. Finally, to Belinda Needham, Nancy Fleischer, and Sara Adar, thank you for being such great women, scholars, mothers, and strong shoulders, without you I cannot imagine this life change occurring so smoothly.

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List of Abbreviations

Abbreviation	Definition
A1c	Hemoglobin A1C
ACASI	Audio Computer Assisted Self-Interview
ACTH	Adrenocorticotropic hormones
Add Health	National Longitudinal Study of Adolescent to Adult Health
aOR	Adjusted Odds Ratio
AZ	Arizona
BMI	Body Mass Index
BW	Birthweight
CAPI	Computer Assisted Personal Interviewing
CDC	Centers for Disease Control and Prevention
CI	Confidence Interval
CIA	Central Intelligence Agency
CVD	Cardiovascular Disease
DAG	Directed acyclic graph
DC	District of Columbia
DID	Difference-in-Difference
dL	Deciliter
EPA	Environmental Protection Agency
exp	Exponent/anti-log
FBI	Federal Bureau of Investigation
FHA	Federal Housing Authority
FL	Florida
FPL	Federal Poverty Level
FWC	Flint Water Crisis
FWC declaration	State of emergency declaration in Flint due to the FWC
GA	Gestational Age
GED	General Educational Development
GEE	General Estimating Equations
GI	Government Issue
GM	General Motors
HS	High School
HUD	Housing and Urban Development

Abbreviation	Definition
IA	Iowa
ICD	International Classification of Diseases
ICE	Index of Concentration of Extremes
ID	Identification
IoD	Index of Dissimilarity
IoI	Index of Isolation
km	kilometer
LA	Louisiana
LBW	Low Birth Weight
LMP	Last menstrual period
MDHHS	Michigan Department of Health and Human Services
mg	Milligrams
MI	Myocardial Infarction
NHB	Non-Hispanic Black
NHW	Non-Hispanic White
NY	New York
NYC	New York City
NYPD	New York Police Department
PD	Police Department
PE	Police Encounter
PTB	Preterm Birth
PTSD	Post-Traumatic Stress Disorder
ref	Reference
SCOTUS	Supreme Court of The United States
SD	Standard Deviation
SE	Standard Error
SES	Socioeconomic Status
SzGA	Size-for-Gestational Age
SmGA	Small-for-Gestational Age
STI	Sexually Transmitted Infections
US	United States
VA	Virginia
W#	Wave I-IV or Wave 1-4
WIC	Women, Infant, and Children Public Assistance Program
WWII	World War II
yrs	years

Abstract

This dissertation focuses on measuring the health effects of exposure to structural racism, or racism that forms through historic and contemporary policies that produce a disparate impact by race. After defining various forms of racism and describing how structural racism relates to health outcomes, I examine three distinct measures of exposure to structural racism arising from housing and criminal justice policies in relation to birth outcomes (Aim 1) and 30-year cardiovascular risk (Aims 2 and 3). In each of these studies I hypothesize that exposure to structural racism will be more harmful to Blacks than Whites. This hypothesis is grounded in research and theory suggesting that, despite color-blind policies that on the surface appear to affect all people in the same way, the racially disparate impacts of the policies are simultaneously privileging Whites while disadvantaging Blacks. The first measure of structural racism is *in utero* exposure to the Flint Water Crisis (FWC) emergency declaration, which is considered to be a racialized stressor due to Flint's long history of racial segregation. The sample includes Michigan women (outside of Flint) who were pregnant before and during the declaration of a State of Emergency. Here I examine the relationship between the FWC declaration and birth outcomes between babies born to Black and White mothers using a before and after linear regression analytic methodology. I find that both babies born to Black and White mothers after the emergency declaration have lower birthweight, gestational age, and size-for gestational age compared to babies born before the declaration, but exposed babies born to Black mothers have marginally significantly greater decline in gestational age compared to exposed babies born to White mothers. The second measurement is direct exposure to the police through self-reported police encounters. Data are from a nationally representative sample of young adults interviewed during the War on Drugs, which included foundational policies that created incarceration disparities in the US. Using a standard linear regression with domain analysis, I examine the association between sex-specific levels of police encounters and the Framingham 30-year cardiovascular risk score, which predicts a major cardiovascular event occurring in the next 30 years. I find that the relationship between police encounters and

cardiovascular risk differs by race, where a high level of police encounters is associated with higher risk for cardiovascular disease among Whites but lower risk for cardiovascular disease among Blacks. The final measure of structural racism is community- and school-level segregation, using standard measures of residential segregation and a novel measure of school segregation. Data are from a nationally representative sample of adolescents entering adulthood. Here I examine the association between residential and school segregation and the Framingham 30-year cardiovascular risk score using a general estimating equations analytic methodology. I find that a higher level of residential segregation measured with the Index of Concentration of Extremes is associated with an increased cardiovascular disease risk for both Blacks and Whites, but the association is stronger for Blacks. In addition, I find that a higher level of school segregation measured with the Index of Concentration of Extremes is associated with increased cardiovascular disease risk in Blacks but decreased risk in Whites. Overall, I find mixed support for the central study hypothesis. I conclude with suggestions for future research and policy considerations.

Chapter 1 Introduction – Structural Racism and Health Outcomes

"All persons born or naturalized in the United States, and subject to the jurisdiction thereof, are citizens of the United States and the State wherein they reside. No State shall make or enforce any law which shall abridge the privileges or immunities of citizens of the United States; nor shall any State deprive any person of life, liberty, or property, without due process of law; nor deny to any person within its jurisdiction the *equal protection of the laws*."

~Section 1, 14th Amendment, US Constitution, 1866

1.1 Background

Several recent city- and community-level mortality analyses demonstrate that racial disparities have persisted or even worsened over the last 20 years in the United States (US.)[1-5] Sociologic and epidemiologic research suggest that racism is at the root of these disparities.[6-18] Racism is defined as an ideology that ranks social groups hierarchically according to race, a socially defined classification based on physical features.[19,20] Structural racism, the most pervasive form of racism, results from policies that create structural barriers which limit (unintentionally or intentionally) minority groups' access to power and resources while simultaneously privileging Whites (unintentionally or intentionally).[6,13,15,21] While acts of interpersonal racism can be observed, structural racism is embedded into our laws and social order, making it difficult to assess.[13]

1.2 Race and Racism

Race is a social construct that is extremely complex.[22,23] It differs from culture and ethnicity, although these are often conflated with race.[20] On the surface race is a construct that organizes people based on physical characteristics.[19,20] Race is an unusual social construct, as opposed to purely biological or genetic.[22,24] Race is an "unusual" construct because while we often define race based on arbitrary physical characteristics, race is often conflated with other constructs like

racism because, at least in countries that were colonized by European nations, race carries the weight of other social issues such as racism or poverty.[20,25] However, to understand how people differ physically, early historians and philosophers defined the Black race with some sort of biologic inferiority because of how slaves were forced to live[20,26-28] and later based on how Blacks lived in modern times without taking into account the government's role in creating those circumstances.[29,30] Throughout the history of race in America, we began a process of reification, or making an abstract concept (i.e., race) into something real.[14,31,32] These beliefs became common in medical school teachings--even today.[33] Additionally, these debunked scientifically racist studies are often cited by White supremacists of the past and present.[20,34]

Race implies a social stratification and as a concept is difficult to decouple with racism due to the substantial racist history of this country that continues to persist.[26] This social stratification was born out of the need to maintain slavery to ensure the economic future of the fledgling country.[26] While a concept of racism likely existed across the globe as powerful countries conquered nations with fewer resources and power, often meeting people with very different appearances and cultures, it became entrenched into early American life as a means to justify the treatment of Blacks and American Indians (and Latinos, Southern and Eastern Europeans, and Asians).[26] Anti-Black racism, however, has been codified into our social and legal structures through very early policies written into the US Constitution to concentrate power in the southern states in order to ensure the protection of slavery (U.S. Const. art. 1, § 2). This portion of the Enumeration Clause was eliminated with the passage of the 14th Amendment, however, by then the damage had been done and Black Americans have had to endure the effects of being placed on a lower rung on the racial stratification ladder ever since.

Much of the discrimination Blacks face in the modern US is based on laws, policies, and customs, called structural or institutional racism.[6,7,13,15,16] So, what is racism and where does it come from? Racism is a state of mind in which one believes, and often acts, as if his or her race is superior to another race.[15] This definition of racism, which emphasizes interpersonal discrimination, leaves out much of the codified racism in the social order that is beyond "bad apples" or individual actors being racists to another person. Human beings defined race, created the social rankings, and acted upon these social rankings by establishing rules.[15,20,26,31,35,36] The rules were developed to favor those in power, and in the US, that meant Northern European

Whites and their descendants.[26,35] The rules and social stratification and thus the treatment of non-Whites were justified by the most respected scholars and clergy of the time,[20,26] thereby forgiving slaveholders for the cruelty of slavery. Whites depended on slavery for economic freedom from the tether of the English crown. Without financial independence from England, the US would never have been an independent nation. Slaves were a major part of the growing US economy.[26,37]

While the economy and wealth accumulated as slavery grew, so did the hateful treatment toward Blacks in the US.[38] Many of the early US policies around race relations were aimed to keep slaves "in-line." As time went on, new policies and laws were born from old versions that were either overtly racist (e.g., slave patrols) or covertly racist (e.g., vagrancy laws) and produced disparate outcomes by socially defined racial groups.[35]

Modern scholars have defined four forms or levels of racism.[13,39] Structural racism is the most pervasive form of racism as it is cooked into our everyday life. There is not one single force establishing or imposing it on others, it is how we live and exist together.[6,10-13,15,16,18-20,39-45] Structural racism refers to a *system* of policies and actions that are created or "structured" to both oppress some while privileging others.[7,39] While structural racism and institutional racism are often confused with each other, the difference is nuanced. Institutional racism is formed when our institutions (education, criminal justice, among others) build themselves in a way to create "barriers" for racial and ethnic minorities in a race-neutral way (but not always), again by privileging Whites while harming Blacks.[39,46] This is opposed to multiple-system structural racism which may also include cultural racism, which is defined as discrimination directed toward differences in cultures, the basis for racially/culturally motivated stereotypes.[47] The next level of racism is interpersonal discrimination. This form of racism is what the average American understands as acts of racism. This widely studied form of racism is observable as it requires an offensive action or statement that indicates racial superiority, even if in a seemingly innocuous way (e.g., microaggressions), as well as the victim perceiving the treatment as racist.[25,48-59] The final form of racism is called internalized racism which occurs when a racial minority believes in the stereotypes, microaggressions, and racism of the group with which they identify as inferior and those who are of a majority racial identity are superior.[10,13,25,39,60] All of

these forms of racism have consequences for mental and physical health for those directly impacted[61-73] as well as those indirectly or vicariously impacted.[74-77,79]

To date, racism research has largely focused on experiences of discrimination or interpersonal racism.[10-12,17,18] This body of work has demonstrated that racism affects numerous health outcomes.[80-85] A smaller body of literature has recently emerged which focuses on health outcomes resulting from structural forms of racism.[75,77,86-93] For example, Lukachko et. al.[77] reported that Blacks living in states with high levels of structural racism, defined as the racial disparities in the effects of historic policies (e.g., racial disparities in educational attainment, incarceration, and employment), had a higher odds of reporting past year myocardial infarction (MI) than Blacks living in states with lower levels of structural racism, whereas Whites living in high racism states had the same or lower odds of MI compared to Whites in low racism states. Other studies have indicated that abolishing overtly racist US policies that target Blacks can improve the health of the Black population.[92] Despite the large body of literature devoted to interpersonal racism, many unanswered questions remain about the health consequences of exposure to structural racism. For example, a large body of research suggests that residential segregation is associated with a myriad of health conditions.[94] Residential segregation is a classic example of structural racism, a structure generated through a policy (1934 Homeowners Loan Act – New Deal), written as a color-blind policy, but executed in an overtly racist fashion thus creating a disparate outcome in homeownership. Additionally, segregation affects other institutions such as education and economic resources. Current research suggests that redlining maps drawn in the 1930's still affect health today.[62,68,95-101] Some argue that this is a result of interpersonal discrimination[102], and perhaps in some individual cases that is true, but a map drawn in the 1930s provides little evidence of individually directed racism today, since it affects the entire community. It is this concept that is understudied. How can dated bank loan maps still affect the health of residents in those communities today? How does a criminal justice system, born from slave patrols, affect the health of those directly and indirectly affected? How does living, learning and playing in an area with differential effects of policies by race affect health?

1.3 Theoretical Framework

The theory that undergirds this research is grounded in the ecosocial theory. Krieger notes that humans are both biologic and social creatures, and as such, studies examining racial disparities

need to incorporate both aspects of our existence. Race is thought to be an entirely social construct,[9,23] although some scholars believe that race, while mainly social, has some biological elements to it as well.[24] Most racism scholars lambaste researchers who, rather than properly critique their own work in discussion sections, choose to suggest that racial disparities beyond the controls presented in models must exist because of genetic or biologic reasons.[22] However this view of race may stem from scientific racism, when "doctors" made observations about differences between Blacks and Whites that were selectively chosen to promote their own racial bias or the racial bias of the time.[27,103-105] While researchers rarely publish the explicitly racist views of the past, these biases along with a biological definition of race, are taught in medical science courses, which can translate to poor care and thus poor health outcomes.[33,106,107]

The ecosocial theory posits that in the context of structural racism, racialized policies harm Blacks while simultaneously benefiting Whites; "racialized biology" has been used to justify the creation of racial categories; and these create social, occupational, behavioral, and physical environments that can both harm and help one's health.[7-9] As such, racial disparities are formed through "embodiment," or a process by which we absorb the social and physical world around us. Embodiment results in a biological response (e.g., higher blood pressure, shorted telomeres, dysregulated stress response, etc.), which over time results in health outcomes that differ by race.[10,11,47,49,94,108,109] Gravlee (2009) suggests that embodiment mediates the relationships between levels and layers of social structures (e.g., structural racism) and epigenetic changes (e.g., weathering), which phenotype (e.g., race) moderates, resulting in racial disparities in health outcomes over time and possibly across generations.[24] In addition to insults to the body through embodiment, there are also circumstances and environments that can buffer or prevent the health insult, thereby promoting health (e.g., social support, exercise, etc.).[10] A major aspect of the ecosocial model is that it accounts for temporality, place, scale, levels of exposure, historical context, and intergenerational transfer of risks across the lifecourse.[7-9,24,110-112] While each aspect need not be examined in every study, much of social epidemiological research can be grounded in the ecosocial theory of health.

The ecosocial model is also consistent with Link and Phelan's Fundamental Cause theory which suggests that flexible resources (i.e., power, money, prestige, and social connections) provide the bearer with the tools to help avoid risks, while those without are unable to do so.[6] Racism is

considered a fundamental cause because despite improvements in prevention strategies and treatment for many diseases, racial disparities remain favoring Whites.[2-5,113] There are then many pathways in which racism creates health disparities. Once circumstances change, a new pathway can open maintaining poor health. Thus, intervening on any one pathway will likely not affect the fundamental cause. Racism or racist policies that are either explicitly racist or color-blind have been determined to be fundamental causes of disease.[6,92,93]

Finally, Williams and Mohammed suggested a framework for examining racism and health.[10] The framework suggests that there are basic causes, such as biology, society, and racism, all of which interact with each other to stratify people into groups based on socioeconomic status, sex, race, etc. Then there are pathways such as stress or constrained opportunities that produce a biological response which either harms or promotes health.[10] This, too, is consistent with the ecosocial theory and embodiment.

1.4 Policies

Approximately 400 years of American racial policies are nearly impossible to chronicle in their entirety, but it is clear that early American leaders struggled with the morality of slavery.[26] The struggle was clear when, despite its importance to the economics of the early US states, race and slavery were intentionally not directly mentioned in the US Constitution; however, race and slavery are indirectly mentioned in several places leaving the issues of slavery in a "grey area."[26,114] It is argued that because of the intentional grey area surrounding slavery in the Constitution, slavery grew and persisted until the 13th Amendment was passed in 1865.[26] While the American Civil War ended slavery in its original form in the mid-1800s, it did not take long to recreate slavery through other means. In fact, the 13th Amendment includes a statement excluding anyone who has been convicted of a crime. Thus, slavery became an issue of criminal justice through the Black Codes, a series of local vagrancy laws that differentially affected Blacks.[35,115] It was not until 1964 that a sweeping Civil Rights bill was passed, giving racial minorities, among others, the full rights of US Citizens (e.g., voting rights, and anti-discrimination rights in education and employment) that should have been provided at the passage of the 13th through 15th Amendments. The 1964 Civil Rights Act made discrimination illegal, yet many would argue that the Civil Rights Act and the anti-discrimination bills that followed have yet to be fulfilled.[116,117]

The present research focuses on two "lanes" of policies that often cross over in effect: housing and criminal justice. These policy "lanes" were selected because they have contributed to substantial limitations in access to power, resources, and social connections.[26,35,36] While there are likely hundreds of policies, legal rules, and practices that have contributed to structural racism, I have curated several in Table 1.1. These policies were selected because they are considered the most relevant to the present circumstances for Black and White Americans in the areas of housing and criminal justice which have merged into other areas such as education, economic opportunities, and health care access, among others.

1.4.a. Housing and Residential Segregation

"Recorded deed restrictions should strengthen and supplement zoning ordinances and to be effective should include the provisions listed below. The restriction should be recoded with the deed and should run for a period of at least twenty years. Recommended restrictions include the following:

⋮

(g*) prohibition of the occupancy of properties except by the race for which they are intended*."

~Underwriting Manual, Federal Housing Administration, 1936.

Segregation policies were selected because housing and homeownership is the pathway to wealth for most Americans.[36] Historically, there have been federally sanctioned policies that have maintained racial oppression through residential segregation, including redlining, racially restrictive covenants and zoning laws.[36] These policies and practices have been developed by the US Congress, supported by presidents, and often made legal through the Supreme Court.[36] Indeed, residential segregation bleeds into other facets of life, including education, employment and economics, environmental hazards, poor green space, health care access, and crime and victimization.[94] For example, segregated communities result in segregated schools, which results in both fewer opportunities for employment or income as well as lower property values.[36]

Redlining, a mortgage risk mapping system in which nearly all Black communities were outlined in red indicating the highest level of lending risk, is the most cited policy that stimulated the racial divide in housing; however, it started long before that.[36,118-121] In the South, segregation was a de facto custom, although not technically codified through a federal policy.[36] When Blacks began to gain some freedoms, such as equal educational opportunities or voting rights for

Black men, Southern politicians began to become uncomfortable and willing to chip away at these rights through federal policies.[26] While not specifically about housing segregation, in 1896, the US Supreme Court ruled that racial segregation was constitutional.[122] This court ruling precipitated the formation of segregation in nearly every aspect of American life, including housing. This era, which lasted approximately 100 years between Reconstruction and the Civil Rights Movement was named "Jim Crow."[36] Segregation was not restricted to the South; there were versions in the North as well through racially restrictive covenants (i.e., racist language in property deeds determining who can purchase and live in a property), contract lending, local zoning laws, and eventually through redlining.[36,123] This form of northern segregation was so pervasive that the effects remain today in nearly every large northern US city and has been identified as a major driver of health disparities.[94,97,124-128]

1.4.b. Criminal Justice

"Neither slavery nor involuntary servitude, *except as a punishment for crime whereof the party shall have been duly convicted*, shall exist within the United States, or any place subject to their jurisdiction."

~Section 1, 13th Amendment of the US Constitution, 1865

The criminal justice system is grounded in racism but was not "organized" before the Civil War.[35] At that time, the system was extremely local and equivalent to private security.[129] Even in its earliest phases, it was based on slave patrols where citizens would round up runaway slaves and ship them back to their "owners."[130] After the 13th Amendment passed, the police force began to organize and become an institution.[35] This institution used specific language in the 13th Amendment to rebrand slavery through criminal justice. Because of the Civil War, overtly racist ideology became unfashionable, or associated with old Southern thinking.[26] Racism was changing. It was no longer acceptable, or legal, to enslave Black people.[131] However, workers were still needed to harvest agricultural goods and since southern plantation owners relied heavily on slave labor, slavery needed a name change.[26] A series of criminal justice reforms began being developed in the South called the "Black Codes." These codes/laws enabled the incarceration of many newly freed slaves because vagrancy or unemployment became illegal. When unemployed Blacks were arrested, they were sent back to perform slave labor (i.e., convict leasing), often for their previous masters, and were required to pay a nearly unattainable fee

structure to be freed from this false imprisonment.[35,115] Many suggest that this form of slavery was even more brutal, because there was a never-ending supply of prisoners.[35] This is the racist underpinning of our current criminal justice system as is the brutish stereotype of Black men that emerged during slavery and Reconstruction.[38]

In more recent decades, criminal justice policies have led to disproportionate arrests and convictions of Blacks compared to Whites and has done so at every level of the system, thereby creating a concept known as mass incarceration – a result of the "War on Drugs."[35] A series of legislation and court rulings created a criminal justice system that has enabled the police to disproportionately target Blacks, allowed prosecutors to have extreme power to disproportionately charge Blacks with felonies, and a judicial system that rules on disproportionately longer sentences for Blacks, leading to substantially limited access to essential services and resources,[35,132] a limitation that has consequences, including to health[73,133-143] even when experienced vicariously.[75,144] Once a person has served their time in prison, they are released into an often very changed society and have been stripped of nearly all of their rights and benefits, including public aid, housing, voting, employment, and jury service.[35,142] These restrictions severely limit opportunities and ensure that the ex-convict remains in poverty and is unable to fully engage back into society as a free American, thus recreating Jim Crow.[35] Many have likened the recent police-involved killings of innocent Black men and women, as well as death row in prison, to modern-day lynchings.[145,146] Several policies that both created and codified racism in modern policing are listed in Table 1.1.

These policies and their effects are stratified by race. For example, the US has the largest prison population in the world and incarcerates a disproportionately high rate of Blacks compared to Whites.[147,148] Additionally, these disparate outcomes of the criminal justice system are also maintained by stereotypes that stem from US slavery.[38] Both stereotypes and policies result in racialized "events" such as killing of unarmed Black and Brown people during routine traffic stops or while police are questioning someone.[107,149] These heightened and stressful moments result in disproportionate violence by the hands of police by race, which can result in serious physical injury or death.[73,150] Everyday police encounters with citizens are perhaps as or even more harmful. These stops are called Terry Stops and were made legal in the US in the 1970's, allowing police the power to stop and frisk anyone they deem as "suspicious," due to

stereotypes; this meant Black and Brown people.[35] Everyday encounters with the police are associated with a host of health outcomes including reporting higher levels of fair/poor health,[133] higher waist circumference,[134] shorter leukocyte telomere length (a sign of accelerated aging),[135] and stress and anxiety disorders[139,151] While I have illustrated the issue within the policing portion of the criminal justice system, the effect of racial hierarchy and stratification in criminal justice extends beyond incarceration to disparities in parole, disenfranchisement, isolation, and often recidivism, as well as the effect on the non-incarcerated family members.[35,144,152-155]

1.5 Conceptual Framework

Figure 1.1 displays the general conceptual framework for this dissertation. The US starts its history as colonies with England at the helm. In order for the Crown to secure power in the New World, the colonies needed to become financially strong.[26] This required the exploitation of the natural recourses of the new land and many workers.[26] To fulfil the need for workers, colonists imported and owned people to work in the fields as unpaid and exploited workers (i.e., slaves).[26] The colonists also struggled with the immorality of slavery and the viciousness in which the colonists treated the slaves.[26] Thus, scholars at the time began developing racist ideologies to justify the inhumane treatment of slaves.[26] By creating this hierarchy, with slaves, or all People of Color, colonists, scholars and community leaders justified creating racist policies, court rulings *(de jure)*, and practices *(de facto)* to oppress Blacks and any non-White person while simultaneously enabling Whites to have access to resources, power, and connections.[7,26] The consequences of limited access to resources, power and social connections are generally seen through disparate outcomes of policies.[156,157] For example, we can quantify residential segregation or disparities in mortgage lending resulting from the New Deal housing policies in the mid-1930s. We can also quantify the blanket criminal justice policies that have shaped our modern criminal justice system by examining disparities in incarceration, disparities in police involved violence, or police encounters. These measures of structural racism, those that can be linked to a specific policy or a group of explicitly racist or implicitly racist (color-blind) policies, can be examined as an exposure to structural racism. They can also be observed through natural experiments (Chapter 2), through outcomes of racist institutions (Chapter 3), or through area-level indicators resulting from policies (Chapter 4). Racialized events, such as the Flint Water Crisis, and results of structural racism affect those in certain racial groups differently through various mechanisms or pathways that ultimately lead to poor health outcomes. Additionally, the

events or circumstances created through racist policies can impact one's health through direct exposure, such as by having a violent police encounter, or vicariously, by observing those of one's own racial group experience a racialized insult, such as observing Flint residents' experience of lead tainted water.

1.6 Specific Aims

This dissertation will examine the potential health impacts of exposure to structural racism measured three different ways. The three aims are briefly described below:

Aim 1, Chapter 2: Vicarious Structural Racism and Black - White Birth Outcome Disparities in Michigan: The Flint Water Crisis

Flint, a city in Michigan with a long history of racial residential segregation, experienced a substantial economic downturn after a major employer, and much of the White population, left the city limits in the late 1940's through the 1960's.[36,158] Prior to the Fair Housing Act of 1968, Blacks were severely limited in where they could live in US towns and cities, including Flint, resulting in a resource deprived and deteriorating inner city due to federal segregation policies.[36,158] Over time, the state of Michigan took over Flint's finances, and more recently, as a means to save money, the water source was switched from Lake Huron to the highly contaminated Flint River without lead prevention treatment, leading to the Flint Water Crisis (FWC).[158,159] This manmade environmental disaster went virtually unnoticed by the national media for over a year despite substantial complaints from Flint residents, local government, major employers, and researchers.[158] This chapter will examine the health effects of indirect, or vicarious, exposure to the FWC, as defined by the timing of birth in relation to media coverage of the FWC, on Michigan birth outcomes such as birthweight (BW), gestational age (GA), and size-for-gestational age (SzGA) outside of Flint. I hypothesize that exposure to the media attention surrounding the FWC emergency declaration will be associated with lower BW, GA, and SzGA compared to the same time period three years prior for both Black and White women. I also hypothesize that the association will be modified by race where babies born to Black mothers will have a larger decrease in BW, GA, and SzGA, whereas babies born to White mothers will have a smaller decrease in BW, GA, and SzGA during the same time periods.

These hypotheses will be explored using birth records from the Michigan Department of Health and Human Services for the state of Michigan from 2013 through 2016.

Aim 2, Chapter 3: Do Police Encounters Increase the Risk for Cardiovascular Disease? Police Encounters and Framingham 30-Year Cardiovascular Risk Score

Modern policing stems from slavery and slave patrols.[35] It later morphed into a color-blind series of vagrancy laws that disproportionately targeted the newly freed slaves through what has been called the "Black Codes."[35] The criminal justice system then matured into a modern militarized police force that targets Black communities.[35] Additionally, the police force, and individual officers within it, enjoy an environment in which the legal "grey areas" tend to enable discriminatory practices with impunity. This results in a racist institution aimed at maintaining racial hierarchies through incarceration. Additionally, the after effects of incarceration are disenfranchisement and severely limited access to resources due to several *de jure* policies.[35,160] However, incarceration is not the only "product" of the criminal justice system with historic ties to slavery, White supremacy, and lynching, another "product" has been brutal encounters with the police leading to death and injury.[73,149,161] Perhaps the more pervasive issue is the government sanctioned and legal stopping of people at the discretion of the police. This legal pathway, called *Terry Stops* in which police can stop and frisk any person without probable cause for arrest, is far more common than violent encounters and even arrests.[35,162] For example, between 2015 and 2019 there were approximately 65,000 reported *Terry Stops* in New York City alone, mostly involving innocent minorities.[163] During the same time period, there were approximately 5,000 police-involved killings in the entire US, [164] a clear indication that *Terry Stops* are far more common than fatal police encounters.[160,165-169] Thus, minorities are treated differently by the police compared to Whites in the US.[163,170-172]

Violent police encounters experienced in-person and vicariously are associated with both morbidity and mortality.[73,75,134,135,161,169,173] Yet, there is scant evidence demonstrating associations between commonplace and less violent contacts with police and health.[168] The purpose of this chapter is to examine the association between police encounters and the Framingham 30-year cardiovascular (CVD) risk score, through the framework of the differential vulnerability hypotheses, in the National Longitudinal Study of Adolescent to Adult Health (Add

Health) study, a nationally representative sample of young adults. I hypothesize that the association between a high number of reported police encounters and 30-year CVD risk will depend on race, such that the association of exposure to a high number of police encounters and risk for a CVD event occurring in the next 30 years will be higher for Blacks compared to Whites.

Aim 3, Chapter 4: Community-Level Structural Racism and Individual 30-Year Cardiovascular Risk in the United States: Residential and School Racial Segregation

Research has demonstrated strong associations between interpersonal racism and health.[17,18,174] Research strongly supports that poor health effects stem from systemic forms of racism or downstream consequences of racialized policies that form barriers (e.g., access to material and/or psychosocial resources, power, and social connections) to healthy lives.[10-12,15,43] Epidemiological research has begun developing novel and simple metrics of structural racism that capture the effects of several racialized policies such as Jim Crow, redlining, and the War on Drugs.[12,14,77,175,176] Examples of these metrics include area-level measures of residential segregation.[177,178]

The purpose of this chapter is to examine the association between community- and school-level measures of segregation and the individual's Framingham 30-year cardiovascular risk score. I hypothesize that the association between segregation and 30-year cardiovascular risk will differ between Blacks and Whites. I hypothesize that among Blacks, higher community-level structural racism will be associated with a higher risk of having a major cardiovascular event or death in the next 30 years, whereas among Whites, higher community-level structural racism will have no association with the risk of having a cardiovascular event or death in the next 30 years. This chapter will utilize individual data from the Add Health Study and its associated Census Tract level contextual data from Wave I and individual level data for health outcomes measured at Wave IV.

Together these studies will fill gaps in the literature on the association between vicarious structural racism and birth outcomes (Aim 1, Chapter 2), the downstream consequences of individual exposure to tough-on-crime policies on 30-year CVD risk (Aim 2, Chapter 3), and the association of exposure to multiple forms of area-level segregation and the risk of a CVD event

occurring in the next 30 years (Aim 3, Chapter 4). Additionally, this dissertation will be examining three different methods to measure and examine structural racism: a before/after a traumatic event linear regression approach following exposure to a racialized stressor (Aim 1, Chapter 2), direct experiences with the police using linear regression (Aim 2, Chapter 3), and disparities in community-level segregation using a multilevel modeling approach (Aim 3, Chapter 4).

1.7 Tables and Figures

Table 1. 1 United States Policies and Court Rulings in the Domains of Civil Rights, Criminal Justice, Economics and Labor, Education, and Housing Which Have Resulted in Disparate Outcomes for Blacks and Whites.

Year	Policy Name	Government Body	Purpose	Outcome
Civil Rights				
1865	Freedman's Bureau Act	Congress[179,180]	Created a court aimed to hear cases in which employers infringed on the rights of the newly freed slaves. Also provided resources to displaced Southerners after the war, including newly freed slaves, such as medical care, food, clothing, shelter, etc. Revised in 1866 to guarantee a common school for all children.	While this was one of the first social assistance program, Andrew Johnson was vehemently opposed to providing resources to Black citizens. He described this act as a redistribution of White wealth and vetoed it. Congress overrode the veto and later weakened the act through modifications. The Act was very successful in giving newly freed Blacks some support as they navigated freedom, but all was lost when Congress could not maintain its support and it was overturned in 1872.[181,182]
1866	Civil Right Act 1866	Congress[183]	Defined citizenship to all born in the US. Provided a legal pathway for when those acting on behalf of the State to deprive American citizens of their Constitutional rights because of race or alien status. The precursor to the 14th Amendment Equal Protections Clause.[184]	With Congress over-riding President Andrew Johnson's veto gave citizenship to former slaves and those born in the US, guarantee civil rights and equality to all citizens. The act excluded American Indians who were considered "foreigners".[185]
1868	14th Amendment to the US Constitution	Congress[186]	To grant citizenship to anyone born or naturalized in the US, including former slaves, and to guarantee equal protection of the laws to all citizens.	Provided more seats in Congress and thus more power to Southern states by counting former slaves as whole persons in population estimates. Encouraged states to grant the right to vote to Blacks but did not force them to do so.[35]
1870	15th Amendment to the US Constitution	Congress[187]	To prohibit state and federal governments from denying US citizens the right to vote based on race, color, or previous condition of servitude.	Poll taxes, literacy tests, and violent clashes with hate groups such as the Ku Klux Klan continued to prevent Blacks from voting after passage of the 15th Amendment.[35]
1870	Civil Rights Act of 1870	Congress[188]	To enforce the terms of the 15th Amendment.	Provided criminal penalties for the use or threat of violence or other tactics intended to prevent Blacks from voting.

1896 - 1964	Plessey v Ferguson	Supreme Court[122]	Legalized racial segregation in many sectors of life; stemmed from the "Separate Car Act" (1890) in Louisiana (LA) that segregated persons based on race in railway coaches which incurred a fine of $25 or 20 days in jail if violated. Plessy argued that this act violated the 13th and 14th US Constitutional amendments. SCOTUS upheld lower courts decisions that the Separate Car Act was constitutional as long as the separate accommodations were equal to White accommodations, thus legalizing segregation.	Separate but equal doctrine produced differing quality schools, housing, employment, transit, and other areas.[189] Thought to have been repealed by the Brown v Board of Education decision about integrating schools or the 1964 Civil Right Act with prohibited discrimination in employment.
1964	Civil Rights Act	Congress[157]	Prohibits employment discrimination based on race, color, religion, sex, and national origin.	This act banned discrimination AND segregation in employment and public (including publicly funded) spaces. It provided a mechanism for holding accountable those who infringe on one's civil rights. Yet, there remains a considerable wealth and wage gaps between races, ethnicities, and genders.[116] This act was amended in 1991 to include a burden of proof clause for "disparate impact" to be used as evidence in civil rights cases.[190]
1965	Voting Rights Act of 1965	Congress[191]	To outlaw discriminatory practices aimed at suppressing the Black vote and to provide federal oversight of voter registration in areas where less than 50% of the non-white population was not registered to vote.	Increased voter turnout among Blacks and provided the legal mean to challenge voting restrictions.[192]
2013	Shelby County v Holder	US Supreme Court[193]	Ruled that a key provision of the Voting Rights Act of 1965 was unconstitutional.	Resulted in new laws restricting voting, including requiring photo ID, purging of voter rolls, elimination of same day voter registration, closing polling places, and enforcing eligibility restrictions based on address type (e.g., post office box addresses).[192] These laws disproportionately affect minorities.[35,194-196]

Varies	Voting rights for felons	US states	To make convicted felons ineligible to vote.[197]	As a result of mass incarceration, which disproportionately affects Black men, many Americans have lost their constitutional right to vote through felon disenfranchisement.[35,198 152]
Criminal Justice				
1793 & 1850	Fugitive Slave Act	Congress[199,200]	To return slaves who escaped to "free states" back to the plantations in the South and made harboring or helping an escaped slave illegal.[199]	Threatened the safety of all Blacks in the US regardless of their slave/free status. Also provided a mechanism to punish those who harbor escaped slaves. This law began dividing the US in to anti- vs pro-slavery a key issue for the Civil War.[26]
Varies	Vagrancy laws ("The Black Codes")	US states and municipalities	To criminalize unemployment and homelessness.	Though ostensibly colorblind, vagrancy laws disproportionately targeted unemployed former slaves. Once convicted of vagrancy, former slaves were no longer protected under the 13th Amendment and could be forced to work without pay.[35,115,131] Black convicts were often "leased" out to their former owners, worked under extremely violent and substandard working conditions where many died or were significantly disabled as a result, all while the State and plantation owners profited.[35,145]
1866	Civil Rights Act	Congress[183]	Defined citizens to all born in the US, made it illegal for anyone acting on the governments behalf to violate one's Constitutional rights.	Did not ensure voting for Blacks (or women), but did create a legal pathway for violations. Since no resources were set aside for legal challenges, few could afford to initiate a legal case.[184,185] Also, the federal penalties for this law did not occur until the 1960's Civil Rights Acts were written.
1871, 1971, 1982	Qualified Immunity; Civil Rights Act (Section 1983); Bivens v Six Unknown Named Agents of the Federal Bureau of Narcotics (1971); Harlow v Fitzgerald (1982)	Congress[201] and Supreme Court[202,203]	The 1871 Civil Rights Act provides a mechanism for enforcing the 13th – 15th Constitutional Amendments by authorizing individual citizens to bring civil law suits for violations made by police or others in local government. The Bivens case formed the legal doctrine that shields state actors (including police) from being held	Section 1983 cases are still heard today, but the later court cases have made winning a civil rights law suit against the police (or other state actors) extremely difficult by requiring proof of a violation of a Constitutional right AND a legal precedent of a ruling in a case with near identical circumstances.[35,204]

			personally liable in the event that they, in the course of their regular job, violate a citizen's Constitutional rights.	
1876	United States v Cruikshank	Supreme Court[205]	A challenge to the 1870 Enforcement Act after the Colfax Massacre (after an all Black militia seized control of a governmental building out of fear that the Democrats would win the governor seat in a hotly contested election, a large White mob killed nearly all of the Black militia). The court ruled that the 14th Amendment applies to only to situations where the State denies any person life, liberty, or property without due process of the law. It made no clarification on individuals denying these same rights without due process.	This ruling made it extremely difficult for Blacks to receive justice for mob lynchings where a group of White individuals take the lives of someone else. After this ruling, all legal cases against those participating in lynchings were dropped and rarely were individuals held accountable for lynchings.[145]
1877 - 2005	Failure of the US Congress to pass ~ 200 attempts for Anti-Lynching laws	Congress	To create a pathway for the surviving family of victims of lynching to receive justice for the murders of those lynched without any form of due process of the courts.	Had anti-lynching laws been passed there would have been a stark reduction in lynchings after holding accountable those responsible for carrying out, witnessing, aiding, abetting, or inaction by law enforcement or government. There was an estimated 4,400 documented lynchings that occurred in 20 US in both the South and North between about 1877 and 1950. [145] In 2005 the US Congress formally apologized to Black Americans for their inaction to protect their ancestors.[145]
1950	Boggs Act	Congress[206]	Mandatory minimum sentencing and fines for marijuana possession.	This was the first law enacted in which mandatory sentencing was established for an illicit drug. Notably, in the 1950's marijuana use was high in the jazz culture among Black "hepsters," among Mexican immigrants and White beatniks.[a]
1953 – 1973	Operation CHAOS	Central Intelligence Agency; Federal	To weed out foreign communist influences.	This program morphed into a War on Drugs program in which the CIA/FBI spied on citizens

[a] https://www.pbs.org/wgbh/pages/frontline/shows/drugs/buyers/socialhistory.html

		Bureau of Investigation		and civil rights protesters of the 1960s. Collected the names of over 300,000 people and groups and made no connections among them to foreign communist influencers.[207] Martin Luther King, Jr. was in the database.
1968	Terry v Ohio	Supreme Court[162]	Stop and frisk by the police is Constitutional.	High levels of police harassment in order to control, surveil, and arrest "suspicious persons". The ruling gave police discretion on whom to fight the drug war.[35]
Beginning in 1971	War on Drugs	Office of the President[208-210]; Congress[211,212]	To increase the size and presence of federal drug control agencies and to criminalize the possession, distribution, and use of drugs.[35,162,213-221]	The War on Drugs resulted in mass incarceration, with disproportionate effects on people of color, particularly Black men.[35,218,222,223]
1978	Monell Rule (Monell v Department of Social Services of New York; Monroe v Pape)	Supreme Court[224,225]	In Monroe v Pape (1961) the court ruled that a city could not be held liable for a civil right infraction of its employees, but reverse course a bit in the Monell case where the city can be held liable in civil rights trials, if an "official policy" caused the infraction.	The Monell rule has created a major barrier to police accountability when an officer in its employ commits misconduct at the level of a civil rights violation. Qualified immunity protects the individual officer and the Monell rule protects the police department and the city that overlooks the patterns of misconduct department wide enabling police misconduct with impunity.[226]
1981	Posse Comitatus Act	Congress[215,216]	A revision of the 1878 act of the same name. Originally was enacted to ensure that the military could not be used for domestic law enforcement, unless the local police were unable or unwilling to protect citizens. The later amendment allowed the Department of Defense to assist local law enforcement in any way possible when called as well as provides funding when using the Act.	Initially it was used to secure a Yea vote for the presidency of Rutherford B. Hayes from Southern Democrats during reconstruction (1878). Later it was used to protect kids going to integrated schools in the 1950's. It has also led the militarizing of police.[227,228]
1986	Anti-Drug Abuse Act	Congress[219]	Mandatory minimum sentencing for possession and trafficking of illicit drugs.	Created a 100 to 1 disparity in sentencing for crack cocaine, despite its chemical make-up is nearly identical to powder cocaine, the only difference is who used it.[35]

1989	Graham v Connor	Supreme Court[229]	Prior to this case excessive use of force by the police was determined acceptable if a reasonable person in the general public would view the force as excessive in a similar situation. This case redefined what was considered "reasonable force" as what amount of force a police officer would use in a similar encounter but rather than an ordinary citizen.	This definition provided a mechanism for police officers to protect others by stating they would have done the same thing, thereby reducing the likelihood of being held accountable as long as police stick together about the use of force.[230]
1994	Violent Crime Control and Law Enforcement Act	Congress[231]	Largest crime bill in US history. Provided new statutes with penalties, increased police forces, prison funding, crime prevention, federal assault weapons ban, expansion of the death penalty, and required the Attorney General to create a Use of Force Registry	Many argue that this bill is responsible for mass incarceration.[35]
1996	Wren et. al. v US	Supreme Court[213]	Using race as a determinant of criminal suspicion was deemed constitutional.	Provided more legal support to police on whom to fight the drug war.[35]
1997	National Defense Authorization Act (Federal 1033 Program)	Congress[232]	Facilitate the transfer of surplus military equipment to local police agencies.	Provided substantial weaponry and militarized the local police departments.[35]
2000	Illinois v Wardlow	Supreme Court[214]	Using neighborhood context as a method of confirm police suspicion of a crime.	Allowed police to nearly constantly surveil minority neighborhoods.[35]
2010	Fair Sentencing Act	Congress[233]	To reduce the sentencing disparity for various types of cocaine.	Reduced from 100:1 to 18:1, increased the amount in possession of crack cocaine to 1 ounce.
Economics and Labor				
1865	13th Amendment to the US Constitution	Congress[131]	To abolish slavery and involuntary servitude, except as punishment for a crime.	No laws were ever passed to compensate former slaves for their enslavement, so most Southern blacks had no choice but to work as laborers on the farms and plantations of white landowners.[26]
Beginning in 1865	The Black Codes	Former Confederate state legislatures[35,115,234]	To restrict the labor activities of former slaves	Former slaves were forced to sign annual labor contracts with white landowners or risk being arrested and jailed for vagrancy.[26,115]
1935	The Social Security Act	Congress[235,236]	To establish a system of old-age benefits for workers, unemployment insurance, and	Agricultural and domestic service workers were excluded from receiving Social Security

			aid for dependent mothers and children, the blind, and the physically disabled.	benefits and unemployment protections. In 1935, 65% of blacks worked in one of these professions compared to 27% of whites.[235]
1935	National Labor Relations Act	Congress[235,237]	To protect the rights of employees and employers, to encourage collective bargaining, and to limit private sector labor and management practices that harm workers, businesses, and the economy.	Agricultural and domestic service workers were excluded from protections.[235]
1938	Fair Labor Standards Act	Congress[235,238]	To establish minimum wage, overtime pay, recordkeeping, and child labor standards for private sector and government workers.	Agricultural and domestic service workers were excluded from protections.[235]
Education				
1954	Brown v Board of Education of Topeka	US Supreme Court[239,240]	Ruled that separate educational facilities for black students were unconstitutional.	One year later, the court issued a second ruling ordering schools to integrate "with all deliberate speed." The court set out rules for desegregation and explained how the government would monitor progress on desegregation.[239]
1964	Civil Rights Act, Title IV, Sections 407 & 401b	Congress[157]	Defines desegregation as assignment of schools without regard to race, color, religion, or national origin and not as a means to overcome racial imbalance. Also limited federal oversight in areas with *de facto* segregation.	These 2 sections made bussing and desegregation orders relevant only to former Jim Crow states, enabling northern areas to remain segregated.[241]
1971	Swann v Charlotte-Mecklenburg Board of Education	US Supreme Court[242]	Upheld busing programs designed to racially integrate schools.	Busing continued in most major cities until the late 1990s.
1974	Milliken v Bradley	US Supreme Court[243-245]	Struck down a multi-district plan to join the Detroit school system with 53 outlying suburban districts.	"White flight" from cities to suburbs made it increasingly difficult to desegregate schools within districts.[36]
1974	General Education Provisions Act	Congress[246-248]	Banned the use of federal funds to provide transportation for the purpose of overcoming racial imbalance.	Barriers to bussing in schools with limited resources (still in effect today).
Beginning in 1990s	Zero tolerance policies	States and school districts[249,250]	To impose specific punishments, such as suspension or expulsion, when certain	Played a role in the school to prison pipeline and disproportionately affected minority students.[249]

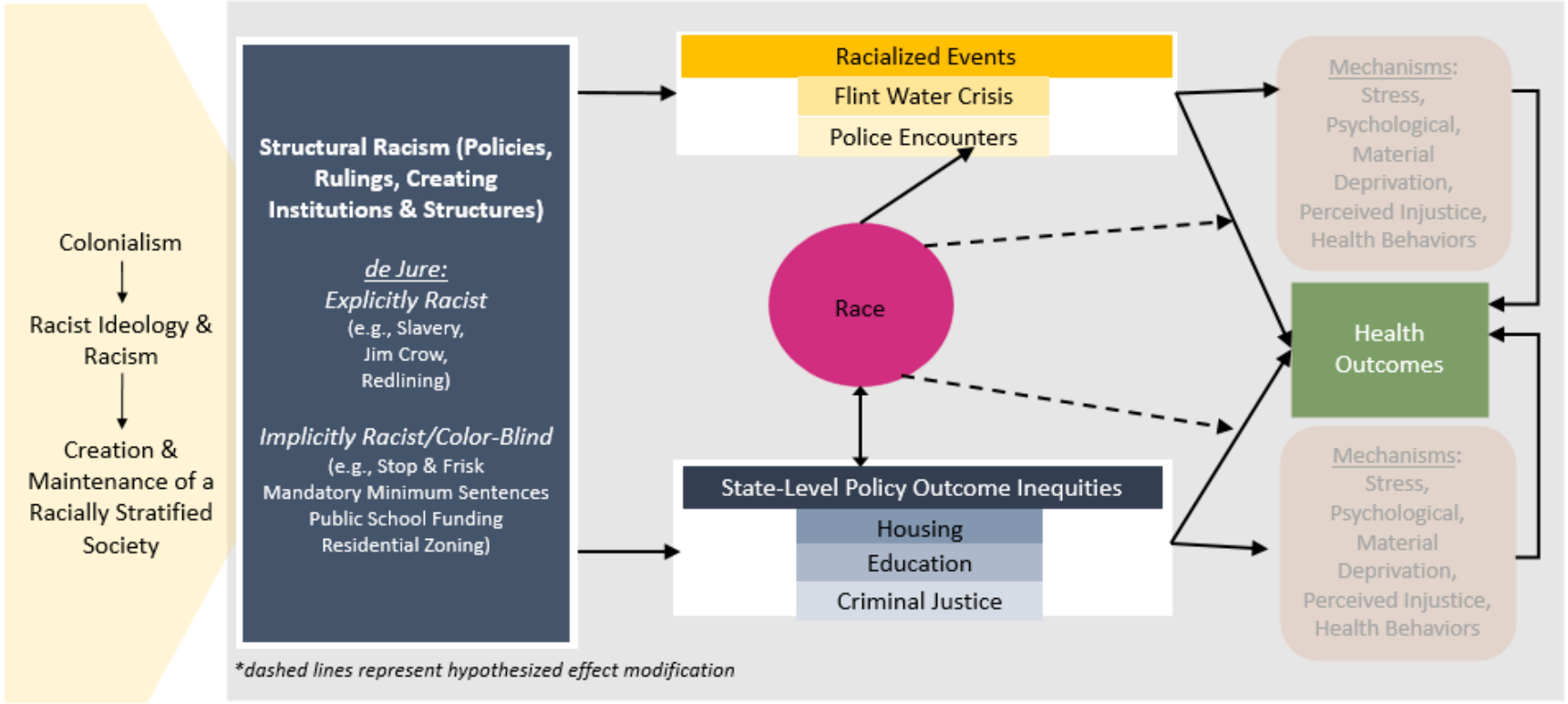
			school rules are broken, regardless of circumstances.[251]	
1991	Oklahoma City Board of Education v Dowell	US Supreme Court[252,253]	Ruled that district courts could remove desegregation decrees once school districts had complied with the order for a reasonable period of time.[252]	Contributed to the re-segregation of schools by providing a mechanism for the end of federal court desegregation orders.[239]
2007	Parents Involved in Community Schools v Seattle School District	US Supreme Court[254,255]	Struck down plans to use race as a factor in student assignment in two school systems that were not under court supervision for desegregation.	Limited the options available to schools to voluntarily desegregate. In response, the US Department of Education Office for Civil Rights published "Guidance on the Voluntary Use of Race to Achieve Diversity and Avoid Racial Isolation in Elementary and Secondary Schools" in 2011.[256]
Housing				
1916	Buchanan v Warley	Supreme Court[36,257]	Ruled that restricted covenants are unconstitutional because they infringe on the rights of a homeowner to sell their home to whomever they please.	Local areas began adding by-laws to home sellers' contracts to reinforce racial segregation through private homeowner sales contracts restricting the sale of homes to Whites only.[36] Communities skirted the 1916 ruling by maintaining that these by-laws in sale contracts were based on private agreements and not policy.[36] Additionally, communities began creating zoning laws that were colorblind, but intended to keep the community segregated.[36]
1926	Corrigan v Buckley	Supreme Court[258]	Ruled that restrictive covenants were binding and made selling a property to a Black family a void in contract.	Made racially restrictive covenants, where deeds limited who could buy homes in certain neighborhoods, common practice.[36]
1934	National Housing Act	Congress[121,259,260]	To establish the Federal Housing Administration (FHA) to set standards for construction and underwriting and to insure mortgage loans made by banks.	The underwriting rules deemed properties in predominantly Black neighborhoods high risk for mortgage loans and prohibited blacks from purchasing homes in predominantly White areas, resulting in (continued) racial residential segregation and low rates of home ownership among blacks. The policy significantly increased home ownership among Whites by providing long-term mortgages with low

				interest rates. Disparities in home ownership have contributed to the substantial gap in wealth between Whites and Blacks in the US.[121,259]
1944	Federal-Aid Highway Act	Congress[261]	To designated a national system of interstate highways (although funding for the highways was not provided until the 1950s).	Facilitated "white flight" to the racially segregated suburbs. As a means of "slum clearance" or "urban renewal", typically constructed new highways through over-crowded segregated Black neighborhoods maintain racial segregation.[36,262,263]
1948	Shelley v Kraemer	Supreme Court[264]	Ruled that enforcement of a restrictive covenant between individuals is voluntary, unless it is an agreement made by the state then it would be a violation of the 14th Amendment.	Since the court defined restrictive covenants illegal only if it is written by the government, the language in deeds remained for decades after, further maintaining segregation.[36]
1968	Civil Rights Act (1968), Title VIII – IX (Fair Housing Act)	Congress[265]	To ban discrimination in housing sales or rentals based on race, religion or national origin and to revise the 1964 Civil Rights Act by including provisions for enforcement.	Only affected new purchases or rentals, did not correct or dismantle past discrimination. While banning housing discrimination, the portions devoted to enforcement were weakened in order for it to pass through Congress. Additionally, victims of housing discrimination were allowed to file a civil suit, they were only allowed to retrieve actual damages and investigations had to occur within 4 months of the alleged incident.[266]
1968	Jones v Alfred H. Mayer Co.	Supreme Court[267]	Citing the 1866 Civil Right Act, the court ruled that Congress can regulate the sale of private property to prevent racial discrimination.	This case reversed many of the rulings and policies that created and maintained racial segregation.[268]
1975	Home Mortgage Disclosure Act	Congress[269]	Required financial institutions to maintain, report and publically disclose information about mortgages to decrease discriminatory mortgage lending.	Provided a way to evaluate whether institutions are discriminating against minorities. This was amended with the Frank-Dodd Wall Street Reform and Consumer Protection Act (2010) by adding indicators such as credit scores which may explain why loans are denied.[270]
1977	Community Reinvestment Act	Congress[271]	Ended racially discriminatory mortgage lending practices.	While the act increased homeownership among low to moderate income families, including

				many Black families, this effect was short-lived and there remains a substantial homeownership gap between Blacks and White sin the US.[272,b]
2015	Affirmatively Furthering Fair Housing	Housing and Urban Development[273]	This rule provided a mechanism to evaluate and ultimately for reversing racial segregation in housing among all HUD properties.	As a political move, the Trump Administration rescinded this rule in 2020[274] with hope to attract White suburban voters who have benefited from racial segregation. It is unclear what effect this will have on changing residential segregation. The Biden Administration reinstated it in 2021.[275]

[b] https://www.urban.org/urban-wire/are-gains-black-homeownership-history

Figure 1. 1 Conceptual Model



Chapter 2 Vicarious Structural Racism and Black - White Birth Outcome Disparities in Michigan: The Flint Water Crisis

"Recorded deed restrictions should strengthen and supplement zoning ordinances and to be effective should include the provisions listed below. The restriction should be recorded with the deed and should run for a period of at least twenty years. Recommended restrictions include the following:

⋮

(g) prohibition of the occupancy of properties except by the race for which they are intended."

~Underwriting Manual, Federal Housing Administration, 1936.

2.1 Background

Babies born to Black mothers have at least a 2-times higher infant mortality rate than babies born to White mothers.[1,276-278] Additionally, there are persistent racial disparities in birthweight (BW), gestational age (GA), and size-for-gestational-age (SzGA), which are causally associated with infant mortality (Figure 2.1).[279,280] Despite these long-standing disparities, differences in birth outcomes between Blacks and Whites are not fully explained by standard risk factors such as prior preterm births (PTB), clinical factors (e.g., short cervical length, prior cervical surgery, infections), lack of prenatal care, smoking, low pre-pregnancy body mass index (BMI), substance use, short inter-pregnancy interval, or age.[108,281] Poor birth outcomes are not only a major risk factor for infant mortality, but affect long-term health as well. For example, low BW and prematurity are associated with learning difficulties and behavioral problems during childhood,[279,282] as well as the development of chronic conditions in adulthood such as hypertension, coronary heart diseases, and diabetes.[282-285]

Many researchers have posited that racism or discrimination is a major mechanism by which poor birth outcomes develop.[6,13,74,85,87,286-292] Most research has focused on interpersonal discrimination, or individual actions in which another individual or individuals are treated unfairly because of their race.[7,10] This is problematic because it ignores the pervasive but less visible exposure to structural racism, a more common form of racism that no longer targets an

individual but rather reflects policies that have differentially affected one group over another and may be more detrimental to one's health than interpersonal discrimination.[10-13,15,16,45,51,56,293,294]

Because structural racism is intertwined into the social order of American life, it is a ubiquitous and an often ignored exposure, making it very challenging to study.[16,45] One way to examine larger macro discrimination-related stressors is to compare health outcomes before and after major racialized events—a situation in which marginalized groups are more affected. Historically, events that are experienced acutely or chronically and become racialized either through intent, such as failing to safely change the water sources in Flint, Michigan, or by society's response to an event (e.g., 9/11 attacks or Hurricane Katrina), are prime for such study. These major events that often occur without warning may produce an immediate threat to one's well-being.[78,295,296] Additionally, the societal response, or lack of a response, may affect longer-term health due to fear, worry, or a reminder of one's place among the social rankings.[10,49,60,297-299] Several studies that incorporate a quasi-experimental design have demonstrated that after major stressful and often racialized events, birth outcomes are substantially worse after the event regardless of whether the event was experienced directly[300-314] or indirectly (vicariously).[78,79,315-322] However, some studies have shown mixed[301,305,308,314] or no association between stressful events and birth outcomes.[323-327] In general, this body of literature suggests that a vicarious exposure to a racialized disaster has an effect on birth outcomes and that the effect differs by race/ethnicity.[78,79,316] For example, Novak et. al. (2017) demonstrated an increased risk of low birth weight (LBW) among Latinas compared to White women in Iowa after a vicarious exposure to an immigration raid in Postville, IA.[78] Additionally, Lauderdale et. al. (2006) reported that Arabic-named women in California had a higher risk of LBW and PTB after a vicarious exposure to the 9/11 attacks in 2001.[79] Finally, two international studies that examined vicarious exposures to two different terrorist attacks (London on 7/7/2005 and the 9/11 attacks in the US) reported decreased birthweights and increased risks of small-for-gestational-age (SmGA).[319,320] Although not specifically studying race, these studies provide evidence that vicarious exposure to highly stressful events through some form of media can affect birth outcomes.[319,320]

A recent example of a racialized event is the Flint Water Crisis (FWC) in Michigan, which began on April 25, 2014, after the state-appointed city manager changed the water source from Lake

Huron to the Flint River (Figure 2.2).[158,328-330] As a cost cutting decision, the water was not treated properly, resulting in substantial lead contamination.[158,159,328,331-333] Despite emphatic public complaints that were largely ignored, the residents of Flint endured lead exposure through the improperly treated water for 1.5 years, as well as Legionnaire's disease outbreaks resulting in at least 12 deaths, documented elevated blood lead levels in local children, a major financial burden, and an immeasurable loss of trust.[158,329,331,332,334-341] Although the water source was changed back to Lake Huron and the lead contamination declined, the corroded pipes remained, placing Flint residents at continued risk for exposure to lead and other contaminants, particularly Legionnaire's disease, until at least September 2017.[342] Despite the magnitude of the FWC, only the local media covered the story initially, which was largely ignored by national media.[343,344] Once the national spotlight was shined on Flint after the declaration of a state of emergency in Flint, herein referred to as the FWC declaration, on January 5, 2016, the images seen nationally were of poor Black residents (Figure 2.3).[158,343]

The FWC was the result of longstanding and historic legally sanctioned structural racism that includes "northern style" segregation (i.e., segregation occurring in non-Jim Crow states that relied upon local ordinances, *de facto* property contracts, and federal laws) that was pervasive in nearly every facet of life in Flint, including housing, employment opportunities, education, and other areas since Flint became a city.[158,262] The consequences of federal policies that enabled redlining and "white-flight" are at the very core of the demographic and economic make-up of modern day Flint. In its early days, the city of Flint flourished with a strong tax base of mostly White residents and substantial employment opportunities.[158,262] Flint was also highly segregated, initially permitting the Black residents to reside in only one corner of the city.[158,262] As jobs became available for all skill-levels, the Black community quickly became overcrowded.[158,262] With a population of about 100,000, after "white flight" (White residents leave when non-Whites move into a predominately White neighborhood)[345] the demographics shifted to a slightly majority Black and disadvantaged community. This shift in finances and a strengthening of the Michigan emergency management laws in 2011 enabled the Michigan governor to appoint a city manager with accountability only to the governor.[158,262,333,346]

The Michigan Civil Rights Commission, an official state agency that investigates complaints and enforces civil rights and fair housing laws in the State of Michigan, firmly and publicly

recognized the FWC as a form of structural racism.[158] The failure of the media to illustrate examples of racism and the government's failure to intervene on racism are stressful and may lead to severed trust of these institutions.[347] The FWC was undoubtedly stressful, especially for residents directly impacted by significant lead contamination in their drinking water.[18,348] However, the stress likely did not stop at the Flint city limits. It is quite possible that non-Flint residents who were indirectly exposed to the FWC experienced this crisis vicariously when the media began to reporting on the FWC in January 2016.[13,349] The FWC, along with everyday stressors, may have contributed to poorer health, especially among those most vulnerable in our population--newborns.[350-359] Vicarious exposure to the FWC includes mothers who were likely exposed to the FWC through the substantial increase in social and traditional media coverage when President Obama (US), Governor Snyder (Michigan), and Mayor Karen Weaver (Flint) declared a state of emergency in Flint in early January 2016.[344] While the Michigan Civil Rights Commission defined the FWC as the result of structural racism, and despite national attention raised by the #BlackLivesMatter movement, much of the media neglected to tell the story about the FWC.[343] In fact, the increased media attention occurred about three months ***after*** the contaminated water was switched back to the treated water via Detroit/Lake Huron (Figures 2.2 and 2.3).

The vicarious exposure to the FWC is the crux of this study. Thus, the purpose of this study is to examine the association between birth timing surrounding the increased media coverage after the FWC declaration was made in Flint and the birth outcomes of babies born to Black and White women in Michigan, but outside of Flint. I hypothesize that there will be a decrease in BW, GA, and SzGA for babies born to Black and White mothers in Michigan (but outside of Flint) in the 37 weeks following the FWC declaration compared to the same 37 weeks in the prior three years. I further hypothesize that the association between the FWC declaration and birth outcomes will be modified by race such that babies born to Black mothers will have a larger decrease in BW, GA, and SzGA compared to babies born to White mothers whereas babies born to White mothers will have a smaller decrease in BW, GA, and SzGA compared to babies born to Black mothers during the same time periods.

2.2 Methods

2.2.a. Data Source

This study utilizes a pre-post quasi-experimental design to investigate the effects of a vicarious exposure to the FWC declaration on BW, GA, and SzGA in Michigan. I obtained data from the Michigan Department of Health and Human Services (MDHHS) - Division of Vital Records and Health Statistics including all singleton live births in Michigan that occurred in calendar years 2013-2016 (n=437,713). MDHHS provided our team de-identified files after Institutional Review Boards for both the University of Michigan and the State of Michigan approved the use of this data.

As any change in BW, GA, and SzGA in Flint may be directly caused by the consumption of lead contaminated water among pregnant women, the study population for this aim was restricted to babies born to Black and White mothers in Michigan but outside of Flint to exclude babies most likely directly exposed to Flint's contaminated water.[78,360] The analytic sample includes babies born to Non-Hispanic Black (Black) and Non-Hispanic White (White) mothers residing outside of the city of Flint, Michigan (n=373,434, See Figure 2.4). In the event that a mother had more than one baby (n=59,806) during the study period, one birth was randomly selected using simple random sampling methodology resulting in 315,686 births. After excluding births with implausible or missing GA and/or BW (n=519), as well as births not occurring between January and September in any year (n=88,212) and those missing residential information (n=15) the analytic sample size was 226,672 births.

2.2.b. Exposure

Babies born to Black or White mothers up to 37 weeks after the declaration of a state of emergency in Flint (FWC declaration), or January 5, 2016, through September 20, 2016, were classified as exposed. Thirty-seven weeks is the earliest number of weeks in which a baby is considered full term.[361] There was a three year unexposed period, thus, babies born between January 5 through September 20 in each of the years between 2013 and 2015 were classified as unexposed. The FWC was not a discrete event that happened and then ended. In fact, the water contamination occurred over 18 months, but there was a low level of media coverage until the FWC declaration, which began in January 2016. Because the FWC occurred over such a long period, there may have been people who were aware of the FWC while it was happening

between April 25, 2014, through October 16, 2015. Given this timing overlap, I included births from 2013, which completely predates the FWC. Since many things could have happened between 2013 and 2016, I did not want to compare these two years alone, thus I included the data for births occurring in 2013-2015 prior to the FWC declaration. However, because of this, there is a potential for misclassification of exposure where some women in Michigan may have been exposed to the news about Flint prior to the declaration of the state of emergency or may have known someone who lived in Flint during the Flint Water Crisis.

2.2.c. Outcomes

The primary outcomes for this aim are BW, GA, and SzGA. BW was measured in grams at the time of birth by the birthing team and is recorded on the standard birth certificate. Missing BWs or those recorded as <375 grams were excluded (n=357, see Figure 2.4).

GA is provided on the standard birth certificate and is an obstetric estimate of weeks gestation (estimated), but may also be based on the mother's last menstrual period (LMP) in which GA was verified by calculating the number of days between the mother's self-reported date of LMP and the date of birth of the baby.[362,363] There were 96 births in which both GA measures were missing, 61 were excluded because GA was <22 weeks (non-viable births), and 5 births where the GA is >44 weeks, leaving 315,167 births.[364] When there were two GAs listed on the birth certificate, I used a data-cleaning algorithm developed by Basso et. al. to ensure the accuracy of the selected GA.[363] In short, GA was converted to a z-score based on the Basso et. al. paper using the 2010 sex-specific standard newborn population and the difference between the two provided GA measures (estimated and LMP) was calculated. If the difference was less than 2 weeks, and the z score was between -/+5 for term babies and -4/+3 for preterm babies the physician estimated GA was selected (n=231 excluded because of an out of range z-score). If the difference in GAs was greater than 2 weeks and the baby was term with a z score between -/+5 then the physician estimate of GA was selected. If the baby was preterm and the z score was between -3/+2, then the GA ages based on the LMP was selected (n=25 excluded). In the event that the GAs did not fit either of these criteria, the births were excluded (n=12 excluded).[364] There were 226,672 births within the acceptable ranges (Figure 2.4). Once a final GA was decided using the cleaning algorithm, the z-scores were recalculated with the final GA. Using the 2010 sex-specific BW distribution, standardized z-scores were calculated as the SzGA.[364]

2.2.d. Covariates

I hypothesized *a priori* that baby's sex, maternal age, maternal education, marital status, pre-pregnancy body mass index, parity, insurance status, and WIC status are important predictors of the outcomes (Figure 2.5 - DAG).[281,365-368] Research suggests that controlling for these will improve precision of estiamtes.[369] Additionally, there are temporal trends in some variables that may be predictors of the outcomes, thus they are included as control variables to help ensure that the only difference between groups is the exposure status. Baby's sex was recorded on the birth certificate.[370] Maternal age was provided on the standard birth certificate. Maternal education was a 5-level categorical variable (less than high school, high school graduate or GED, some college, college graduate or more, and unknown) derived from education recorded on the birth certificate. Marital status, a self-reported 4-level variable, was provided on the birth certificate. The categories are: not married, married, divorced or widowed, and unknown. The continuous pre-pregnancy body mass index (BMI) was calculated from pre-pregnancy weight in pounds and height in inches with the following formula: $(\text{weight}/\text{height}^2)*702$. Parity was calculated as the sum of three variables recorded in the birth certificate, which provides a count of prior births both living and dead. Insurance status was provided on the birth certificate in the following categories: private insurance, Medicaid, self-pay, other, and unknown. Women, Infant, and Children (WIC) public assistance nutrition program status was provided on the birth certificate in the following categories: received, not received, or unknown. Some of these hypothesized outcome predictors may also operate through other variables such as the use of public assistance (measured) or some unmeasured covariate such as area level macro-economic conditions (unmeasured) which may increase stress and likely contributes to poor birth outcomes.[354,371-373] While macro-economic conditions are unmeasured, whether or not the mother relied on WIC, a binary variable, was used as a proxy for income which is associated with poor birth outcomes.[281,354,366,367]

Race was hypothesized as an effect modifier because I hypothesize that Black and White women experienced the FWC declaration differently.[374,375] Additionally, residential geographic region may be a risk factor for the outcome because while it is assumed that the all women had heard of the FWC at the time of the FWC declaration, those living closer to Flint may have become aware of the FWC during the water change.[79,301,316,323] County was the primary indicator for distance

from Flint. Geographic region was derived from county and city of residence of the mother at the time of birth. When county was not available, the city of residence was used to identify the county in which the city is located. I categorized each county within one of the 10 State of Michigan Prosperity Regions (www.michigan.gov) as a proxy for the distance from Flint. Although mothers from the city of Flint were excluded, mothers from the rest of Genesee County, the county in which Flint is located, were included.

Seasonality of birth is associated with birthweight where lower temperatures are associated with lower birthweights.[376] While climate or weather is not a variable we include in the models it is controlled through restriction of births between January and September in all years.

I determined--*a priori*—that maternal smoking and alcohol use, gestational diabetes, adequacy of prenatal care, other medical conditions (i.e., pre-pregnancy diabetes and hypertension, preeclampsia or intrauterine growth restriction, and sexually transmitted infections during pregnancy), and prior pre-term births were mediating variables because they are likely in the causal pathway between exposure and outcomes. These variables will be included as covariates in sensitivity analyses.[281,377-397]

2.2.e. Statistical Analysis

I began by examining the structure of the data to assess outliers, implausible or missing data, and distributions of all variables by conducting a univariate analysis. Bivariate associations were explored, first, by examining the relationships between the outcome and the covariates and by examining the associations between the exposure and covariates (Table 2.1). In the event that an important covariate based on prior studies is not statistically significant via bivariate analysis, these variables remained in the model (see Figure 2.5). Statistically significant differences were assessed using t-tests for continuous variables, Pearson chi-square tests for binary variables, and ANOVA for categorical variables. Two-sided statistical significance was established at the 0.05 level.

For this quasi-experimental study a before/after linear regression analytic approach was used. I ran three regression models. In the first, I examined a model including only the FWC declaration exposure and race, unadjusted for covariates. In the second model I adjusted for all *a priori*

covariates, to test the first research question to see if there was a difference in outcomes before and after the FWC declaration. In the third model I tested for an interaction between the FWC declaration and race, to see if the impact of the declaration on birth outcomes differed by race. The following models were examined for all outcomes (1-3):

(1) $Y_{Outcome} = \beta_0 + \beta_1 race + \beta_2 eFWC\ declaration + \varepsilon$

(2) $Y_{Outcome} = \beta_0 + \beta_1 race + \beta_2 FWC\ declaration + \beta_4 mom's\ age + \beta_5 pre-preg.BMI + \beta_6 parity + \beta_7 baby's\ sex + \beta_8 mom's\ education + \beta_9 marital\ status + \beta_{10} insurance + \beta_{11} WIC + \beta_{12} geog.region + \varepsilon$

(3) $Y_{Outcome} = \beta_0 + \beta_1 race + \beta_2 FWC\ declaration + \beta_3 race * FWC\ declaration + \beta_4 mom's\ age + \beta_5 pre-preg.BMI + \beta_6 parity + \beta_7 baby's\ sex + \beta_8 mom's\ education + \beta_9 marital\ status + \beta_{10} insurance + \beta_{11} WIC + \beta_{12} geog.region + \varepsilon$

I report the least-squared means with 95% confidence intervals (Wald-type) and p-values assessed at the $p<0.05$ level. The analysis was conducted in SAS 9.4 (SAS Institute Inc., Carey, NC).

2.2.f. Model Assumptions

There are a few standard model assumptions that are examined after the final model is fit. It is assumed that the errors in the fitted model are independent and follow a normal distribution. Additionally homoscedasticity or constant variance is another assumption of linear models.

2.2.g. Sensitivity Analyses

To examine the robustness of the main findings several sensitivity analyses were performed. I re-analyzed the main models by including hypothesized mediators. Additionally, the main analysis was performed again by comparing the exposed group to those unexposed in each year between 2013 and 2015 (three separate analyses). In another analysis, the 3-year exposure period was included as a 3-level categorical variable and compared to the 2016 exposed period. I also examined the binary outcomes of LBW, PTB, and SmGA. These analyses were performed using the same covariates as the primary analysis using a logistic regression analysis. A birth was defined as LBW when the birthweight is <2500g.[278] PTB was defined as a birth in which the baby's GA is <37 weeks at birth.[363,398-400] SmGA was provided by MDHHS and is defined as babies born in the <=10th percentile for GA.[364]

Finally, to understand which trimester was the highest risk for exposure I restricted the primary analysis to those who were exposed during the first, second, and third trimester separately and compared them to those in the same trimester in the unexposed years. Trimester of exposure was calculated by multiplying gestational week (provided on the birth certificate) by seven to get days gestation. Then I estimated a conception date by subtracting days gestation at birth less 14 days (LMP typically occurs 14 days before ovulation) from the baby's date of birth. I then categorized the trimesters as follows: 1st trimester (0 to 13.9 weeks), 2nd trimester (14 to 27.9 weeks), and 3rd trimester (28 to 40.9 weeks).[401]

2.3 Results

Table 2.1 displays the descriptive statistics for all study variables by race and exposure status. About 20% of all births were to Black mothers both before and after the FWC declaration. There are statistically significant racial difference in maternal age, education, marital status, payment source, WIC usage, pre-pregnancy BMI, parity, the Kessner index/prenatal care, pregnancy risk factors, at least 1 STI during pregnancy and residential geography. A higher proportion of male babies are born to White mothers compared to Black mothers who have a higher percent of female babies. While there are many racial differences, there are fewer differences by exposure status. Namely, unexposed mothers reported lower education levels, higher WIC usage, higher parity and more tobacco usage, whereas those exposed reported a higher pre-pregnancy BMI, higher level of adequate prenatal care, higher percent of pre-pregnancy diabetes, hypertension or gestational hypertension (all contribute to birthweight and prematurity) and had a higher percent reporting at least 1 STI during pregnancy. There is no infant sex difference by exposure status. Additionally, all outcomes were higher among Whites ($p<0.0001$ for all outcomes), and BW and SzGA were higher among those unexposed ($p<0.0001$). While differences by exposure status noted above are statistically significant, the absolute differences in values are small in magnitude due to the large sample size.

2.3.a. Primary Regression Analysis

Table 2.2 displays the predicted means of BW, GA, and SzGA (z-score), and the p-values for the FWC declaration exposure, race, and their interaction among births to Black and White mothers (See Figure 2.6 for a graphical depiction of the results). Infants born in the 37 weeks after the governor's FWC declaration had significantly lower BWs and lower SzGA (exposure: $p<0.0001$ in both Models 1 and 2 for BW and SzGA) than infants born during the same 37 weeks in 2013-

2015. After adjustment of all covariates, the decline in BW was 25.2g among babies born to Black mothers (Model 3 unexposed: 3,099.1g (95% CI: 3,055.5, 3,142.5); exposed to the FWC declaration: 3,073.9g (95% CI: 3,029.6, 3,118.1)) and 18.4g among infants born to White mothers (unexposed: 3,318.7g (95% CI: 3,275.8, 3,361.6); exposed: 3,300.3g (95% CI: 3,257.2, 3,343.4)). The difference in SzGA (z-score) was 0.04 among babies born to both Black (unexposed: -0.32 (95% CI: -0.40, -0.24); exposed: -0.36 (95% CI: -0.44, -0.28)) and White (unexposed: 0.03 (95% CI: -0.05, - 0.11); exposed: -0.01 (95% CI: -0.08, 0.07)) mothers. Despite significantly lower BWs for babies born to Black mothers compared to White mothers in both the unexposed (219.6g) and exposed periods (226.4g), the interaction between race and exposure to the FWC declaration was not statistically significant (p=0.3160). Similarly, the interaction between race and exposure to the FWC declaration was not statistically significant for SzGA (p=0.8835), despite differences in SzGA by the FWC declaration exposure status and race.

In contrast, the interaction between the FWC declaration and race was marginally significant for GA in the adjusted model (Model 3 race*exposure p=0.0812). In the adjusted model, GA was 0.04 weeks lower among exposed to the FWC declaration vs. unexposed babies born to Black mothers (unexposed: 38.38 weeks (95% CI: 38.23, 38.54); exposed: 38.34 weeks (95% CI: 38.18, 38.50)); whereas, there was no difference in GA for babies born to White mothers following the FWC declaration (unexposed: 38.79 (95% CI: 38.64, 38.94); exposed 38.79 (95% CI: 38.64, 38.94)).

2.3.b. Sensitivity Analyses

In the first sensitivity analysis I include hypothesized mediators in the model (Table 2.3). While the results are nearly identical for BW and SzGA, they slightly differ for GA. After including adequacy of prenatal care, tobacco use, alcohol use, gestational diabetes, pre-pregnancy diabetes or hypertension, prior preterm births, and reporting at least 1 STI during pregnancy, the interaction between race and exposure becomes statistically significant (Model 4 race*exposure: p=0.0158). GA was 0.06 weeks lower for FWC declaration exposed vs. unexposed babies born to Black mothers (unexposed: 37.37 weeks (95% CI: 37.20, 37.54); exposed: 37.31 weeks (95% CI: 37.13, 37.49)); whereas there was no difference in GA for babies born to White mothers following the FWC declaration.

2.3.b.1. Pairwise Analysis

Infants born in the 37 weeks after the FWC declaration had significantly lower BW (exposure: $p<0.0001$ in all models) and SzGA (exposure: $p<0.0001$ in all models) than infants born during the same 37 weeks in 2013--the only year that completely predates the FWC (Table 2.4). After adjustment of covariates, there was a statistically significant difference in BW and SzGA by race (race: $p<0.0001$ for both outcomes, Models 1 and 2) but the interaction between exposure and race was not statistically significant for either outcome (Model 3: race*exposure: $p=0.3669$ (BW); $p=0.5658$ (SzGA)). This pattern was similar to the main analysis (Table 2.2). In contrast, there was a significant interaction between exposure and race for GA in Model 3 (race*exposure: $p=0.0056$). In the adjusted model, GA was 0.08 weeks lower among babies born to Black mothers following the FWC declaration (unexposed: 38.28 weeks (95% CI: 38.06, 38.50); exposed: (38.20 weeks (95% CI: 37.98, 38.42)); whereas, there was a slight increase in GA for babies born to White mothers following the declaration (unexposed: 38.65 weeks (95% CI:38.43, 38.87); unexposed: (38.66 weeks (95% CI: 38.44, 38.88)).

The data presented in Table 2.5 and Table 2.6 represent similar patterns to the primary analysis in that there are racial and FWC declaration exposure status differences, but no significant race by exposure interactions for BW, GA or SzGA in the model comparing 2014 to 2016 or in the model comparing 2015 to 2016.

2.3.b.2. Disaggregated Unexposed

The data presented in Table 2.7 displays the regression coefficients and predicted means for the BW outcome when the unexposed group is disaggregated by year of birth (2013, 2014, or 2015) and compared to the FWC declaration exposed group (born in 2016). These results are similar to the main analysis. Both Black and White babies born in the 37 weeks following the FWC declaration had lower BW compared to the same 37 weeks in each year before the FWC declaration (Model 2 exposure: $p<0.0001$ for each year). Additionally, babies born to Black mothers in the 37 weeks following the FWC declaration have lower BW compared to each of the prior years in the adjusted model (Model 2 race: $p<0.0001$), however the relationship between the FWC declaration and BW does not differ race (Model 3 race*exposure interactions all p-values are not statistically significant). The similar BW slopes between Black and White mothers are also observed graphically in Figure 2.7.

Table 2.8 displays the regression coefficients and predicted means for the GA outcome. In this model I observe that there is no difference in GA between exposed and unexposed years (Model 2 exposure coefficients where all p-values are not statistically significant). Babies born to Black mothers have lower GAs each of the prior years in the adjusted model (Model 2 race: $p<0.0001$), however the relationship between the FWC declaration and GA differs by race for only the 2013 unexposed year compared to 2016 (Model 3 race*exposure $p=0.0035$ for 2013 and not significant for 2014 and 2015) despite the predicted means appearing to decline each year for babies born to Black mothers and appearing unchanged for babies born to White mothers.

Table 2.9 displays the regression coefficients and predicted means for the SzGA outcome disaggregated by year. Similar to the BW results, both Black and White babies born in the 37 weeks following the FWC declaration had lower SzGA compared to the same 37 weeks in each year before the FWC declaration (Model 2 exposure: $p<0.0001$for each year). Additionally, babies born to Black mothers in the 37 weeks following the FWC declaration have lower SzGA compared to each of the prior years in the adjusted model (Model 2 race: $p<0.0001$). Finally, the relationship between SzGA and the FWC declaration is not dependent on race (Model 3 race*exposure interactions are not statistically significant for each year compared to 2016).

2.3.b.3. Binary Outcomes

Infants born within the 37 weeks following the FWC declaration had higher odds of LBW. In the unadjusted and adjusted models (Models 1 and 2, Table 2.10), the FWC declaration was associated with statistically significantly higher odds of LBW, PTB, and SmGA, with the exception of the adjusted models for PTB (Model 2). After adjustment of covariates, babies born to Black mothers had 0.9 percentage points higher proportion of LBW babies (unexposed: 12.8% (95% CI: 10.3, 15.8); unexposed 13.7% (95% CI: 11.0, 16.9)), a 0.3 percentage points higher proportion of PTB (unexposed: 10.9% (95% CI: 8.5, 13.9); exposed: 11.2% (95% CI: 8.7, 14.3)), and 0.5 percentage points higher proportion of SmGA (unexposed: 16.9% (95% CI: 13.9, 20.5); exposed: 16.4% (95% CI: 15.1, 22.3)). Similarly, babies born to White mothers had 0.4 percentage points higher proportion of LBW before the FWC declaration compared to after it (unexposed: 6.8% (95% CI: 5.4, 8.5); exposed 7.2% (95% CI: 5.7, 9.0)), a 0.2 percentage points higher proportion of PTB (unexposed: 7.2 (95% CI: 5.5, 9.2); exposed: 7.4 (95% CI: 5.7, 9.5)), and 0.4 percentage points higher proportion of SmGA (unexposed: 10.0 (95% CI: 8.1, 12.3); exposed: 10.4 (95% CI: 8.4, 12.9)). Consistent with the main analysis there was no statistically

significant race*exposure interactions for LBW or SmGA (Model 3 race*exposure: p=0.7351 (LBW); p=0.1106 (SmGA)). However, the results for PTB (Model 3 race*exposure: p=0.9735) were not consistent with the main findings for GA, which found a marginally statistically significant race*exposure interaction (Model 3, Table 2.2).

2.3.b.4. Trimester of Exposure

I compared birth outcomes for babies who were exposed in a specific trimester during the 37 weeks following the FWC declaration compared to women unexposed but in the same trimester in the same 37 weeks in 2013 through 2015. Mothers exposed to the FWC declaration in their first trimester had similar results for BW and SzGA to the main analysis (Table 2.11). Babies born to both Black and White mothers who were exposed during their first trimester had lower BWs, lower GAs, and lower SzGAs after the FWC declaration and there were no statistically significant interactions for any outcome (Model 3 race*exposure: p=0.6326 (BW); p=0.8255 (GA); p=0.2862 (SzGA)). There were similar results for mothers exposed in their second trimester (Table 2.12) compared to mothers unexposed in their second trimester. In contrast, there was a non-significant interaction for mothers exposed in their third trimester for BW (Model 3 race*exposure: p=0.7166; see Table 2.13), a significant interaction for GA (Model 3 race*exposure: p=0.0039), and a marginally significant interaction for SzGA (Model 3 race*exposure: p=0.0726).

2.4 Discussion

The first aim of this study was to examine the association between the declaration of a state of emergency in Flint, Michigan on birth outcomes among babies born to Black and White mothers in Michigan, but outside of Flint. In order to evaluate this aim I hypothesized that there would be changes in birth outcomes in the 37 weeks following the FWC declaration for babies born to both Black and White mothers in Michigan, but outside of Flint. The data presented in this study provide ample support for this hypothesis. In every model, for BW and SzGA, I found that there were statistically significant or marginally significant associations for the FWC declaration and birth outcomes, where BW and SzGA were lower after the FWC declaration than before it (Tables 2.2 – 2.13, Figures 2.6 -2.7). This suggests that vicariously observing the FWC through a sharp rise in news stories after the FWC declaration (Figure 2.2), which occurred 1.5 years after the untreated water was switched back to clean water, affects birth outcomes in Michigan for babies born to both Black and White mothers. I confirmed these findings in nearly all

sensitivity analyses, thus, the support for this hypothesis is robust to various ways to examine the effect of the FWC declaration on birth outcomes. The FWC and the FWC declaration in Flint were, and likely remain, stressful events for both Black and White women in Michigan. Various forms of stress, including stress resulting from both natural and man-made disasters, as well as those experienced directly or indirectly (vicariously) are associated with a higher risk or odds of poor birth outcomes.[78,79,296,304,311,313,316,317,320,402,403]

The second aim examined effect modification between the FWC declaration and race on birth outcomes. I hypothesized that changes in birth outcomes following the FWC declaration would be more harmful to Black mothers than to White mothers or I would observe significant effect modification between race and exposure. My analysis revealed less support for this hypothesis. In the main analysis, a marginally significant interaction was observed only for GA (Table 2.2). However, I did not observe any significant interactions when examining the standard binary outcomes of LBW, PTB, and SmGA (Table 2.10) in a sensitivity analysis. In other sensitivity analyses, I observed a statistically significant decrease in GA among babies born to Black mothers following the FWC declaration compared to both unexposed Black mothers and White mothers whose babies were born in 2013 (Table 2.4). Additionally, when comparing the disaggregated FWC declaration exposure by year, I observed a statistically significant race by exposure interaction in the adjusted model (Model 3) for the 2013 (compared to 2016) year with a gradual decline in GA for babies born to Black mothers over the 4 years and virtually no change over time for babies born to White mothers (Table 2.8) a trend observed nationally as well.[404] Given that during the study years there were several high-profile incidents of violence inflicted on Black men and women at the same time as the FWC—both the water portion of the crisis and during the spike in media attention after the FWC declaration (Figures 2.3 and 2.8) – this suggests that these other racialized co-occurring stressors, in addition to the racialized FWC, may have played a role in these birth outcome decreases.

This second hypothesis testing a racial disparity in birth outcomes associated with the FWC declaration is also supported by the scant literature. Novak et. al., reported a higher risk of LBW and PTB for babies born to Iowa Latina mothers and no change in birth outcomes were observed for babies born to White Iowa mothers after an immigration raid at a food processing plant.[78] Additionally, Lauderdale et. al. (2006), using birth records from California, observed an

increased risk in LBW and PTB in babies born to women of Arabic decent after vicariously experiencing the 9/11 World Trade Center attacks in 2001. This association was observed to be more harmful to women naming their newborns traditional or ethnically Arabic names.[79] This finding, among others focusing on a vicarious racially motivated exposure, are consistent with our study.[316,405,406] Other studies have reported no racial disparities in birth outcomes as a result of a vicarious traumatic or racialized stressors.[313,323]

The results of our study suggest that as exposures occur later in the pregnancy, there is a higher risk to Black women for delivering babies early (Tables 2.11-2.13). Table 2.13 demonstrates that babies born to Black women who were exposed to the declaration of a state of emergency in Flint during their third trimester of pregnancy delivered babies 0.11 weeks earlier than those unexposed with no change among babies born to White mothers. This finding is somewhat consistent with the mixed literature on this topic. For example, Bakker et. al. (2011) reported a nearly 6-fold higher odds of PTB with acute blood pressure changes in the 2nd and 3rd trimester of pregnancy.[407] Other studies support that a stressful exposure during the 2nd or 3rd trimester may affect fetal growth.[302,408,409] Class et. al., suggests that a stressful exposure starting at the 5th or 6th month of pregnancy can result in lower birthweights, and shorter pregnancies, consistent with my study.[302] Still other studies suggest that exposure in the 1st trimester is more harmful, which was not observed in this study.[410-412]

2.4.a. Limitations and Strengths

This study is not without limitations. First, there is the potential for exposure misclassification. It is possible that women in Michigan knew about the FWC before the FWC declaration. I examined this potential error by including births from 2013 (Table 2.6). The 2013 births completely pre-date the period of contaminated water exposure from the FWC. I found that the largest decline in GA was observed when comparing births in 2013 to births in 2016 (Table 2.6). If exposure misclassification is present, it is unlikely due to the timing of the declaration. Evidence from traditional media mentions, Google searches, and tweets suggest that the timing of the FWC declaration coincided with a sharp spike in news reporting and social media mentions of Flint (Figure 2.3).[344] Another possible source of exposure misclassification is that there may be differential exposure by race, where Black women may have been more likely to hear about the FWC prior to the emergency declaration compared to White women. A recent

report suggests that women and nonwhite persons are more likely to obtain their news from local television, but there are no racial or gender differences for network and cable television.[413,414] Given that earliest media coverage occurred locally and that there was a small spike in local coverage in late 2015 according to a recent PEW report it is possible that Black women had heard of the FWC before the FWC declaration was made.[344]

Second, the FWC is not a discrete event. In fact, the contamination of the water occurred over about 1.5 years before the governor declared a state of emergency in Flint, which was followed by several months of substantial media attention (Figure 2.3). There may have been awareness of the FWC before the declaration, especially if the woman had a personal connection to Flint which could lead to differential misclassification of the exposure which could bias the estimate in an unpredictable way.

Third, women directly exposed to the FWC may leave Flint but stay in Michigan during the study period leading to babies born who were directly exposed to the contaminated water, which could lead to poor birth outcomes due to lead and not vicarious exposure to the FWC. Additionally, if women outside of Flint leave the state of Michigan during the study period, this could affect the results. The population in Michigan increased slightly over this period, approximately 0.24% (23,505 people) between 2013 and 2016. The population in Flint decreased by approximately 2.7% (2,731 people).[c] Additionally, there was a decline in the population of White women by about 1.8% and for Black women it was 2.3% in Michigan. Similar data for both Black and White women in Flint city are not available. If the population changes occurred before the FWC declaration (but remained in MI), this may inflate the number of mothers with poor birth outcomes (due to direct exposure to the FWC) in the unexposed period, while if it occurs after, it may increase the number of mothers with poor birth outcomes in the exposed group.

Fourth, given that the FWC has been determined to be the result of systemic structural racism,[158] it is possible that other nationally recognized racialized stressors were co-occurring around the same time that could have contributed to worse birth outcomes for main effects. During 2013

[c] US Census, American Community Survey 1-year estimates 2013-2016

and beyond, the FWC and the exposure period overlaps with several police involved killings of Black people across the US, police violence, or other forms of structural racism – many high profile violent acts against People of Color occurred during this time (see Figure 2.8). Since at least 2013, crowdsourced data on police-involved killings generally agree that police kill about 1,000 Black and Brown people each year. These numbers appear to be declining, however cases that were highly publicized during this time appear to have remained steady in the news cycles suggesting that vicarious exposure to police violence was consistent across the study period (see Fatal Encounters/Mapping Police Violence https://mappingpoliceviolence.org/). Other racialized stressors such as a racially charged national election also occurred during this time. Thus, I cannot rule out that other social stressors could have contributed to worse birth outcomes for main effects. Nonetheless, I am unable to disentangle the effects of the structural racism associated with these killings and the media attention for the FWC. Our results indicate that the FWC likely affected mean BW and SzGA for Blacks and Whites, but the results suggest that the racial difference in the changes in the outcomes may be related to other racialized risks.

Finally, research suggests that there are wide estimates of reliability and validity among *some* elements in the birth certificate.[415] It is reported that mother's race/ethnicity is generally reliable, especially for Blacks and Whites, as are birthweight and insurance status, whereas substance use, prenatal care and GA are deemed less reliable.[416,417] In 2014 the standard birth certificate changed from a GA based on the date of last menstrual period to obstetric observed GA resulting in fewer newborns being labeled as preterm mainly affecting Black, Hispanic and American Indian/Alaskan Native newborns.[418] To examine this possibility a GA cleaning algorithm was employed which accounted for these potential differences due to reporting.[419]

Despite these limitations, there are strengths worth noting. This study assumes that the effect of the news of the FWC declaration operates through a stress mechanism to affect birth outcomes. There is a substantial body of literature suggesting a biologic plausibility of the effect of stress on birth outcomes, specifically related to GA, BW, and SzGA.[355-359,420] This literature is supported by the epidemiologic and social science literature which suggests that stress and chronic worry about racial discrimination, among other concerns, affects GA.[317,410,412,421-423] This is consistent with the "fetal origins hypothesis" which posits that prenatal environmental exposures may have lasting effects across the life, including while *in utero*, which could result in

poor birth outcomes or other health conditions later in life.[424] Stress as an environmental exposure *in utero* has physiological consequences to the growing fetus via releases of hormones targeting the pituitary gland to release adrenocorticotropic hormones (ACTH). These hormones are responsible for regulating pregnancy duration and fetal development as well as the synthesis of glucocorticoids into the bloodstream, such as cortisol—the stress hormone—which crosses the placenta and could affect the growth of the fetus. [424-426]

This study relied on birth outcomes, which are sensitive to stressors.[356,427] Additionally, these outcomes were continuous outcomes that were objectively measured. The use of continuous measures enables us to examine smaller changes in BW, GA, and SzGA that would be masked by binary outcomes using cut-points. Additionally, there is evidence that the birth outcomes listed on the birth certificate are reliably measured.[400] Thus, even with the noted limitations of the reliability of some values recorded on birth records, the use of vital statistics for the state of Michigan is one the strengths of this study. Research suggests that the data on BW is highly accurate for both Blacks and Whites.[400]

I also restricted births to those that did not occur in Flint. Babies born in Flint during this time would have been exposed at some level to the contaminated water which would have impacted birth outcomes.[428] This exclusion allows us to quantify the vicarious exposure to the FWC.

The main strength lies within the innovation of this study. This study is the first to examine the association of the FWC on those not directly exposed to the contaminated water in Flint. This study adds to the small body of literature that collectively suggests that racially motivated events or responses to natural and man-made disasters have a negative effect on pregnant women and their newborns even if the women were not directly exposed to the disaster.[78,79,315-322] Additionally, there are few studies that empirically examine the effects of vicarious racism, thus this study fills a gap in the literature by doing so. Most notably, vicarious racism could happen anywhere, the health effects of this form of racism in one area can affect the health in another. Studies such as these both demonstrate this risk, but also highlight the need for new methods to examine vicarious racism.

2.5 Conclusion

The FWC was an extremely stressful man-made disaster for all affected, especially those directly affected by the contaminated water.[158,347,429-432] However, fears and distrust of institutions did not stop at the borders of the city of Flint. In fact, our study suggests that a vicarious exposure to Flint may have contributed to smaller babies being born earlier than had they not been exposed to the widespread media attention focused on Flint. This study suggests that the FWC raised concerns and worry for Black and White mothers across the state of Michigan, but at least for GA, the FWC may been more stressful and thus more harmful for Black mothers. This study illustrates the need for more studies on vicarious structural racism given the current state of race relations in the United States with increased media attention devoted to racialized environmental disasters, police killings of unarmed Black and Brown people, and the harmful political rhetoric stemming from our leaders. Racism has been called out as a public health crisis (the first government entity to do so was Milwaukee County and Flint City did so in June 2020),[39,66,433] but more evidence is needed to understand its effect on health outcomes.

2.6 Tables and Figures

Table 2. 1 Descriptive Statistics by Maternal Race and Exposure to the Flint Water Crisis Emergency Declaration in Michigan (n=226,672).

	Total n=226,672	Non-Hispanic Black n=45,613	Non-Hispanic White n=181,059	*P*-value	Unexposed n=171,328	Exposed n=55,344	*P*-value
Maternal Race, n (%)							
Non-Hispanic Black	45,613 (20.1)				34,540 (20.2)	11,073 (20.0)	0.4364
Non-Hispanic White	181,059 (79.9)				136,788 (79.8)	44,271 (80.0)	
Maternal Age, mean (SD)	28.13 (5.66)	26.18 (5.93)	28.62 (5.48)	**<0.0001**	28.08 (5.69)	28.27 (5.58)	**<0.0001**
Maternal Education, n (%)							
Less than High School	23,074 (10.2)	7,916 (17.4)	15,158 (8.4)	**<0.0001**	17,786 (10.4)	5,288 (9.6)	**<0.0001**
High School/GED	56,426 (24.9)	16,120 (35.3)	40,306 (22.3)		42,712 (24.9)	13,714 (24.8)	
Some College	58,811 (25.9)	14,067 (30.8)	44,744 (24.7)		45,031 (26.3)	13,780 (24.9)	
College or More	86,958 (38.4)	6,897 (15.1)	80,061 (44.2)		64,897 (37.9)	22,061 (39.9)	
Unknown	1,403 (0.6)	613 (1.3)	790 (0.4)		902 (0.5)	501 (0.9)	
Maternal Marital Status, n (%)							
Never Married	90,073 (39.7)	35,626 (78.1)	54,447 (30.1)	**<0.0001**	68,116 (39.8)	21,957 (39.7)	**0.9417**
Married	129,535 (57.1)	9,194 (20.2)	120,341 (66.5)		97,869 (57.1)	31,666 (57.2)	
Divorced/Widowed	7,005 (3.1)	773 (1.7)	6,232 (3.4)		5,300 (3.1)	1,705 (3.1)	
Unknown	59 (0.0)	20 (0.0)	39 (0.0)		43 (0.0)	16 (0.0)	
Source of Payment for Delivery, n (%)							
Private Insurance	127,197 (56.1)	15,941 (34.9)	111,256 (61.4)	**<0.0001**	95,814 (55.9)	31,383 (56.7)	**<0.0001**
Medicaid	93,369 (41.2)	28,001 (61.4)	65,368 (36.1)		70,660 (41.2)	22,709 (41.0)	
Self-Pay	3,198 (1.4)	485 (1.1)	2,713 (1.5)		1,788 (1.0)	298 (0.5)	
Other	2,086 (0.9)	1,069 (2.3)	1,017 (0.6)		2,379 (1.4)	819 (1.5)	
Unknown	822 (0.4)	117 (0.3)	705 (0.4)		687 (0.4)	135 (0.2)	
Receipt of WIC during Pregnancy, n (%)							
Yes	90,749 (40.0)	29,871 (65.5)	60,878 (33.6)	**<0.0001**	70,363 (41.1)	20,386 (36.8)	**<0.0001**
No	132,620 (58.5)	14,923 (32.7)	117,697 (65.0)		98,396 (57.4)	34,224 (61.8)	
Unknown	3,303 (1.5)	819 (1.8)	2,484 (1.4)		2,569 (1.5)	734 (1.3)	

Pre-pregnancy BMI, mean (SD)	27.22 (6.91)	28.88 (7.73)	26.82 (6.64)	**<0.0001**	27.18 (6.90)	27.35 (6.95)	**<0.0001**
Parity (including birth on record), mean (SD)	2.49 (1.70)	2.83 (1.20)	2.41 (1.61)	**<0.0001**	2.52 (1.70)	2.42 (1.70)	**<0.0001**
Infant Sex, n (%)							
Female	110,644 (48.8)	22,592 (49.5)	88,052 (48.6)	**0.0006**	83,698 (48.9)	26,946 (48.7)	0.5014
Male	116,028 (51.2)	23,021 (50.5)	93,007 (51.4)		87,630 (51.1)	28,398 (51.3)	
Residential Geographic Region, n (%)							
Upper Peninsula Prosperity Alliance	5,689 (2.5)	25 (0.1)	5,664 (3.1)	**<0.0001**	4,353 (2.5)	1,336 (2.4)	0.1505
Northwest Prosperity Region	6,363 (2.8)	36 (0.1)	6,327 (3.5)		4,777 (2.8)	1,586 (2.9)	
Northeast Prosperity Region	3,800 (1.7)	19 (0.0)	3,781 (2.1)		2,896 (1.7)	904 (1.6)	
West Michigan Prosperity Alliance	37,843 (16.7)	3,753 (8.2)	34,090 (18.8)		28,649 (16.7)	9,194 (16.6)	
East Central Michigan Prosperity Region	12,113 (5.3)	1,481 (3.2)	10,632 (5.9)		9,194 (5.4)	2,919 (5.3)	
East Michigan Prosperity Region	17,133 (7.6)	1,345 (2.9)	15,788 (8.7)		12,956 (7.6)	4,177 (7.5)	
South Central Prosperity Region	10,381 (4.6)	1,537 (3.4)	8,844 (4.9)		7,779 (4.5)	2,602 (4.7)	
Southwest Prosperity Region	19,023 (8.4)	2,952 (6.5)	16,071 (8.9)		14,480 (8.5)	4,543 (8.2)	
Southeast Michigan Prosperity Region	21,567 (9.5)	1,895 (4.2)	19,672 (10.9)		16,332 (9.5)	5,234 (9.5)	
Detroit Metro Prosperity Region	92,760 (40.9)	32,570 (71.4)	60,190 (33.2)		69,911(40.8)	22,849 (41.3)	
Kessner Index, n (%)							
Adequate	154,832 (68.3)	25,216 (55.3)	129,616 (71.6)	**<0.0001**	116,178 (67.8)	38,654 (69.8)	**<0.0001**
Intermediate	51,428 (22.7)	13,323 (29.2)	38,105 (21.0)		39,429 (23.0)	11,999 (21.7)	
Inadequate	19,472 (8.6)	6,654 (14.6)	12,818 (7.1)		14,781 (8.6)	4,691 (8.5)	
Unknown	940 (0.4)	420 (0.9)	520 (0.3)		940 (0.5)	0 (0.0)	
Tobacco Use during Pregnancy, n (%)							
Yes	48,140 (21.2)	7,851 (17.2)	40,289 (22.3)	**<0.0001**	37,367 (21.8)	10,773 (19.5)	**<0.0001**

No	177,610 (78.4)	37,481 (82.2)	140,129 (77.4)		133,356 (77.8)	44,254 (80.0)	
Unknown	922 (0.4)	281 (0.6)	641 (0.4)		605 (0.4)	317 (0.6)	
Alcohol Use during Pregnancy, n (%)							
Yes	1,654 (0.7)	340 (0.7)	1,314 (0.7)	**<0.0001**	1,241 (0.7)	413 (0.7)	**<0.0001**
No	222,676 (98.2)	45,115 (98.9)	177,561 (98.1)		168,536 (98.4)	54,140 (97.8)	
Unknown	2,342 (1.0)	158 (0.3)	2,184 (1.2)		1,551 (0.9)	791 (1.4)	
Gestational Diabetes, n (%)							
Yes	10,955 (4.8)	1,628 (3.6)	9,327 (5.2)	**<0.0001**	8,295 (4.8)	2,660 (4.8)	**0.0009**
No	215,104 (94.9)	43,893 (96.2)	171,211 (94.6)		162,530 (94.9)	52,574 (95.0)	
Unknown	613 (0.3)	92 (0.2)	521 (0.3)		503 (0.3)	110 (0.2)	
Pre-Pregnancy Diabetes, Pre-Pregnancy Hypertension and/or Gestational Hypertension, n (%)							
Yes	17,739 (7.8)	3,655 (8.0)	14,084 (7.8)	**0.0018**	12,723 (7.4)	5,016 (9.1)	**<0.0001**
No	208,320 (91.9)	41,866 (91.8)	166,454 (91.9)		158,102 (92.3)	50,218 (90.7)	
Unknown	613 (0.3)	92 (0.2)	521 (0.3)		503 (0.3)	110 (0.2)	
Previous Preterm Birth and/or Other Previous Poor Pregnancy Outcome, n (%)							
Yes	9,287 (4.1)	1,952 (4.3)	7,335 (4.1)	**0.0005**	7,467 (4.4)	1,820 (3.3)	**<0.0001**
No	216,767 (95.6)	43,569 (95.5)	173,198 (95.7)		163,357 (95.3)	53,410 (96.5)	
Unknown	618 (0.3)	92 (0.2)	526 (0.3)		504 (0.3)	114 (0.2)	
At Least One STI during Pregnancy, n (%)							
Yes	49,291 (21.7)	12,536 (27.5)	36,755 (20.3)	**<0.0001**	36,626 (21.4)	12,665 (22.9)	**<0.0001**
No	173,353 (76.5)	32,717 (71.7)	140,636 (77.7)		131,742 (76.9)	41,611 (75.2)	
Unknown	4,028 (1.8)	360 (0.8)	3668 (2.0)		2,960 (1.7)	1,068 (1.9)	
Birthweight in Grams, mean (SD)	3331.6 (571.1)	3110.8 (603.9)	3387.2 (548.7)	**<0.0001**	3335.7 (569.4)	3319.0 576.2)	**<0.0001**

Gestational Age in Weeks, mean (SD)	38.76 (2.00)	38.33 (2.44)	38.87 (1.86)	**<0.0001**	38.76 (1.99)	38.75 (2.04)	0.4230
Size for Gestational Age Z-score, mean (SD)	0.05 (1.03)	-0.29 (0.99)	0.14 (1.02)	**<0.0001**	0.06 (1.03)	0.03 (1.0)	**<0.0001**

Bolded *P-values* denote statistical significance at $\alpha = 0.05$. SD=standard deviation

Table 2. 2 Linear Regression Analysis of Exposure to the Flint Water Crisis Emergency Declaration in Michigan: Predicted Means and 95% Confidence Intervals (CI) for Birthweight, Gestational Age, and Size-for-Gestational-Age (n=226,672).

	Unexposed[a] n=171,328		Exposed[a] n=55,344		Race x Exposure	Race	Exposure
	Non-Hispanic Black n=34,540	Non-Hispanic White n=136,788	Non-Hispanic Black n=11,073	Non-Hispanic White n=44,271	*P-value*	*P-value*	*P-value*
Birthweight, grams (95% CI)							
Model 1[b]	3,117.3 (3,111.0, 3,123.7)	3,390.8 (3,387.9, 3,393.7)	3,090.5 (3,079.3, 3,101.8)	3,376.2 (3,371.1, 3,381.3)	NA	**<0.0001**	**<0.0001**
Model 2[c]	3,127.3 (3,022.9, 3,223.7)	3,296.3 (3,248.3, 3,344.4)	3,103.9 (2,999.0, 3,103.9)	3,277.7 (3,229.5, 3,325.9)	NA	**<0.0001**	**<0.0001**
Model 3[d]	3,099.1 (3,055.8, 3,142.5)	3,318.7 (3,275.8, 3,361.6)	3,073.9 (3,029.6, 3,118.1)	3300.3 (3,257.2, 3,343.4)	0.3160	**<0.0001**	**<0.0001**
Gestational Age, weeks (95% CI)							
Model 1[b]	38.34 (38.31, 38.37)	38.87 (38.86, 38.88)	38.27 (38.23, 38.32)	38.87 (38.86, 38.89)	NA	**<0.0001**	0.3751
Model 2[c]	38.29 (37.87, 38.71)	38.78 (38.62, 38.95)	38.25 (37.83, 38.68)	38.78 (38.62, 38.95)	NA	**<0.0001**	0.3520
Model 3[d]	38.38 (38.23, 38.54)	38.79 (38.64, 38.94)	38.34 (38.18, 38.50)	38.79 (38.64, 38.94)	0.0812	**<0.0001**	0.9427
Size-for-Gestational-Age z-score, mean (95% CI)							
Model 1[b]	-0.29 (-0.30, -0.27)	0.14 (0.14, 0.15)	-0.32 (-0.34, -0.30)	0.11 (0.10, 0.12)	NA	**<0.0001**	**<0.0001**
Model 2[c]	-0.19 (-0.36, -0.02)	-0.03 (-0.12, 0.06)	-0.23 (-0.40, -0.05)	-0.06 (-0.15, 0.03)	NA	**<0.0001**	**<0.0001**
Model 3[d]	-0.32 (-0.40, -0.24)	0.03 (-0.05, 0.11)	-0.36 (-0.44, -0.28)	-0.01 (-0.08, 0.07)	0.8835	**<0.0001**	**<0.0001**

[a] Exposed infants were born between 1/5/2016 and 9/30/2016; unexposed infants were born in the same 37-week period in 2013, 2014, or 2015.

[b] Linear model (1) includes race and exposure.

[c] Linear model (2) adjusted for covariates: Model 1 + covariates (maternal age, education, marital status, source of payment for delivery, receipt of WIC during pregnancy, pre-pregnancy BMI, parity, infant sex, and residential geographic region).

[d] Linear model (3) including Model 2 + interaction between race and exposure

Bolded *P-values* denote statistical significance at $\alpha = 0.05$.

Table 2. 3 Linear Regression Analysis of Exposure to the Flint Water Crisis Emergency Declaration: Predicted Means and 95% Confidence Intervals for Birthweight, Gestational Age, and Size for Gestational Age (n=226,672).

	Unexposed[a] n=171,328		Exposed[a] n=55,344	Race x Exposure		Race	Exposure
	Non-Hispanic Black n=34,540	**Non-Hispanic White n=136,788**	**Non-Hispanic Black n=11,073**	**Non-Hispanic White n=44,271**	***P-value***	***P-value***	***P-value***
Birthweight, grams (95% CI)							
Model 1[b]	3,117.3 (3,111.0, 3,123.7)	3,390.8 (3,387.9, 3,393.7)	3,090.5 (3,079.3, 3,101.8)	3,376.2 (3,371.1, 3,381.3)	NA	**<0.0001**	**<0.0001**
Model 2[c]	3,127.3 (3,022.9, 3,223.7)	3,296.3 (3,248.3, 3,344.4)	3,103.9 (2,999.0, 3,103.9)	3,277.7 (3,229.5, 3,325.9)	NA	**<0.0001**	**<0.0001**
Model 3[d]	3,099.1 (3,055.8, 3,142.5)	3,318.7 (3,275.8, 3,361.6)	3,073.9 (3,029.6, 3,118.1)	3300.3 (3,257.2, 3,343.4)	0.3160	**<0.0001**	**<0.0001**
Model 4[e]	2,849.3 (2,800.4, 2,898.1)	3,089.5 (3,041.1, 3,138.0)	2,818.1 (2,768.5, 2,867.8)	3068.6 (3,019.9, 3,117.2)	0.1314	**<0.0001**	**<0.0001**
Gestational Age, weeks (95% CI)							
Model 1[b]	38.34 (38.31, 38.37)	38.87 (38.86, 38.88)	38.27 (38.23, 38.32)	38.87 (38.86, 38.89)	NA	**<0.0001**	0.3751
Model 2[c]	38.29 (37.87, 38.71)	38.78 (38.62, 38.95)	38.25 (37.83, 38.68)	38.78 (38.62, 38.95)	NA	**<0.0001**	0.3520
Model 3[d]	38.38 (38.23, 38.54)	38.79 (38.64, 38.94)	38.34 (38.18, 38.50)	38.79 (38.64, 38.94)	0.0812	**<0.0001**	0.9427
Model 4[e]	37.37 (37.20, 37.54)	37.81 (37.64, 37.99)	37.31 (37.13, 37.49)	37.81 (37.64, 37.98)	**0.0158**	**<0.0001**	0.8060
Size-for-Gestational-Age z-score, mean (95% CI)							
Model 1[b]	-0.29 (-0.30, -0.27)	0.14 (0.14, 0.15)	-0.32 (-0.34, -0.30)	0.11 (0.10, 0.12)	NA	**<0.0001**	**<0.0001**
Model 2[c]	-0.19 (-0.36, -0.02)	-0.03 (-0.12, 0.06)	-0.23 (-0.40, -0.05)	-0.06 (-0.15, 0.03)	NA	**<0.0001**	**<0.0001**
Model 3[d]	-0.32 (-0.40, -0.24)	0.03 (-0.05, 0.11)	-0.36 (-0.44, -0.28)	-0.01 (-0.08, 0.07)	0.8835	**<0.0001**	**<0.0001**
Model 4[e]	-0.48 (-0.57, -0.39)	-0.09 (-0.18, 0.00)	-0.52 (-0.61, -0.43)	-0.13 (-0.22, -0.04)	0.7750	**<0.0001**	**<0.0001**

[a] Exposed infants were born between 1/5/2016 and 9/30/2016; unexposed infants were born in the same 37-week period in 2013, 2014, or 2015.

[b] Linear model (1) includes race and exposure.

[c] Linear model (2) adjusted for covariates: Model 1 + covariates (maternal age, education, marital status, source of payment for delivery, receipt of WIC during pregnancy, pre-pregnancy BMI, parity, infant sex, and residential geographic region).

[d] Linear model (3) including Model 2 + interaction between race and exposure

[e] Linear model (4) including Model 4 + hypothesized mediators (Adequacy of prenatal care, mom's smoking status, mom's alcohol intake, gestational diabetes, diabetes/hypertension, prior poor birth outcome, and sexually transmitted infection during pregnancy)

Bolded *P-values* denote statistical significance at $\alpha = 0.05$.

Table 2. 4 Linear Regression Analysis of Exposure to the Flint Water Crisis Emergency Declaration: Predicted Means and 95% Confidence Intervals for Birthweight, Gestational Age, and Size-for-Gestational-Age, 2013 vs. 2016 (n=105,418).

	Unexposed: 2013[a] n=55,074		Exposed: 2016[a] n=55,344		Race x Exposure	Race	Exposure
	Non-Hispanic Black n=11,444	Non-Hispanic White n=43,630	Non-Hispanic Black n=11,073	Non-Hispanic White n=44,271	*P-value*	*P-value*	*P-value*
Birthweight, grams (95% CI)							
Model 1[b]	3,121.2	3,393.4	3,090.5	3,376.2	NA	**<0.0001**	**<0.0001**
	(3,110.1, 3,132.2)	(3,388.3, 3,398.6)	(3,079.3, 3, 101.8)	(3,371.1, 3,376.2)			
Model 2[c]	3,108.2	3,297.8	3,078.1	3,273.4	NA	**<0.0001**	**<0.0001**
	(2,926.9, 3,289.4)	(3,233.9, 3,361.8)	(2,896.7, 3,259.4)	(3,209.5, 3,337.3)			
Model 3[d]	3,085.3	3,305.7	3,053.4	3,281.3	0.3669	**<0.0001**	**<0.0001**
	(3,024.1, 3,146.6)	(3,245.4, 3,365.9)	(2,992.2, 3,114.6)	(3,221.1, 3,341.6)			
Gestational Age, weeks (95% CI)							
Model 1[b]	38.37	38.85	38.27	38.87	NA	**<0.0001**	0.9916
	(38.32, 38.41)	(38.83, 38.87)	(38.23, 38.32)	(38.86, 38.89)			
Model 2[c]	38.32	38.65	38.25	38.66	NA	**<0.0001**	0.6048
	(37.58, 39.05)	(38.43, 38.87)	(37.52, 38.25)	(38.44, 38.66)			
Model 3[d]	38.28	38.65	38.20	38.66	**0.0056**	**<0.0001**	0.4471
	(38.06, 38.50)	(38.43, 38.87)	(37.98, 38.42)	(38.44, 38.88)			
Size-for-Gestational-Age z-score, mean (95% CI)							
Model 1[b]	-0.28	0.15	-0.32	0.11	NA	**<0.0001**	**<0.0001**
	(-0.30, -0.27)	(0.14, 0.16)	(-0.34, -0.30)	(0.10, 0.12)			
Model 2[c]	-0.15	0.03	-0.19	-0.03	NA	**<0.0001**	**<0.0001**
	(-0.45, 0.16)	(-0.09, 0.15)	(-0.49, 0.11)	(-0.14, 0.09)			
Model 3[d]	-0.30	0.06	-0.34	0.01	0.5658	**<0.0001**	**<0.0001**
	(-0.41, -0.19)	(-0.05, 0.17)	(-0.45, -0.23)	(-0.10, 0.12)			

[a] Exposed infants were born between 1/5/2016 and 9/30/2016; unexposed infants were born in the same 37-week period in 2013, 2014, or 2015.

[b] Linear model (1) includes race and exposure.

[c] Linear model (2) adjusted for covariates: Model 1 + covariates (maternal age, education, marital status, source of payment for delivery, receipt of WIC during pregnancy, pre-pregnancy BMI, parity, infant sex, and residential geographic region).

[d] Linear model (3) including Model 2 + interaction between race and exposure

Bolded *P-values* denote statistical significance at $\alpha = 0.05$.

Table 2. 5 Linear Regression Analysis of Exposure to the Flint Water Crisis Emergency Declaration in Michigan: Predicted Means and 95% Confidence Intervals for Birthweight, Gestational Age, and Size-for-Gestational-Age, 2014 vs. 2016 (n=113,944).

	Unexposed: 2014[a] n=58,600		Exposed: 2016[a] n=55,344		Race x Exposure	Race	Exposure
	Non-Hispanic Black n=11,596	**Non-Hispanic White n=47,004**	**Non-Hispanic Black n=11,073**	**Non-Hispanic White n=44,271**	***P-value***	***P-value***	***P-value***
Birthweight, grams (95% CI)							
Model 1[b]	3,119.2	3,391.4	3,090.5	3,376.2	NA	**<0.0001**	**<0.0001**
	(3,108.2, 3,130.3)	(3,386.5, 3,396.4)	(3,079.2, 3,101.8)	(3,381.3, 3,376.2)			
Model 2[c]	3,153.9	3,265.7	3,133.4	3,247.9	NA	**<0.0001**	**<0.0001**
	(2,994.0, 3,313.8)	(3,194.5, 3,336.8)	(2,973.2, 3,293.6)	(3,176.7, 3,319.0)			
Model 3[d]	3,080.8	3,302.9	3,058.0	3,285.3	0.5459	**<0.0001**	**<0.0001**
	(3,015.7, 3,145.8)	(3,238.6, 3,367.3)	(2,992.8, 3,123.2)	(3,221.0, 3,349.5)			
Gestational Age, weeks (95% CI)							
Model 1[b]	38.34	38.87	38.27	38.87	NA	**<0.0001**	0.6040
	(38.29, 38.38)	(38.85, 38.88)	(38.23, 38.27)	(38.86, 38.89)			
Model 2[c]	38.56	38.63	38.54	38.63	NA	**<0.0001**	0.7372
	(37.91, 39.21)	(38.39, 38.87)	(37.89, 39.18)	(38.39, 38.88)			
Model 3[d]	38.32	38.73	38.29	38.73	0.2405	**<0.0001**	0.8369
	(38.09, 38.56)	(38.50, 38.96)	(38.06, 38.52)	(38.50, 38.96)			
Size-for-Gestational-Age z-score, mean (95% CI)							
Model 1[b]	-0.28	0.14	-0.32	0.11	NA	**<0.0001**	**<0.0001**
	(-0.30, -0.26)	(0.13, 0.15)	(-0.34, -0.30)	(0.10, 0.12)			
Model 2[c]	-0.28	-0.04	-0.32	-0.08	NA	**<0.0001**	**<0.0001**
	(-0.55, -0.02)	(-0.17, 0.09)	(-0.58, -0.05)	(-0.21, -0.08)			
Model 3[d]	-0.35	0.01	-0.39	-0.03	0.8722	**<0.0001**	**<0.0001**
	(-0.46, -0.23)	(-0.11, 0.12)	(-0.50, -0.27)	(-0.15, 0.09)			

[a] Exposed infants were born between 1/5/2016 and 9/30/2016; unexposed infants were born in the same 37-week period in 2013, 2014, or 2015.

[b] Linear model (1) includes race and exposure.

[c] Linear model (2) adjusted for covariates: Model 1 + covariates (maternal age, education, marital status, source of payment for delivery, receipt of WIC during pregnancy, pre-pregnancy BMI, parity, infant sex, and residential geographic region).

[d] Linear model (3) including Model 2 + interaction between race and exposure

Bolded *P-values* denote statistical significance at $\alpha = 0.05$.

Table 2. 6 Linear Regression Analysis of Exposure to the Flint Water Crisis Emergency Declaration in Michigan: Predicted Means and 95% Confidence Intervals (CI) for Birthweight, Gestational Age, and Size-for-Gestational-Age, 2015 vs. 2016 (n=112, 998).

	Unexposed: 2015[a] n=57,654		Exposed: 2016[a] n=55,344		Race x Exposure	Race	Exposure
	Non-Hispanic Black n=11,500	**Non-Hispanic White n=46,154**	**Non-Hispanic Black n=11,073**	**Non-Hispanic White n=44,271**	***P-value***	***P-value***	***P-value***
Birthweight, grams (95% CI)							
Model 1[b]	3,111.7	3,387.7	3,090.5	3,376.2	NA	**<0.0001**	**<0.0001**
	(3,100.6, 3,122.8)	(3,382.7, 3,392.7)	(3,101.8, 3,090.5)	(3,371.1, 3,381.3)			
Model 2[c]	3,075.1	3,270.3	3,054.9	3,256.6	NA	**<0.0001**	**<0.0001**
	(2,922.9, 3,227.3)	(3,209.4, 3,331.2)	(2,902.5, 3,207.2)	(3,195.7, 3,317.5)			
Model 3[d]	3,070.8	3,290.9	3,049.0	3,277.2	0.3329	**<0.0001**	**0.0002**
	(3,013.8, 3,127.8)	(3,234.8, 3,346.9)	(2,991.9, 3,106.1)	(3,221.1, 3,333.2)			
Gestational Age, weeks (95% CI)							
Model 1[b]	38.31	38.89	38.27	38.87	NA	**<0.0001**	0.1034
	(38.27, 38.31)	(38.87, 38.91)	(38.23, 38.32)	(38.86, 38.89)			
Model 2[c]	38.48	38.75	38.46	38.73	NA	**<0.0001**	0.1795
	(37.86, 39.09)	(38.54, 38.96)	(37.84, 39.46)	(38.52, 38.73)			
Model 3[d]	38.37	38.80	38.34	38.78	0.8343	**<0.0001**	0.2652
	(38.16, 38.57)	(38.60, 39.00)	(38.14, 38.55)	(38.58, 38.99)			
Size-for-Gestational-Age z-score, mean (95% CI)							
Model 1[b]	-0.29	0.13	-0.32	0.11	NA	**<0.0001**	**0.0003**
	(-0.31, -0.29)	(0.12, 0.14)	(-0.34, -0.30)	(0.10, 0.12)			
Model 2[c]	-0.41	-0.07	-0.44	-0.09	NA	**<0.0001**	**<0.0001**
	(-0.66, -0.16)	(-0.18, -0.04)	(-0.69, -0.19)	(-0.21, 0.02)			
Model 3[d]	-0.39	-0.04	-0.43	-0.07	0.3933	**<0.0001**	**0.0004**
	(-0.49, -0.29)	(-0.15, 0.06)	(-0.53, -0.32)	(-0.17, 0.03)			

[a] Exposed infants were born between 1/5/2016 and 9/30/2016; unexposed infants were born in the same 37-week period in 2013, 2014, or 2015.

[b] Linear model (1) includes race and exposure.

[c] Linear model (2) adjusted for covariates: Model 1 + covariates (maternal age, education, marital status, source of payment for delivery, receipt of WIC during pregnancy, pre-pregnancy BMI, parity, infant sex, and residential geographic region).

[d] Linear model (3) including Model 2 + interaction between race and exposure

Bolded *P-values* denote statistical significance at $\alpha = 0.05$.

Table 2. 7 Linear Regression Coefficients, Predicted Means, and 95% Confidence Intervals for Regression of Birthweight on Exposure to the Flint Water Crisis Emergency Declaration in Michigan, for, 2013, 2014, and 2015 vs. 2016 (n=226,672).

	Model 1[b]			Model 2[c]			Model 3[d]		
Variable	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	3374.340	2.453	**<0.0001**	3227.840	9.603	**<0.0001**	3228.740	9.648	**<0.0001**
Maternal Race									
Non-Hispanic Black	-276.470	2.935	**<0.0001**	-221.290	3.456	**<0.0001**	-226.380	6.222	**<0.0001**
Non-Hispanic White	Ref			Ref					
Exposed[a] year									
2013	19.958	3.372	**<0.0001**	25.668	3.350	**<0.0001**	24.194	3.737	**<0.0001**
2014	17.929	3.321	**<0.0001**	18.397	3.292	**<0.0001**	17.524	3.655	**<0.0001**
2015	13.441	3.334	**<0.0001**	15.460	3.293	**<0.0001**	13.879	3.666	**0.0002**
2016	Ref			Ref			Ref		
Maternal Age				-3.886	0.280	**<0.0001**	-3.884	0.280	**<0.0001**
Maternal Education									
< High School				-150.210	4.921	**<0.0001**	-150.260	4.921	**<0.0001**
High School/GED				-91.641	3.628	**<0.0001**	-91.625	3.628	**<0.0001**
Some College				-43.576	3.313	**<0.0001**	-43.579	3.313	**<0.0001**
College or More				Ref					
Unknown				-117.120	18.978	**<0.0001**	-117.050	18.978	**<0.0001**
Maternal Marital Status									
Never Married				-64.530	3.174	**<0.0001**	-64.555	3.174	**<0.0001**
Married				Ref					
Divorced/Widowed				-93.186	6.937	**<0.0001**	-93.186	6.937	**<0.0001**
Unknown				-190.680	83.097	**0.0218**	-190.960	83.098	**0.0216**
Source of Payment for Delivery									
Private Insurance				Ref					
Medicaid				-58.232	3.155	**<0.0001**	-58.143	3.160	**<0.0001**
Self-Pay				49.904	10.129	**<0.0001**	49.933	10.129	**<0.0001**
Other				-31.122	12.601	**0.0135**	-31.032	12.676	**0.0144**
Unknown				-2.111	24.610	0.9316	-2.022	24.611	0.9345
Receipt of WIC During Pregnancy									
Yes				-1.426	3.156	0.6515	-1.468	3.156	0.6419
No				Ref					

Unknown	3.549	12.559	0.7775	3.539	12.559	0.7781
Pre-pregnancy BMI	8.927	0.173	**<0.0001**	8.928	0.173	**<0.0001**
Parity (including birth on record)	13.524	0.797	**<0.0001**	13.524	0.797	**<0.0001**
Infant's Sex						
Female	Ref					
Male	121.380	2.330	**<0.0001**	121.370	2.330	**<0.0001**
Residential Geographic Region						
Upper Peninsula Prosperity Alliance	39.971	7.591	**<0.0001**	39.987	7.591	**<0.0001**
Northwest Prosperity Region	49.226	7.180	**<0.0001**	49.225	7.180	**<0.0001**
Northeast Prosperity Region	21.403	9.149	**0.0193**	21.422	9.149	**0.0192**
West Michigan Prosperity Alliance	25.624	3.480	**<0.0001**	25.634	3.480	**<0.0001**
East Central Michigan Prosperity Region	12.347	5.371	**0.0215**	12.363	5.371	**0.0213**
East Michigan Prosperity Region	1.958	4.690	0.6763	1.974	4.690	0.6738
South Central Prosperity Region	9.224	5.733	**0.1077**	9.222	5.733	0.1077
Southwest Prosperity Region	15.175	4.468	**0.0007**	15.179	4.468	**0.0007**
Southeast Michigan Prosperity Region	14.428	4.279	**0.0007**	14.447	4.279	**0.0007**
Detroit Metro Prosperity Region	Ref					
Interactions						
Race*Exposure (2013)				7.540	8.405	0.3697
Race*Exposure (2014)				4.521	8.379	0.5895
Race*Exposure (2015)				8.193	8.337	0.3258
Race*Exposure (2016)				Ref		

Predicted Means	**Model 1[b]**		**Model 2[c]**		**Model 3[d]**	
	Non-Hispanic Black	**Non-Hispanic White**	**Non-Hispanic Black**	**Non-Hispanic White**	**Non-Hispanic Black**	**Non-Hispanic White**
Exposure[a] Year	**Mean (95% CI)**	**Mean (95% CI)**	**Mean (95% CI)**	**Mean (95% CI)**	**Mean (95% CI)**	**Mean (95% CI)**
2013	3121.2 (3110.1, 3132.2)	3393.4 (3388.3, 3398.6)	3134.5 (3029.6, 3239.3)	3302.3 (3254.1, 3350.5)	3105.9 (3061.7, 3150.1)	3324.8 (3281.6, 3367.9)
2014	3119.2 (3108.2, 3130.2)	3391.4 (3386.5, 3396.4)	3124.7 (3020.1, 3229.4)	3295.4 (3247.2, 3343.6)	3096.2 (3052.2, 3140.3)	3318.1 (3275.0, 3361.2)
2015	3111.7 (3100.6, 3122.7)	3387.7 (3382.7, 3392.7)	3125.0 (3020.1, 3229.8)	3291.8 (3243.6, 3340.0)	3096.3 (3052.1, 3140.4)	3314.4 (3271.4, 3357.5)
2016	3090.5 (3079.3, 3101.8)	3376.2 (3371.1, 3381.3)	3104.6 (2999.7, 3209.5)	3277.7 (3229.5, 3325.9)	3074.2 (3029.9, 3118.4)	3300.6 (3257.5, 3343.6)

[a] Exposed infants were born between 1/5/2016 and 9/30/2016; unexposed infants were born in the same 37-week period in 2013, 2014, or 2015.
[b] Linear model (1) includes race and exposure.

[c] Linear model (2) adjusted for covariates: Model 1 + covariates (maternal age, education, marital status, source of payment for delivery, receipt of WIC during pregnancy, pre-pregnancy BMI, parity, infant sex, and residential geographic region).
[d] Linear model (3) including Model 1 + interaction between race and exposure
[e] Linear model (4) including Model 2 + interaction between race and exposure
Bolded *P-values* denote statistical significance at $\alpha = 0.05$.
Sample Sizes Non-Hispanic Black: 11,444 (2013), 11,596 (2014), 11,500 (2015), 11,073 (2016); Non-Hispanic White: 43,630 (2013), 47,004 (2014), 46,154 (2015), 44,271.

Table 2. 8 Linear Regression Coefficients, Predicted Means, and 95% Confidence Intervals for Regression of Gestational Age on Exposure to the Flint Water Crisis Emergency Declaration in Michigan, Predicted Means and 95% Confidence Intervals for, 2013, 2014, and 2015 vs. 2016 (n=226,672).

	Model 1[b]			Model 2[c]			Model 3[d]		
Variable	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	38.864	0.009	**<0.0001**	39.841	0.034	**<0.0001**	39.846	0.034	**<0.0001**
Maternal Race									
Non-Hispanic Black	-0.546	0.010	**<0.0001**	-0.419	0.012	**<0.0001**	-0.451	0.022	**<0.0001**
Non-Hispanic White	Ref			Ref			Ref		
Exposed[a] year									
2013	0.000	0.012	0.9937	0.007	0.012	0.5374	-0.010	0.013	0.4542
2014	0.006	0.012	0.6003	0.004	0.012	0.7573	-0.003	0.013	0.8235
2015	0.019	0.012	0.1005	0.016	0.012	0.1760	0.014	0.013	0.2680
2016	Ref			Ref			Ref		
Maternal Age				-0.022	0.001	**<0.0001**	-0.022	0.001	**<0.0001**
Maternal Education									
< High School				-0.279	0.018	**<0.0001**	-0.280	0.018	**<0.0001**
High School/GED				-0.197	0.013	**<0.0001**	-0.197	0.013	**<0.0001**
Some College				-0.105	0.012	**<0.0001**	-0.105	0.012	**<0.0001**
College or More				Ref			Ref		
Unknown				-0.177	0.068	**0.0092**	-0.176	0.068	**0.0094**
Maternal Marital Status									
Never Married				-0.135	0.011	**<0.0001**	-0.136	0.011	**<0.0001**
Married				Ref			Ref		
Divorced/Widowed				-0.243	0.025	**<0.0001**	-0.244	0.025	**<0.0001**
Unknown				-0.526	0.297	0.0763	-0.527	0.297	0.0757
Source of Payment for Delivery									
Private Insurance				Ref			Ref		
Medicaid				-0.101	0.011	**<0.0001**	-0.098	0.011	**<0.0001**
Self-Pay				0.261	0.036	**<0.0001**	0.262	0.036	**<0.0001**
Other				-0.006	0.045	0.8916	-0.005	0.045	0.9149
Unknown				0.100	0.088	0.2545	0.101	0.088	0.2504
Receipt of WIC During Pregnancy									
Yes				0.107	0.011	**<0.0001**	0.106	0.011	**<0.0001**
No				Ref			Ref		

Unknown	0.051	0.045	0.2579	0.051	0.045	0.2599
Pre-pregnancy BMI	-0.002	0.001	**0.0010**	-0.002	0.001	**0.0009**
Parity (including birth on record)	-0.048	0.003	**<0.0001**	-0.048	0.003	**<0.0001**
Infant's Sex						
Female	Ref			Ref		
Male	-0.092	0.008	**<0.0001**	-0.092	0.008	**<0.0001**
Residential Geographic Region						
Upper Peninsula Prosperity Alliance	0.123	0.027	**<0.0001**	0.123	0.027	**<0.0001**
Northwest Prosperity Region	0.193	0.026	**<0.0001**	0.193	0.026	**<0.0001**
Northeast Prosperity Region	0.109	0.033	**0.0009**	0.108	0.033	**0.0009**
West Michigan Prosperity Alliance	0.006	0.012	0.6227	0.006	0.012	0.6187
East Central Michigan Prosperity Region	0.090	0.019	**<0.0001**	0.090	0.019	**<0.0001**
East Michigan Prosperity Region	-0.087	0.017	**<0.0001**	-0.087	0.017	**<0.0001**
South Central Prosperity Region	0.052	0.020	**0.0111**	0.052	0.020	**0.0112**
Southwest Prosperity Region	0.145	0.016	**<0.0001**	0.145	0.016	**<0.0001**
Southeast Michigan Prosperity Region	0.072	0.015	**<0.0001**	0.072	0.015	**<0.0001**
Detroit Metro Prosperity Region	Ref			Ref		
Interactions						
Race*Exposure (2013)				0.088	0.030	**0.0035**
Race*Exposure (2014)				0.034	0.030	0.2543
Race*Exposure (2015)				0.008	0.030	0.8001
Race*Exposure (2016)				Ref		

Predicted Means	**Model 1[b]**		**Model 2[c]**		**Model 3[d]**	
	Non-Hispanic Black	**Non-Hispanic White**	**Non-Hispanic Black**	**Non-Hispanic White**	**Non-Hispanic Black**	**Non-Hispanic White**
Exposure[a] Year	**Mean (95% CI)**	**Mean (95% CI)**	**Mean (95% CI)**	**Mean (95% CI)**	**Mean (95% CI)**	**Mean (95% CI)**
2013	38.37 (38.32, 38.41)	38.85 (38.83, 38.87)	38.32 (37.90, 38.75)	38.77 (38.61, 38.94)	38.42 (38.26, 38.57)	38.78 (38.63, 38.93)
2014	38.34 (38.29, 38.38)	38.87 (38.85, 38.88)	38.28 (37.86, 38.70)	38.78 (38.62, 38.95)	38.37 (38.21, 38.53)	38.79 (38.63, 38.94)
2015	38.31 (38.27, 38.36)	38.89 (38.87, 38.91)	38.27 (37.85, 38.70)	38.80 (38.63, 38.96)	38.36 (38.20, 38.52)	38.80 (38.65, 38.96)
2016	38.27 (38.22, 38.32)	38.87 (38.86, 38.89)	38.26 (37.83, 38.68)	38.78 (38.62, 38.95)	38.34 (38.18, 38.50)	38.79 (38.64, 38.94)

[a] Exposed infants were born between 1/5/2016 and 9/30/2016; unexposed infants were born in the same 37-week period in 2013, 2014, or 2015.
[b] Linear model (1) includes race and exposure.

[c] Linear model (2) adjusted for covariates: Model 1 + covariates (maternal age, education, marital status, source of payment for delivery, receipt of WIC during pregnancy, pre-pregnancy BMI, parity, infant sex, and residential geographic region).
[d] Linear model (3) including Model 1 + interaction between race and exposure
[e] Linear model (4) including Model 2 + interaction between race and exposure
Bolded *P-values* denote statistical significance at $\alpha = 0.05$.
Sample Sizes Non-Hispanic Black: 11,444 (2013), 11,596 (2014), 11,500 (2015), 11,073 (2016); Non-Hispanic White: 43,630 (2013), 47,004 (2014), 46,154 (2015), 44,271.

Table 2. 9 Linear Regression Coefficients, Predicted Means, and 95% Confidence Intervals for Size-For-Gestational-Age (Z-Score) on Exposure to the Flint Water Crisis Emergency Declaration in Michigan, Predicted Means and 95% Confidence Intervals for, 2013, 2014, and 2015 vs. 2016 (n=226,672).

	Model 1[b]			Model 2[c]			Model 3[d]		
Variable	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	0.112	0.004	**<0.0001**	-0.413	0.018	**<0.0001**	-0.412	0.018	**<0.0001**
Maternal Race									
Non-Hispanic Black	-0.429	0.005	**<0.0001**	-0.351	0.006	**<0.0001**	-0.352	0.011	**<0.0001**
Non-Hispanic White	Ref			Ref			Ref		
Exposed[a] year									
2013	0.040	0.006	**<0.0001**	0.051	0.006	**<0.0001**	0.053	0.007	**<0.0001**
2014	0.033	0.006	**<0.0001**	0.037	0.006	**<0.0001**	0.037	0.007	**<0.0001**
2015	0.022	0.006	**0.0003**	0.027	0.006	**<0.0001**	0.024	0.007	**0.0003**
2016	Ref			Ref			Ref		
Maternal Age				-0.001	0.001	**0.0111**	-0.001	0.001	**0.0108**
Maternal Education									
< High School				-0.237	0.009	**<0.0001**	-0.236	0.009	**<0.0001**
High School/GED				-0.138	0.007	**<0.0001**	-0.138	0.007	**<0.0001**
Some College				-0.063	0.006	**<0.0001**	-0.063	0.006	**<0.0001**
College or More				Ref			Ref		
Unknown				-0.188	0.035	**<0.0001**	-0.188	0.035	**<0.0001**
Maternal Marital Status									
Never Married				-0.086	0.006	**<0.0001**	-0.086	0.006	**<0.0001**
Married				Ref			Ref		
Divorced/Widowed				-0.122	0.013	**<0.0001**	-0.122	0.013	**<0.0001**
Unknown				-0.244	0.152	0.1077	-0.244	0.152	0.1073
Source of Payment for Delivery									
Private Insurance				Ref			Ref		
Medicaid				-0.094	0.006	**<0.0001**	-0.095	0.006	**<0.0001**
Self-Pay				0.071	0.018	**0.0001**	0.071	0.018	**0.0001**
Other				-0.064	0.023	**0.0054**	-0.065	0.023	**0.0051**
Unknown				0.003	0.045	0.9467	0.003	0.045	0.9486
Receipt of WIC During Pregnancy									
Yes				-0.059	0.006	**<0.0001**	-0.058	0.006	**<0.0001**
No				Ref			Ref		

Unknown	-0.025	0.023	0.2684	-0.025	0.023	0.2694
Pre-pregnancy BMI	0.021	0.000	**<0.0001**	0.021	0.000	**<0.0001**
Parity (including birth on record)	0.043	0.001	**<0.0001**	0.043	0.001	**<0.0001**
Infant's Sex						
Female	Ref			Ref		
Male	0.016	0.004	**0.0001**	0.016	0.004	**0.0001**
Residential Geographic Region						
Upper Peninsula Prosperity Alliance	0.065	0.014	**<0.0001**	0.065	0.014	**<0.0001**
Northwest Prosperity Region	0.071	0.013	**<0.0001**	0.071	0.013	**<0.0001**
Northeast Prosperity Region	0.021	0.017	0.2138	0.021	0.017	0.2120
West Michigan Prosperity Alliance	0.063	0.006	**<0.0001**	0.063	0.006	**<0.0001**
East Central Michigan Prosperity Region	0.003	0.010	0.7508	0.003	0.010	0.7479
East Michigan Prosperity Region	0.032	0.009	**0.0002**	0.032	0.009	**0.0002**
South Central Prosperity Region	0.016	0.010	0.1233	0.016	0.010	0.1234
Southwest Prosperity Region	-0.001	0.008	0.9445	-0.001	0.008	0.9506
Southeast Michigan Prosperity Region	0.011	0.008	0.1652	0.011	0.008	0.1658
Detroit Metro Prosperity Region	Ref		**<0.0001**	Ref		
Interactions						
Race*Exposure (2013)				-0.010	0.015	0.5026
Race*Exposure (2014)				0.002	0.015	0.8930
Race*Exposure (2015)				0.013	0.015	0.4062
Race*Exposure (2016)				Ref		

Predicted Means	**Model 1[b]**		**Model 2[c]**		**Model 3[d]**	
	Non-Hispanic Black	**Non-Hispanic White**	**Non-Hispanic Black**	**Non-Hispanic White**	**Non-Hispanic Black**	**Non-Hispanic White**
Exposure[a] Year	**Mean (95% CI)**	**Mean (95% CI)**	**Mean (95% CI)**	**Mean (95% CI)**	**Mean (95% CI)**	**Mean (95% CI)**
2013	-0.28	0.15	-0.18	-0.01	-0.31	0.05
	(-0.30, -0.27)	(0.14, 0.16)	(-0.36, -0.01)	(-0.10, 0.08)	(-0.39, -0.23)	(-0.03, 0.13)
2014	-0.28	0.14	-0.19	-0.03	-0.32	0.03
	(-0.30, -0.26)	(0.13, 0.15)	(-0.37, -0.02)	(-0.12, 0.06)	(-0.40, -0.24)	(-0.05, 0.11)
2015	-0.29	0.13	-0.19	-0.04	-0.32	0.02
	(-0.31, -0.27)	(0.12, 0.14)	(-0.37, -0.02)	(-0.13, 0.05)	(-0.40, -0.24)	(-0.06, 0.10)
2016	-0.32	0.11	-0.23	-0.06	-0.36	-0.01
	(-0.34, -0.30)	(0.10, 0.12)	(-0.40, -0.05)	(-0.15, 0.03)	(-0.44, -0.28)	(-0.08, 0.07)

[a] Exposed infants were born between 1/5/2016 and 9/30/2016; unexposed infants were born in the same 37-week period in 2013, 2014, or 2015.

[b] Linear model (1) includes race and exposure.

[c] Linear model (2) adjusted for covariates: Model 1 + covariates (maternal age, education, marital status, source of payment for delivery, receipt of WIC during pregnancy, pre-pregnancy BMI, parity, infant sex, and residential geographic region).
[d] Linear model (3) including Model 1 + interaction between race and exposure
[e] Linear model (4) including Model 2 + interaction between race and exposure
Bolded *P-values* denote statistical significance at $\alpha = 0.05$.
Sample Sizes Non-Hispanic Black: 11,444 (2013), 11,596 (2014), 11,500 (2015), 11,073 (2016); Non-Hispanic White: 43,630 (2013), 47,004 (2014), 46,154 (2015), 44,271.

Table 2. 10 Logistic Regression Analysis of Exposure to the Flint Water Crisis Emergency Declaration in Michigan: Percentages and Adjusted Odds Ratios (aOR) with 95% Confidence Intervals (CI) for Low Birthweight, Preterm Birth, and Small-for-Gestational-Age (n=226,672).

	Unexposed[a] n=171,328		Exposed[a] n=55,344		Race x Exposure	Race	Exposure
	Non-Hispanic Black n=34,540	Non-Hispanic White n=136,788	Non-Hispanic Black n=11,073	Non-Hispanic White n=44,271	*P-value*	*P-value*	*P-value*
Low Birthweight, % (95% CI)							
Model 1[b]	11.2 (10.9, 11.6)	5.0 (4.9, 5.1)	12.4 (11.6, 12.9)	5.3 (5.1, 5.5)	NA	**<0.0001**	**0.0007**
Model 2[c]	10.0 (5.9, 16.7)	7.5 (5.8, 9.6)	10.7 (6.3, 17.8)	7.9 (6.1, 10.2)	NA	**<0.0001**	**0.0017**
Model 3[d]	12.8 (10.3, 15.8)	6.8 (5.4, 8.5)	13.7 (11.0, 16.9)	7.2 (5.7, 9.0)	0.7351	**<0.0001**	**0.0181**
NHB vs. NHW, aOR[e] (95% CI)	2.01 (1.91, 2.11)		2.04 (1.89, 2.20)				
Preterm Birth, % (95% CI)							
Model 1[b]	12.2 (11.9, 12.5)	7.1 (7.0, 7.2)	12.8 (12.2, 13.5)	7.3 (7.0, 7.5)	NA	**<0.0001**	**0.0295**
Model 2[c]	13.8 (9.2, 20.3)	6.5 (4.6, 9.3)	14.2 (9.4, 20.9)	6.7 (4.7, 9.5)	NA	**<0.0001**	0.1170
Model 3[d]	10.9 (8.5, 13.9)	7.2 (5.5, 9.2)	11.2 (8.7, 14.3)	7.4 (5.7, 9.5)	0.9735	**<0.0001**	0.1890
NHB vs. NHW, aOR[e] (95% CI)	1.59 (1.52, 1.67)		1.59 (1.48, 1.71)				
Small-for-Gestational-Age, % (95% CI)							
Model 1[b]	16.0 (15.6, 16.4)	8.1 (8.0, 8.3)	17.5 (16.8, 18.3)	8.4 (8.1, 8.6)	NA	**<0.0001**	**0.0006**
Model 2[c]	12.7 (7.6, 20.6)	10.9 (8.6, 13.7)	13.9 (8.3, 22.4)	11.4 (9.0, 14.3)	NA	**<0.0001**	**0.0002**
Model 3[d]	16.9 (13.9, 20.5)	10.0 (8.1, 12.3)	18.4 (15.1, 22.3)	10.4 (8.4, 12.9)	0.1106	**<0.0001**	**0.0257**
NHB vs. NHW, aOR[e] (95% CI)	1.83 (1.75, 1.91)		1.94 (1.82, 2.07)				

[a] Exposed infants were born between 1/5/2016 and 9/30/2016; unexposed infants were born in the same 37-week period in 2013, 2014, or 2015.

[b] Logistic model (1) includes race and exposure

[c] Logistic regression model (2) includes Model 1 + covariates (maternal age, education, marital status, source of payment for delivery, receipt of WIC during pregnancy, pre-pregnancy BMI, parity, infant sex, and residential geographic region)

[d] Logistic regression model (3) includes Model 2 + Interaction between race and exposure

[e] aOR is based on Model 4

Bolded *P-values* denote statistical significance at $\alpha = 0.05$.

Table 2. 11 Linear Regression Analysis of Exposure to the Flint Water Crisis Emergency Declaration during the First Trimester of Pregnancy in Michigan: Predicted Means and 95% Confidence Intervals (CI) for Birthweight, Gestational Age, and Size-for-Gestational-Age (n=86,618).

	Unexposed[a] n=65,662		Exposed[a] n=20,956		Race x Exposure	Race	Exposure
	Non-Hispanic Black n=13,035	Non-Hispanic White n=52,627	Non-Hispanic Black n=4,265	Non-Hispanic White n=16,691	*P-value*	*P-value*	*P-value*
Birthweight, grams (95% CI)							
Model 1[b]	3,106.7 (3,096.2, 3,117.1)	3,384.2 (3,379.5, 3,388.8)	3,077.8 (3,059.5, 3,077.8)	3,367.5 (3,359.2, 3,375.8)	NA	**<0.0001**	**<0.0001**
Model 2[c]	3,049.2 (2,851.1, 3,247.3)	3,291.0 (3,198.4, 3,342.2)	3,026.4 (2,827.8, 3,224.9)	3,291.0 (3,219.4, 3,362.6)	NA	**<0.0001**	**<0.0001**
Model 3[d]	3,082.4 (3,015.4, 3,149.5)	3,302.6 (3,236.4, 3,368.9)	3,056.7 (2,988.1, 3,125.3)	3,282.2 (3,215.6, 3,348.9)	0.6326	**<0.0001**	**<0.0001**
Gestational Age, weeks (95% CI)							
Model 1[b]	38.25 (38.21, 38.25)	38.82 (38.83, 38.82)	38.20 (38.12, 38.27)	38.82 (38.79, 38.82)	NA	**<0.0001**	0.4617
Model 2[c]	37.83 (37.03, 38.63)	38.85 (38.60, 39.09)	37.82 (37.02, 38.63)	38.84 (38.59, 39.08)	NA	**<0.0001**	0.5029
Model 3[d]	38.34 (38.10, 38.57)	38.79 (38.55, 39.02)	38.32 (38.07, 38.56)	38.78 (38.54, 39.01)	0.8255	**<0.0001**	0.6138
Size-for-Gestational-Age z-score, mean (95% CI)							
Model 1[b]	-0.28 (-0.30, -0.26)	0.14 (0.13, 0.15)	-0.33 (-0.36, -0.30)	0.11 (0.09, 0.12)	NA	**<0.0001**	**<0.0001**
Model 2[c]	-0.14 (-0.47, 0.18)	-0.07 (-0.21, 0.06)	-0.20 (-0.53, 0.13)	-0.11 (-0.25, 0.02)	NA	**<0.0001**	**<0.0001**
Model 3[d]	-0.34 (-0.47, -0.22)	-0.01 (-0.13, 0.11)	-0.40 (-0.53, -0.28)	-0.05 (-0.17, 0.07)	0.3805	**<0.0001**	**<0.0001**

[a] Exposed infants were born between 1/5/2016 and 9/30/2016; unexposed infants were born in the same 37-week period in 2013, 2014, or 2015.

[b] Linear model (1) includes race and exposure.

[c] Linear model (2) adjusted for covariates: Model 1 + covariates (maternal age, education, marital status, source of payment for delivery, receipt of WIC during pregnancy, pre-pregnancy BMI, parity, infant sex, and residential geographic region).

[d] Linear model (3) including Model 2 + interaction between race and exposure

Bolded *P-values* denote statistical significance at $\alpha = 0.05$.

Table 2. 12 Linear Regression Analysis of Exposure to the Flint Water Crisis Emergency Declaration during the Second Trimester of Pregnancy in Michigan: Predicted Means and 95% Confidence Intervals (CI) for Birthweight, Gestational Age, and Size-for-Gestational-Age (n=84,505).

	Unexposed[a] n=63,880		Exposed[a] n=20,625		Race x Exposure	Race[b]	Exposure[b]
	Non-Hispanic Black n=12,495	Non-Hispanic White n=51,385	Non-Hispanic Black n=3,950	Non-Hispanic White n=16,675	*P-value*	*P-value*	*P-value*
Birthweight, grams (95% CI)							
Model 1[b]	3,135.5 (3,125.3, 3,145.7)	3,399.4 (3,394.7, 3,388.9)	3,098.0 (3,079.9, 3,116.2)	3,388.9 (3,380.7, 3,397.9)	NA	**<0.0001**	**0.0004**
Model 2[c]	3,367.7 (3,106.6, 3,428.8)	3,330.3 (3,254.6, 3,406.0)	3,233.2 (3,071.1, 3,395.2)	3,315.7 (3,239.9, 3,391.6)	NA	**<0.0001**	**<0.0001**
Model 3[d]	3,163.5 (3,095.5, 3,231.5)	3,375.9 (3,308.7, 3,443.1)	3,127.5 (3,058.0, 3,197.1)	3,361.6 (3,294.1, 3,429.0)	0.0533	**<0.0001**	**0.0029**
Gestational Age, weeks (95% CI)							
Model 1[b]	38.40 (38.36, 38.44)	38.89 (38.87, 38.90)	38.36 (38.29, 38.43)	38.91 (38.88, 38.91)	NA	**<0.0001**	0.5165
Model 2[c]	39.08 (38.46, 39.70)	38.78 (38.53, 39.03)	39.04 (38.42, 39.67)	38.79 (38.54, 39.05)	NA	**<0.0001**	0.6653
Model 3[d]	38.61 (38.37, 38.84)	38.96 (38.73, 39.19)	38.57 (38.33, 38.81)	38.98 (38.75, 39.21)	0.1227	**<0.0001**	0.2919
Size-for-Gestational-Age z-score, mean (95% CI)							
Model 1[b]	-0.27 (-0.29, -0.25)	0.15 (0.15, 0.16)	-0.32 (-0.36, -0.29)	0.13 (0.11, 0.14)	NA	**<0.0001**	**<0.0001**
Model 2[c]	-0.17 (-0.45, 0.11)	0.08 (-0.06, 0.23)	-0.23 (-0.51, 0.05)	0.05 (-0.10, 0.19)	NA	**<0.0001**	**<0.0001**
Model 3[d]	-0.24 (-0.37, -0.11)	0.12 (-0.01, 0.24)	-0.29 (-0.42, -0.16)	0.08 (-0.04, 0.21)	0.3675	**<0.0001**	**0.0001**

[a] Exposed infants were born between 1/5/2016 and 9/30/2016; unexposed infants were born in the same 37-week period in 2013, 2014, or 2015.

[b] Linear model (1) includes race and exposure.

[c] Linear model (2) adjusted for covariates: Model 1 + covariates (maternal age, education, marital status, source of payment for delivery, receipt of WIC during pregnancy, pre-pregnancy BMI, parity, infant sex, and residential geographic region).

[d] Linear model (3) including Model 2 + interaction between race and exposure

Bolded *P-values* denote statistical significance at $\alpha = 0.05$.

Table 2. 13 Linear Regression Analysis of Exposure to the Flint Water Crisis Emergency Declaration during the Third Trimester of Pregnancy in Michigan: Predicted Means and 95% Confidence Intervals (CI) for Birthweight, Gestational Age, and Size-for-Gestational-Age (n=51,407).

	Unexposed[a] n=38,704		Exposed[a] n=20,625		Race x Exposure	Race	Exposure
	Non-Hispanic Black n=8,122	**Non-Hispanic White n=30,582**	**Non-Hispanic Black n=2,571**	**Non-Hispanic White n=10,132**	***P-value***	***P-value***	***P-value***
Birthweight, grams (95% CI)							
Model 1[b]	3,208.3 (3,197.7, 3,218.9)	3,440.9 (3,435.4, 3,440.9)	3,201.4 (3,182.6, 3,220.3)	3,426.0 (3,416.4, 3,435.5)	NA	**<0.0001**	**0.0081**
Model 2[c]	3,120.6 (2,956.0, 3,285.1)	3,235.7 (3,114.2, 3,357.2)	3,107.6 (2,942.0, 3,273.3)	3,217.7 (3,096.1, 3,339.3)	NA	**<0.0001**	**0.0006**
Model 3[d]	3,096.3 (3,002.8, 3,189.9)	3,283.0 (3,190.1, 3,376.0)	3,082.8 (2,987.8, 3,177.8)	3,265.1 (3,171.9, 3,358.3)	0.7166	**<0.0001**	**0.0011**
Gestational Age, weeks (95% CI)							
Model 1[b]	38.94 (38.90, 38.97)	39.21 (39.19, 39.22)	38.83 (38.77, 38.89)	39.21 (39.18, 39.24)	NA	**<0.0001**	0.2300
Model 2[c]	38.29 (37.77, 38.81)	38.94 (38.58, 39.31)	38.29 (37.77, 38.81)	38.95 (38.58, 39.31)	NA	**<0.0001**	0.1863
Model 3[d]	38.67 (38.39, 38.95)	38.87 (38.59, 39.15)	38.56 (38.28, 38.85)	38.87 (38.59, 39.15)	**0.0039**	**<0.0001**	0.9239
Size-for-Gestational-Age z-score, mean (95% CI)							
Model 1[b]	-0.32 (-0.34, -0.30)	0.14 (0.12, 0.15)	-0.30 (-0.33, -0.26)	0.11 (0.09, 0.13)	NA	**<0.0001**	0.0727
Model 2[c]	-0.28 (-0.62, 0.05)	-0.26 (-0.51, -0.00)	-0.27 (-0.61, 0.06)	-0.29 (-0.55, -0.04)	NA	**<0.0001**	**0.0064**
Model 3[d]	-0.47 (-0.67, -0.28)	-0.10 (-0.29, 0.09)	-0.46 (-0.66, -0.27)	-0.14 (-0.33, 0.06)	0.0726	**<0.0001**	**0.0012**

[a] Exposed infants were born between 1/5/2016 and 9/30/2016; unexposed infants were born in the same 37-week period in 2013, 2014, or 2015.

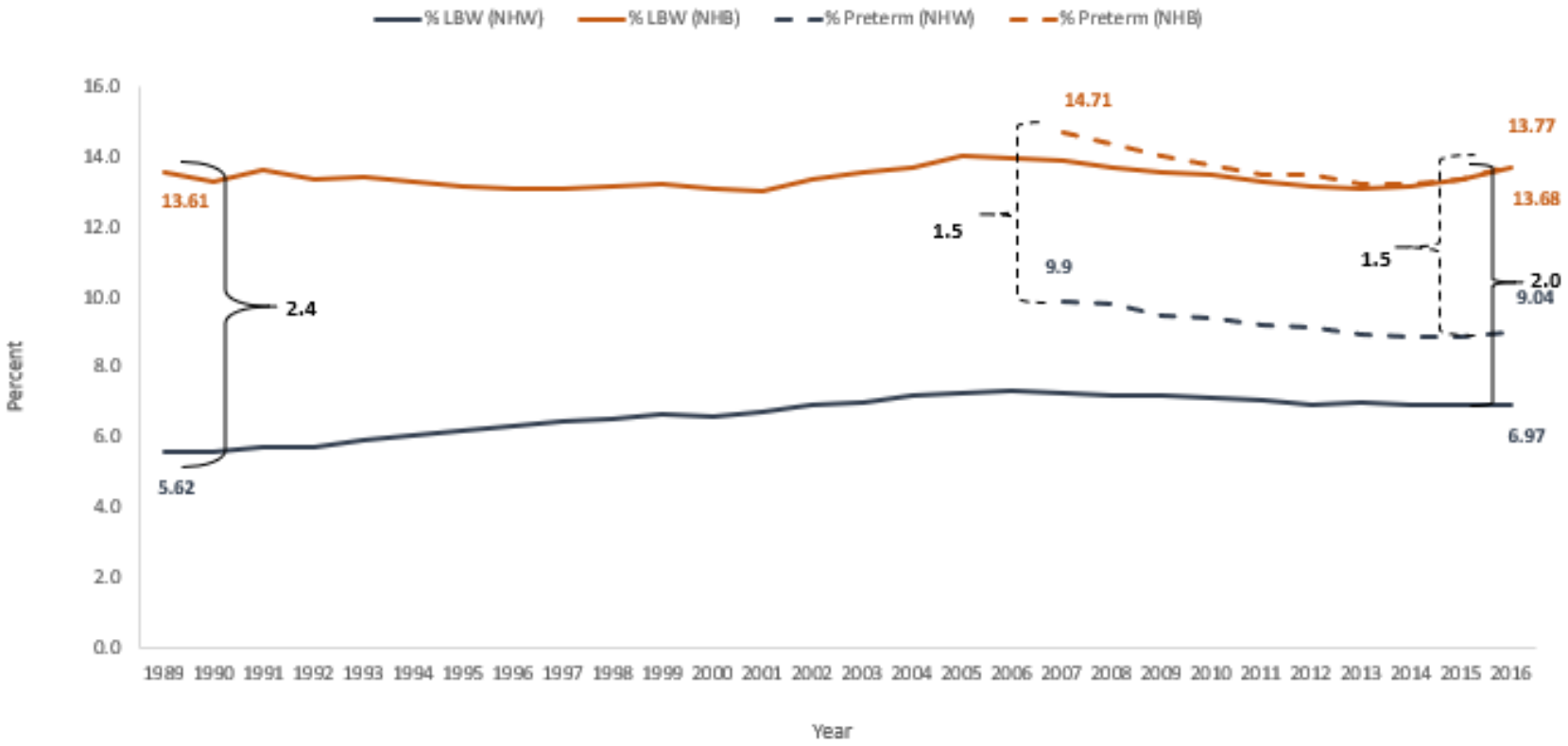
[b] Linear model (1) includes race and exposure.

[c] Linear model (2) adjusted for covariates: Model 1 + covariates (maternal age, education, marital status, source of payment for delivery, receipt of WIC during pregnancy, pre-pregnancy BMI, parity, infant sex, and residential geographic region).

[d] Linear model (3) including Model 2 + interaction between race and exposure

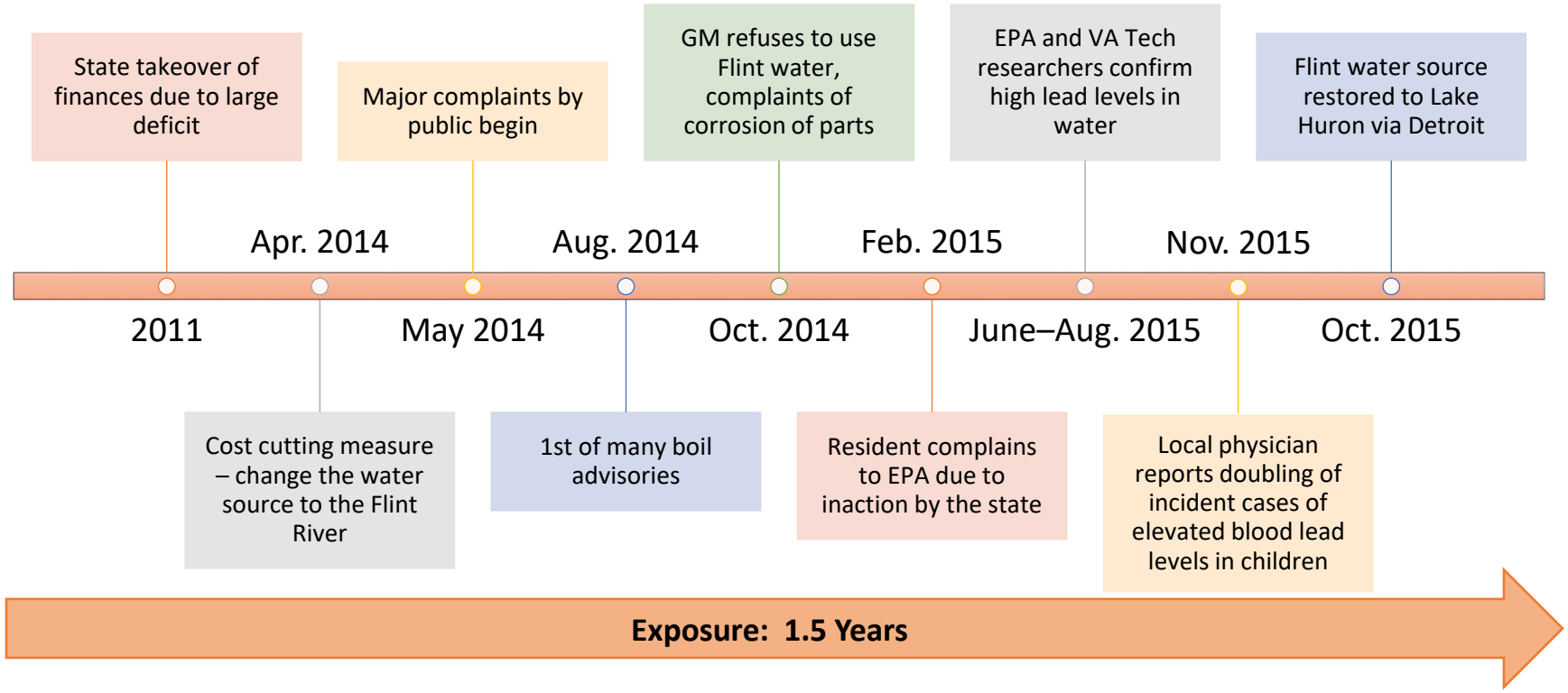
Bolded *P-values* denote statistical significance at $\alpha = 0.05$.

Figure 2. 1 Low Birthweight and Preterm Births for Non-Hispanic Blacks (NHB) and Non-Hispanic Whites (NHW) in the United States, 1989 – 2016.[277,278]



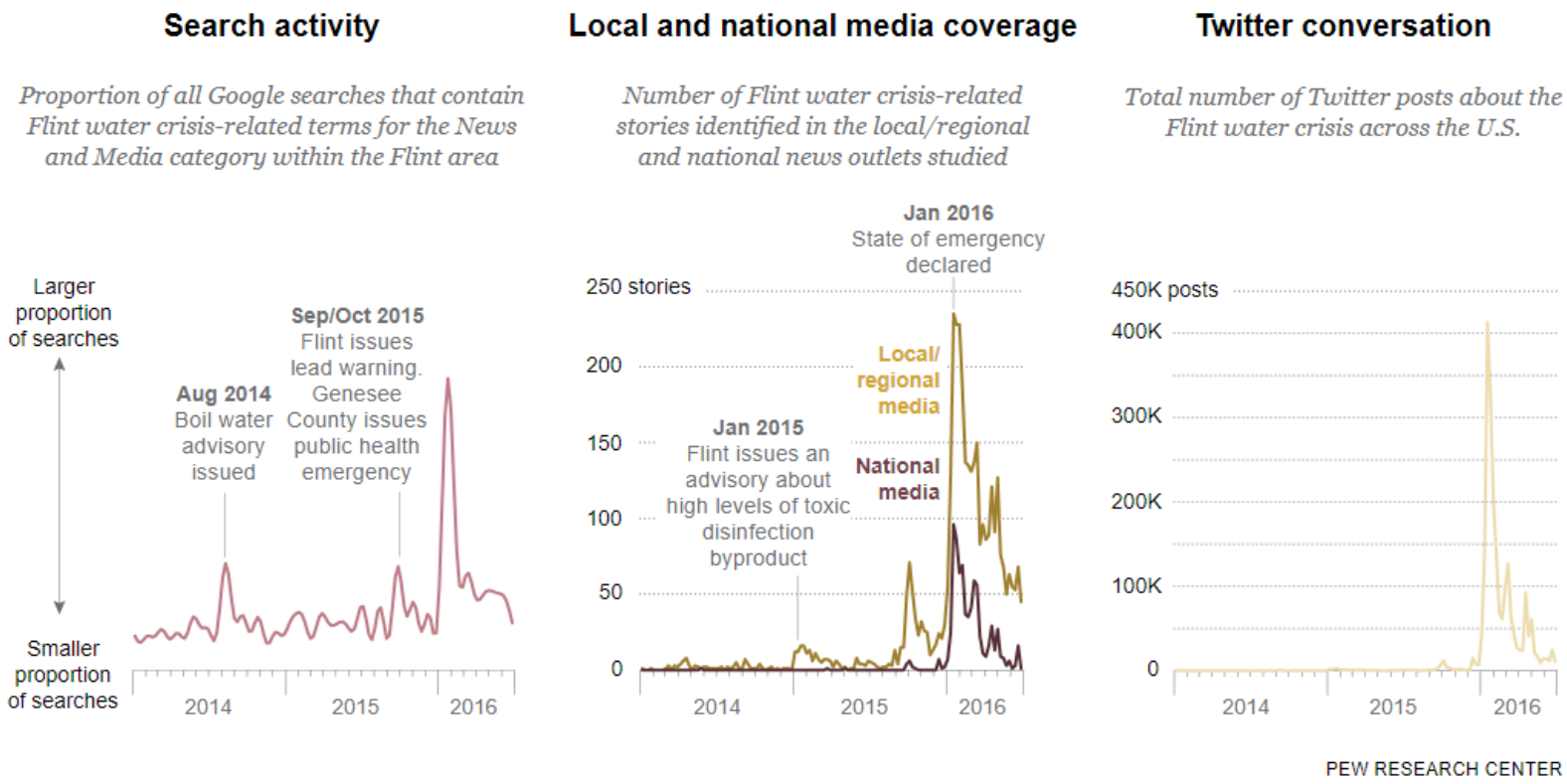
Source: US. Department of Health and Human Services, National Center for Health Statistics. National Vital Statistics Report, Vol 67, Number 1 (2018) & National Vital Statistics Report, Vol 66, Number 6 (2017)

Figure 2. 2 Timeline of the Flint Water Crisis 2011-2016, Flint, Michigan.



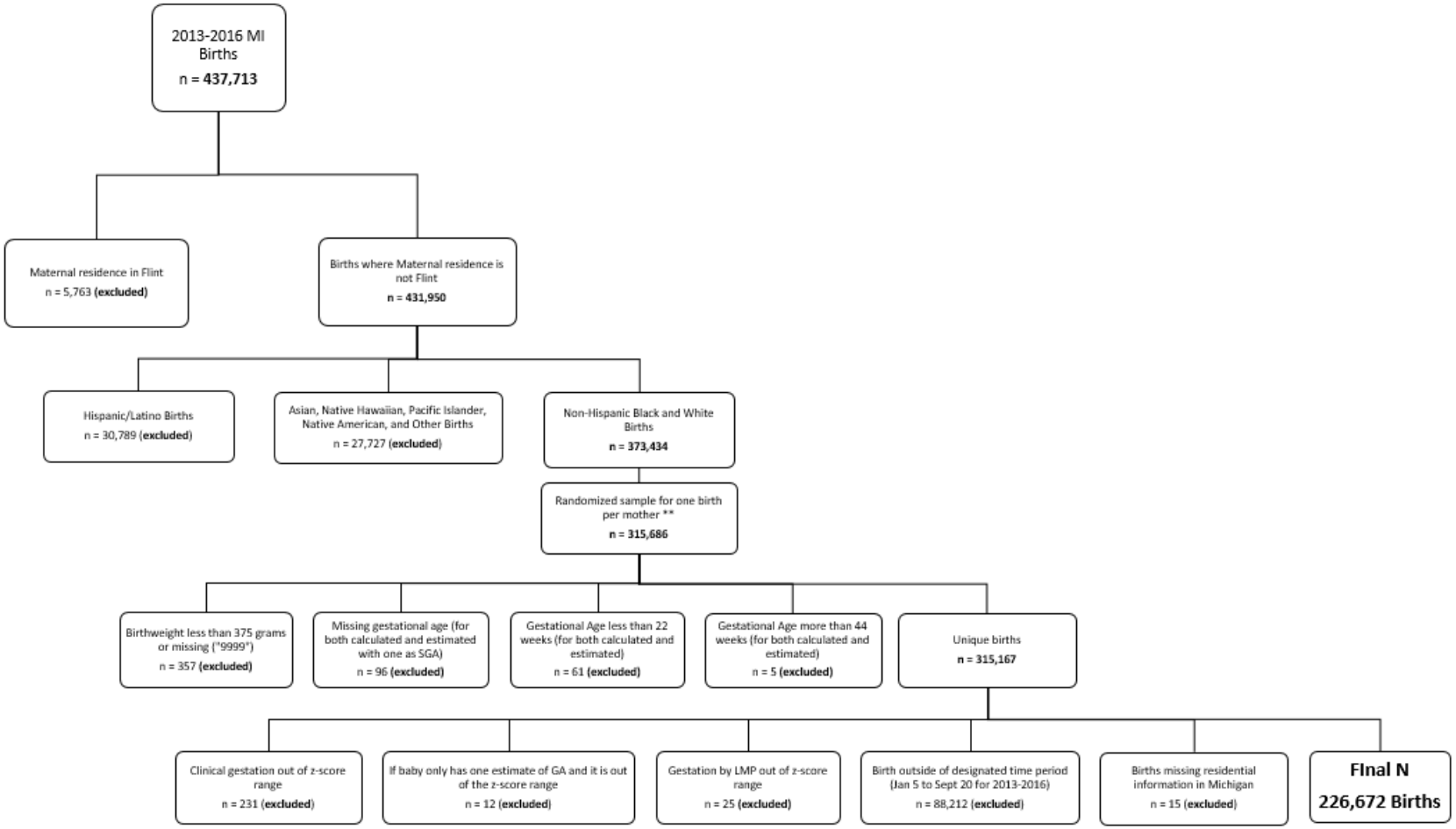
Source: Michigan Civil Rights Commission Report (2017); How Michigan and National Reporters Covered the Flint Water Crisis (2016), mediamatters.org; CNN (2017), Flint water crisis: How years of problems led to lead poisoning.

Figure 2. 3 Examination of News Media, Internet Searchers, and Social Medial Coverage during the Flint Water Crisis, PEW Research Center, 2017.



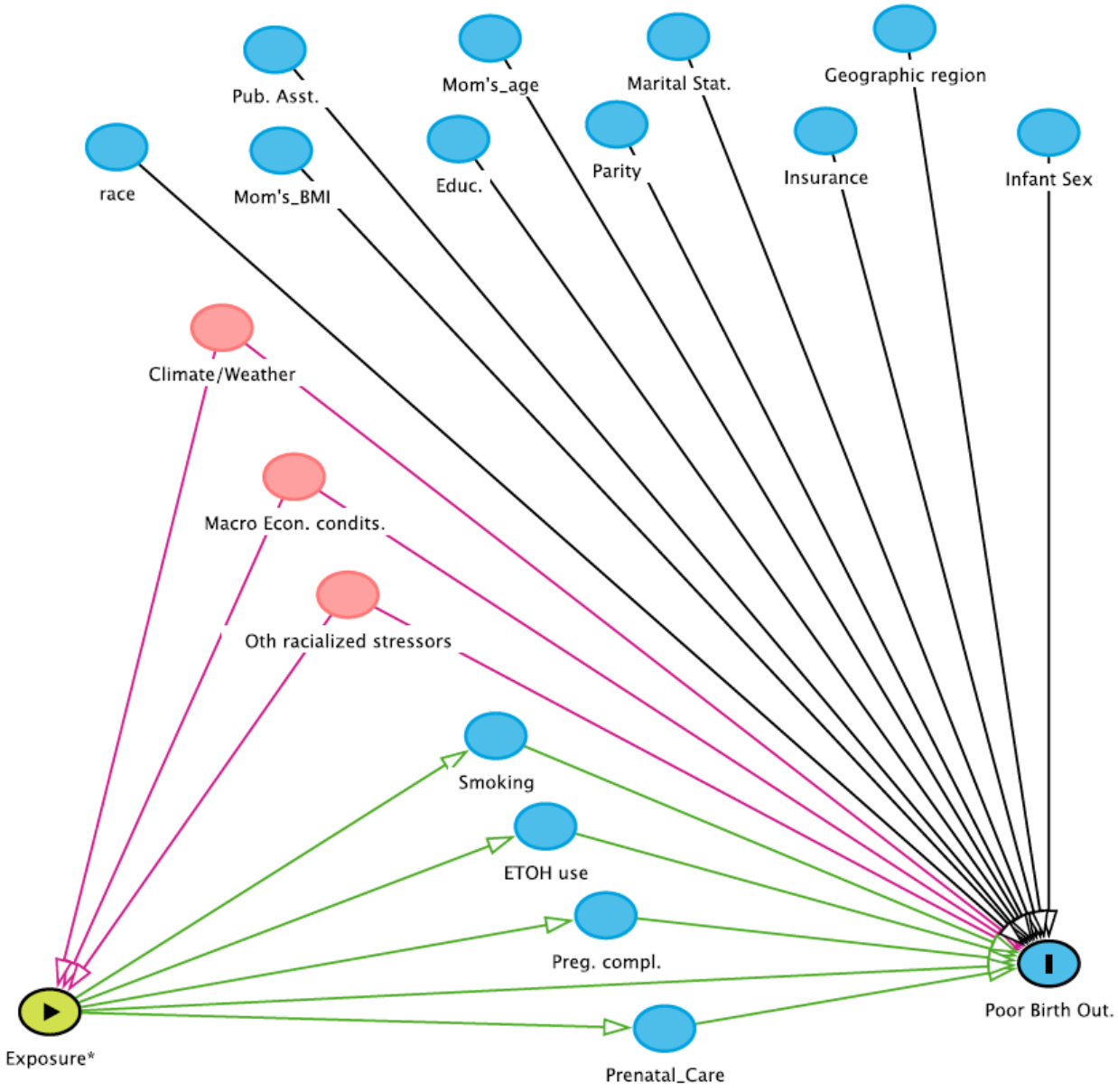
Source: http://www.journalism.org/essay/searching-for-news/

Figure 2. 4 Study Inclusion, Live Births, Michigan (MI), 2013 – 2016.



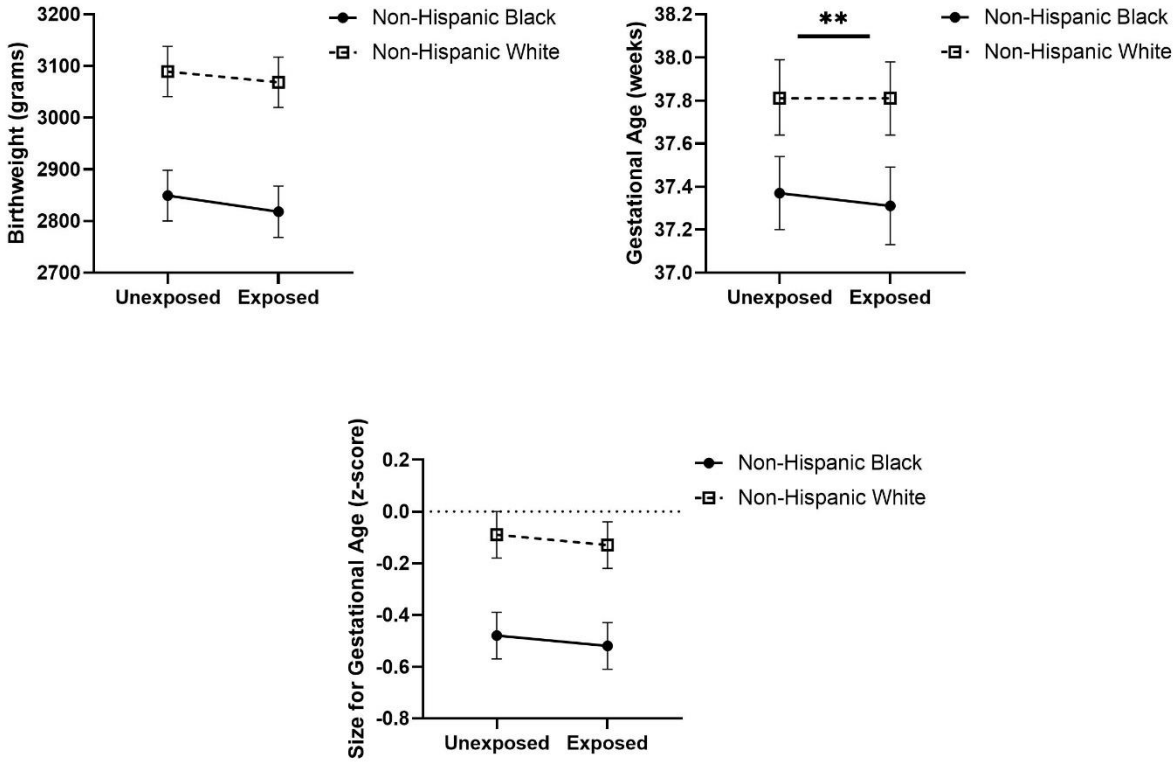
Source: Michigan Department of Health and Human Services, Division of Vital Records and Health Statistics. **Simple Random Sampling used to select baby/mother pairs in which mothers had 2 or more pregnancies during the study period (16% of births to Black or White mothers).

Figure 2. 5 Directed Acyclic Graph (DAG) of the Hypothesized Relationship Between the Declaration of a State of Emergency in Flint and Birth Outcomes (Birthweight, Gestational Age, and Size-for-Gestational-Age).



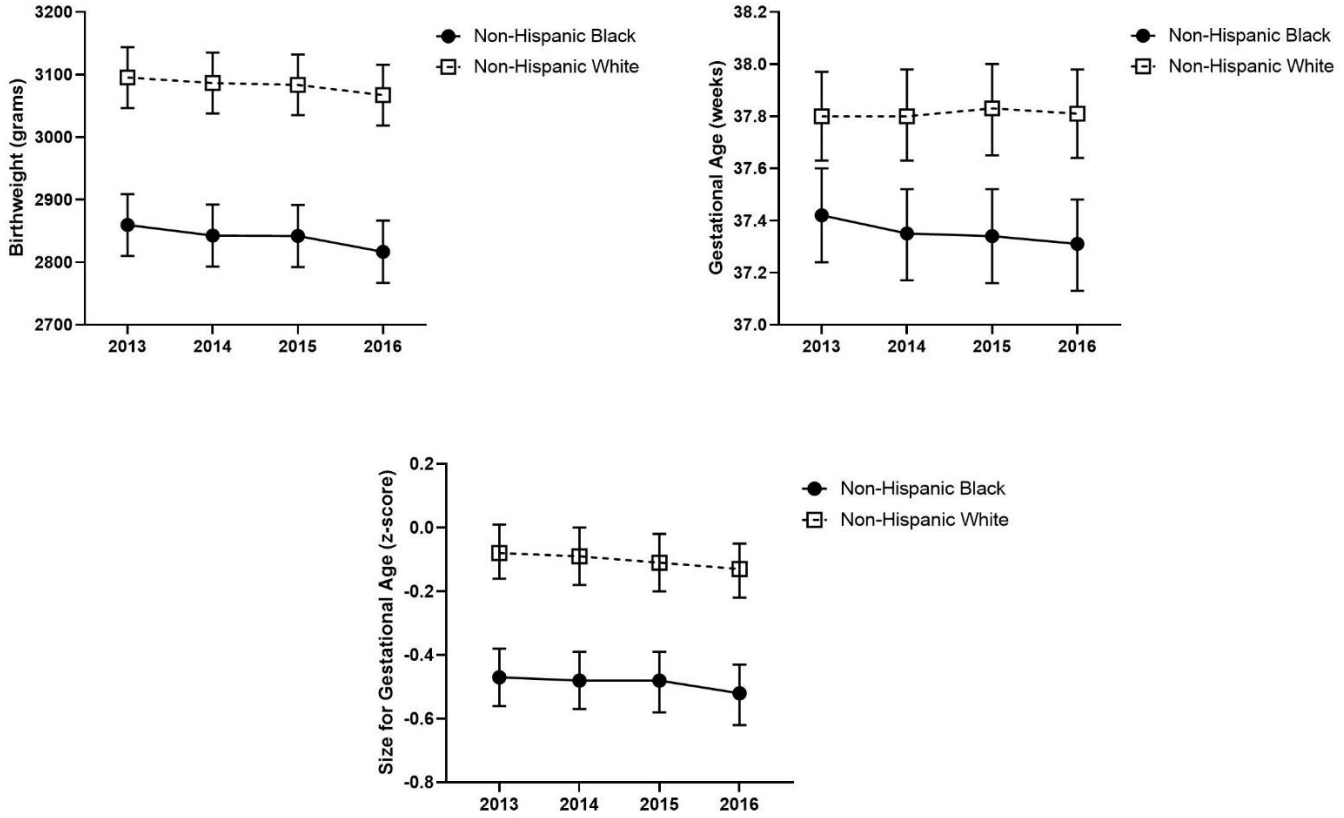
*Exposure=Vicarious exposure to the Flint Water Crisis through the news based on timing of birth

Figure 2. 6 Adjusted Means for Birthweight, Gestational Age, and Size-for-Gestational-Age by Maternal Race and Exposure to the Flint Water Crisis Emergency Declaration in Michigan (n=226,672).



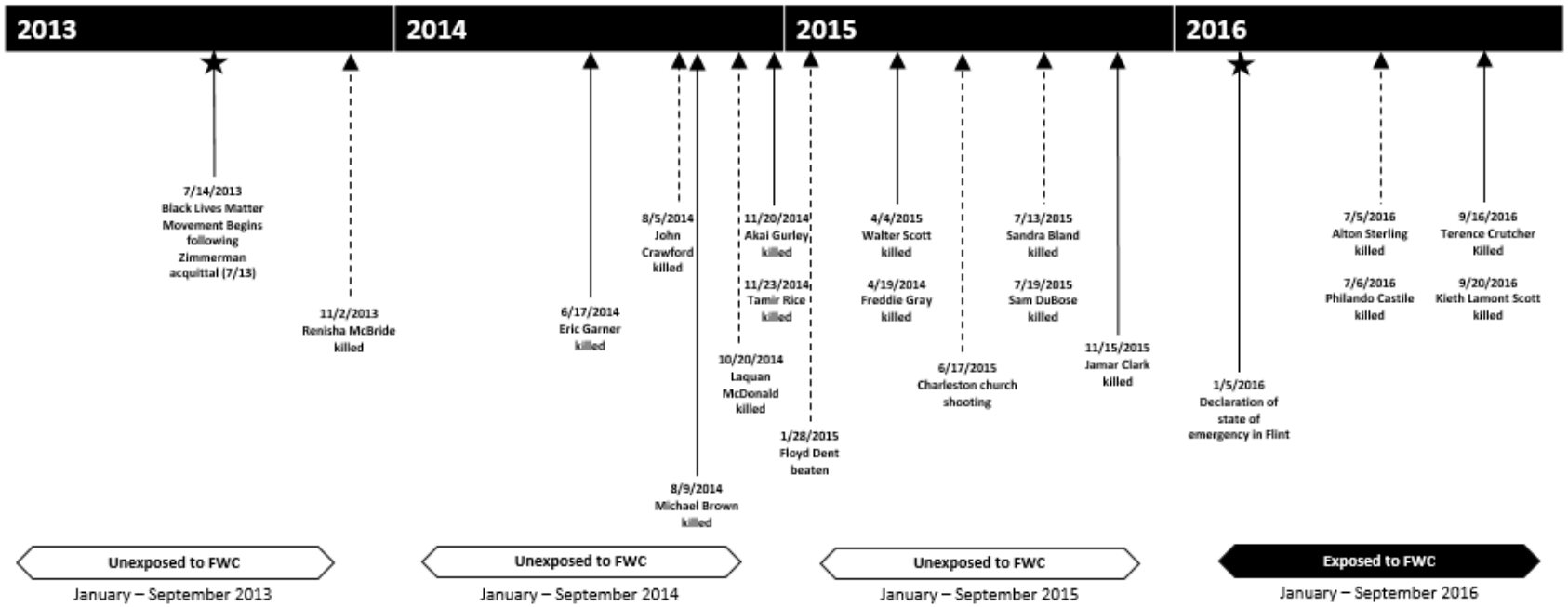
Note: Asterisks (**) denote marginal statistical significance of the race-by-exposure interaction at $\alpha < 0.10$. Means are from adjusted models.

Figure 2. 7 Adjusted Means for Birthweight, Gestational Age, and Size-for-Gestational-Age by Maternal Race and Year in Michigan, 2013-2016 (n=226,672).



Note: The race-by-year interactions at $\alpha = 0.05$ are not statistically significant. Means are from fully adjusted models.

Figure 2. 8 Timeline of Highly Publicized Instances of Racialized Violence Relative to Exposure Period, 2013-2016.



Chapter 3 Do Police Encounters Increase the Risk for Cardiovascular Disease? Police Encounters and Framingham 30-Year Cardiovascular Risk Score

"Neither slavery nor involuntary servitude, *except as a punishment for crime whereof the party shall have been duly convicted*, shall exist within the United States, or any place subject to their jurisdiction."

~Section 1, 13th Amendment of the US Constitution, 1865

3.1 Background

Recent examples of racially biased policing tactics are found in nearly every city across the country.[73,86,138,149,150,434-437] Cellphone cameras and rapid real time documentation of events has led to significant media attention on several recent police killings of unarmed Black men across the country, provoking significant protest demonstrations to rally against modern racial bias in the US criminal justice system.[38,150,168,438-440] The media and public health researchers have illustrated the consequences of aggressive policing,[73,75,89,149,161] but the focus on the rarer instances of deaths and injuries due to police intervention ignores the day-to-day experiences minorities face when dealing with the police which are more common and thus more likely to contribute to racial disparities in stress-related health outcomes,[441] such as cardiovascular disease (CVD), at the population level.[86] These commonplace police encounters (PEs) in minority communities have resulted in higher a likelihood of injury or death due to police intervention among minorities,[73,149,161,442,443] a lack of confidence in police, conflict and mistrust between the community and police,[441,444] and decreased morale of police members.[445] Additionally, the near constant flow of media reports depicting racial bias by the police, both violent and non-violent, suggest that minorities are constantly being reminded of their oppression.[438,439] Thus, it is reasonable to hypothesize that both the quantity and quality of police encounters differ by race and that health outcomes of people who experience police encounters would also differ by race, evaluated by effect modification, referred to as differential vulnerability.[75,438,439,446]

There are near daily news reports of police-involved killings of unarmed racial and ethnic minority groups in the United States (US), making it seem as if fatal PEs are common. However, the prevalence of *non-fatal* PEs is far greater.[172,447] Until recently, when depictions of high profile police-involved violence became commonplace in mainstream media, the health effects of every day PEs have been virtually ignored.[75,77,86,138,448] A small body of literature suggests that PEs are common in minority communities and may have a negative impact on health on those directly and indirectly experiencing PEs.[75,86,138,441,448] Our understanding of the relationship between PEs and health outcomes is severely limited by a lack of reliable data on PEs. More recently, research has focused on outcomes related to violent encounters with law enforcement or those that end in an injury or death.[73,75,138,161,449] While these outcomes are certainly worthy of careful examination, this line of research only captures part of the problem because we do not have a reliable national surveillance system related to PEs or even police use of force. Notably, producing a national database of violent police encounters was codified in the Violent Crime Control and Law Enforcement Act (1994), the largest criminal justice legislation in the US.[450]

3.1.a. Policing and Structural Racism

There is a large body of social science literature documenting the history of modern policing in the US.[35,130,140,160,166,451-454] Most historical research points to several US pro-slavery policies, such as slave patrols, runaway slave laws, a lack of anti-lynching laws, and the ending of the Civil War in 1865 as the foundation of modern policing.[130,131,145,146,200,455,456] While forming the modern-day police force, Reconstruction Era legislators took advantage of specific wording in the 13th Amendment in Section I: "Neither slavery nor involuntary servitude, except as a punishment for crime whereof the party shall have been duly convicted, shall exist within the United States, or any place subject to their jurisdiction."[131] This text provided the impetus needed to generate vagrancy laws in the South during reconstruction called the "Black Codes" and formed a brutish stereotype of black men.[38] These laws were written to be purposefully colorblind, but in the execution of these laws, Blacks were at higher risk for a vagrancy-related arrest.[35,115] The Black Codes generally stated that if a person (regardless of race) cannot prove employment, then they were to be arrested. The legal system was (and still is) racially biased, thus Blacks arrested for vagrancy rarely received due-process or their guaranteed rights afforded to them under the 14th and the 4th Amendments of the US Constitution.[35,115] Black persons who

were often sent back to work in the fields—the same fields they worked as slaves. This resulted in an abundance of free prison labor, and the conditions have been reported to be considerably worse than slavery.[35]

While the Black Codes underpin modern policing, it has evolved much over time. More recently, law enforcement policies are the result of the War on Crime and Drugs initially declared by President Nixon in the late 1960's during a time of extreme racial tensions across US cities.[457] These polices have been carried on by several presidential administrations, several congresses, and supported by the Supreme Court of the United States.

First, *de jure* discriminatory policies have determined the legality of most types of police surveillance at all levels of law enforcement.[35] Over the last several decades, the US Supreme Court has ruled in favor of providing authority to police agencies to fight the War on Drugs by enabling stop-and frisk *(Terry Stops)*, racial profiling, and using the neighborhood context as a rationale for a stop.[160,162,213,214] Coupling these rulings with federally endorsed incentives for police departments and prosecutors to focus almost solely on drug-related crime, local police departments have been engaging in state sanctioned paramilitary style law enforcement for decades.[35] Additionally, police are given full discretion on *whom* to target their Drug War resources and have focused on racial minorities who, based on stereotypes born out of fear of former slave revolts during the Civil War Reconstruction Era and repeated policies that limited access to resources, are assumed to be more prone to criminal behavior than Whites and must be surveilled and controlled.[35,38,128,160,162,213,214,239,266,440,458-462] This is despite the similarities between Whites and Blacks in the use of drugs such as marijuana and cocaine, the two main drug targets in the War on Drugs.[463,464,d] The high frequency of *Terry Stops* cannot be understated; in fact, between 2015 and 2019 there were approximately 65,000 reported *Terry Stops* in New York City alone, mostly involving innocent racial minorities.[163] During the same time period, there were approximately 5,000 police-involved killings in the entire US, [164] a clear indication that *Terry Stops* are far more common than fatal PEs.[160,165-169]

Second, the inaction of local police leadership and the federal justice system in both disciplining or correcting the behavior of problem officers who repeatedly engage in racially biased policing

[d] https://www.samhsa.gov/data/sites/default/files/cbhsq-reports/NSDUHDetailedTabs2017/NSDUHDetailedTabs2017.pdf

reinforces and almost encourages the police to engage in biased and forceful encounters with impunity.[38,150,168,434,436,465] In fact, the judiciary system has provided protection to those acting on behalf of the state through a legal doctrine called "qualified immunity." This protection initially came through the 1871 Civil Rights Act, which protected state actors from personal liability when mistakes were made in the course of their typical job.[201,202,466-470] The qualified immunity practice has been abused by individual officers who commit misconduct despite *de facto* practice of financial liability being directed to the employer or the city at which an officer works in civil trials—which has become very difficult to prove due to *de jure* processes.[35,204,224]

Finally, since the 1980's, Congress has enabled a paramilitary style of policing by providing and incentivizing local police agencies to receive surplus military equipment, making the local police even more deadly.[216,217] This re-distribution of federal resources came with a promise that local agencies had to focus most of their resources and attention to fighting the War on Drugs,[35] which they fought in minority neighborhoods.[35,471] Taken together, these federally sanctioned pathways for law enforcement's legal infringement on a citizen's constitutionally guaranteed rights appear, on the surface, as colorblind – there is no mention of race or ethnicity in any policy or legal ruling. The history of policing is a primary example of a structure or institution born out of racism.

3.1.b. PEs and Health

Researchers have begun to unpack the effect of PEs on health outcomes, despite extremely limited available data on PEs. Individual-level PEs are associated with reporting higher levels of fair/poor health,[133] higher waist circumference,[134] shorter leukocyte telomere length (a sign of accelerated aging),[135] stress disorders and anxiety,[139,151] and death and injury.[73,161,173,472,473] In emerging work at a local level in a sample of individuals in Chicago communities, Hirschtick et. al. (2019) reported a positive, but non-significant, association between PEs and post-traumatic stress disorder and depression.[474] Researchers examining community-level PEs have identified associations with individual outcomes such as diabetes, fair/poor health, obesity, and hypertension.[86,138,448] Other studies have examined vicarious, or indirect exposure to PEs demonstrating effects on mental health[75] and obesity.[134] Despite these studies, we know very little about the relationship between PEs and stress-related health outcomes such as CVD.

3.1.c. Racial/Ethnic Differences in Vulnerability to PEs

The increased media focus on violent police encounters has resulted in heightened fear and worry during routine traffic and street stops.[438,439] These extreme cases of police brutality have instigated public health professionals to recommend more research on the effects of PEs and health.[150,168,169] In addition to the differential exposure to police encounters by race, which could contribute to health disparities between Blacks and Whites, it is also possible that Blacks are more vulnerable to the encounters because of perceived unfairness.[86,138,448] For example, data from national surveys suggest that there are racial disparities in the perception of involuntary contacts with the police, where Blacks are more likely than Whites to report that the officer behaved improperly.[160,443,475] The additional burden of racial bias places racial minorities at an increased vulnerability to police encounters compared to Whites because of the multitude of other stressors that differentially affect racial minorities such as violence, financial issues, health issues, and personal and social traumas, among others.[476-479] Research suggests that because of their disadvantaged status, racial minorities in the US have numerous barriers to accessing the coping resources that may alleviate stress, further contributing to racial and ethnic health disparities.[6,446,480-482]

3.1.d. Racial Disparities in CVD

CVD is the leading cause of death in the US.[483] Despite declines in CVD mortality overall, there are well documented racial disparities in cardiovascular diseases, including mortality, over time.[3,484,485] Researchers have attributed at least some of the disparities in CVD outcomes to social factors, including racism.[486,487] Aside from disparities in traditional risk factors that contribute to CVD morbidity and mortality,[488] Blacks fare worse than Whites even at the same level of socioeconomic status (SES).[487] Additionally, Blacks have an earlier onset of CVD compared to Whites, contributing to differential morbidity and premature mortality.[487,489] Factors such as obesity, diabetes, hypertension, poor diet, and inactivity, among others that contribute to CVD mortality, each have their own racial disparities favoring Whites.[485,490-492] These traditional cardiovascular disease risk factors begin to form early in adulthood producing a higher risk for CVD later in life.[493-495]

3.1.e. Racism and CVD

Research has demonstrated repeatedly that discrimination affects both risk factors for CVD[496-498] and CVD events.[485,499,500] Studies suggest that racism in other sectors, like healthcare, contribute

to the racial disparities in CVD outcomes,[501] some of which stem from the reliance of debunked research in medical training,[27] harmful and unethical medical and research practices,[19,44] and US policies aimed to segregate hospitals.[502-504] While much of the discrimination research has focused on interpersonal experiences of discrimination, there are several forms of racism, the most pervasive form being structural racism.[12,13] Perceived discrimination is associated with a myriad of health effects, but it is not the only source of racism Black Americans experience that can damage health.[8,9,13,41,47,77,92,138,505]

Indeed, some research has emerged suggesting an association between structural racism and CVD.[77,126,506,507] Additionally, given that CVD is the leading cause of death in the US, risk factors for CVD have been well studied.[485,490,491] Many of these risk factors have been found to be associated with discrimination and/or racism.[126,496-498,500,508,509] With a long history of government-sanctioned medical experimentation on Blacks in the US, it is not surprising that Blacks distrust medical institutions.[44,105,501,510] This history likely contributes to racial disparities in CVD.

3.1.f. Stress and CVD

The stress process model suggests that there can be differential exposure and differential vulnerability to stressors.[476,478,511] It is well established that people residing in disadvantaged communities are exposed to more stress.[512] For example, racial minorities are more likely to be exposed to the police both non-violently and violently regardless of crime involvement.[73,138,161,171,172,447,448,459,462,513-515] Even after controlling for race-specific crime rates, Blacks are at least two times more likely than Whites to be stopped by police on the street for suspicion of weapons and drug related crimes.[459] Some of this is explained by the US government's endorsement of surveillance of racial minorities and a justice system that nearly universally permits law enforcement agents to racially profile to justify interactions,[35] which means that PEs experienced by Blacks, as well as other racial minorities, are more likely to involve the use of force[172,516] and lead to injuries, deaths[73,161,515] or other disparate outcomes compared to Whites.[38,73,75,138,448,517,518] Additionally, Blacks are four times more likely to experience violence by the hands of police compared to Whites.[38,519,520] Exposure to the police is undoubtedly stressful, and perhaps more so, to Black Americans who have plainly suffered by the hands of law enforcement since modern policing began.[521,522] Interactions with the police is

a substantial stressor for Blacks, and may contribute to poor health outcomes, like CVD through a biologic stress mechanism.[80,86,138,348,350-352,425,426,448,523-527]

3.1.g. Hypotheses

Law enforcement violence, a form of structural racism, has been described as a modern public health issue.[7,35,110,515] Despite the media attention of several recent cases of extreme police brutality, we know little about the health-related effects of more routine or day-to-day encounters with the police.[475] In the context of substantial and longstanding racial bias, demonstrated by group differences in excessive force and higher levels of surveillance by the police, even these commonplace and routine exposures may incur more stress on racial minorities than Whites, suggesting racial differences in vulnerability to police encounters.[86,438,439,476,528] While there is substantial evidence for differential exposure to the police by race,[73,132,138,161,171,172,447,448,459,462,513-515] in this chapter I explore differential vulnerability by examining the interaction of PEs with race on the Framingham 30-Year Cardiovascular (CVD) Risk Score in a nationally representative cohort of young adults. I hypothesize that the association between a high number of reported PEs and 30-year CVD risk will depend on race, such that the association between exposure to a high number of PEs on risk for a CVD event occurring in the next 30 years will be higher for Blacks compared to Whites.

3.2 Methods

The primary source of data for this chapter is the National Longitudinal Study of Adolescent to Adult Health (Add Health). The Add Health methodology has been extensively published elsewhere.[529-531] Briefly, Add Health is a nationally representative, school-based prospective cohort study of adolescents that examines behavioral, emotional, social, educational, and contextual factors as they transition to adulthood. The baseline sample was gathered beginning in 1995 when participants were in 7^{th} – 12^{th} grade using a complex clustered sampling design. The initial sampling frame was based on a list of schools from the Quality of Education Data, Inc. Eighty high schools and their accompanying feeder middle schools were selected using probability proportionate to size. Students in selected schools were stratified based on grade and sex, and 17 students within each stratum were selected with additional samples taken for ethnic minorities, Black children with at least one parent with a college degree, students with disabilities and siblings. Wave I included 20,745 adolescents who were representative of American adolescents with respect to region, urbanicity, school features, and ethnicity.

Subsequent Waves II-IV followed up with participants who completed Wave I. Field interviews were gathered in the participants' homes using Computer Assisted Personal Interviewing (CAPI) for non-sensitive questions or Audio Computer Assisted Self-Interview (ACASI) technology for sensitive questions. Topics ranged from crime and crime victimization to demographics, socioeconomic status, physical health, and risk behaviors. In addition to data collected through interviews, objective measurements (i.e., blood pressure, measured height and weight, etc.) were also taken after the interview using systematic data collection protocols. Finally, contextual data from the Census were included with the publicly available Add Health data.

Data for this study comes from Waves I, III and IV (specific variables selected are described below). Wave III data was collected in 2001-2002 when the initial cohort was approximately 18-26 years old with a 76% response rate. Wave IV was collected when the cohort was between 24 and 32 years old, or when personal habits and chronic diseases are beginning to affect the individuals. The response rates throughout Add Health were high, ranging from 76% in Wave III to 80% at Wave IV and approximately 9% of respondents who completed Wave III did not complete Wave IV.

3.2.a. Exposure at Wave III

The primary exposure is a high number of police encounters (PE) compared to low number of PEs measured at Wave III. The level of reported PEs is derived from the following question asked in Wave III: "How many times have you been stopped or detained by the police for questioning about your activities? Don't count minor traffic violations." This five-level categorical variable includes response options which range from 0 (never) to 6 or more times. This study is focusing on a high level of PEs. The highest category in the Add Health dataset is 6 or more PEs. After stratifying PEs by sex and race, the cell sizes for those reporting 6 or more PEs for women were considerably lower than that for men (n=11 and n=149, respectively) (Supplementary Table 3.1). The sex-difference in PEs has been established with prior research.[136] Additionally, after examining the primary analytic models with the exposure coded in its original form (5 categories), it became clear that there may be race and sex differences in the relationship between PEs and CVD risk at different levels of PEs for men and women (see Supplementary Tables 3.10-3.13). Thus "high" PEs was defined at 6 or more PEs reported for men and 2 or more for women compared to <6 PEs for men and <2 PEs for women, or "low" PEs. This new exposure classification has n=46 Black and n=103 White men who reported 6 or

more PEs, and n=36 Black and n=147 White women who reported 2 or more PEs or approximately 5% of the sample reported a high level of PEs (See Table 3.6).

3.2.b. Primary Outcome: Individual 30-Year Cardiovascular Risk at Wave IV

This study uses the Framingham 30-year cardiovascular risk score (CVD) calculated from variables collected at Wave IV as the primary outcome. The risk score predicts the risk of CVD events occurring within the next 30 years. CVD risk scores are useful clinical tools that provide a patient with a general risk assuming nothing changes. A CVD risk score can be considered a point of intervention where those with high risk scores can be targeted for therapeutic or behavioral interventions that can reduce a patient's risk for fatal or non-fatal CVD.[490,491] The SAS macro code used to generate the individual risk score was provided by Dr. Pencina and Mr. Williams from Duke University and Kenanco Biostatistics, respectively. The code provided included four macros which predicted cardiovascular risk in 30 years using a Cox proportional hazards model that accounts for competing causes of death using data from the Framingham Heart Study. The Cox model included the following covariates: age, sex, use of antihypertensive medications, smoking status, diabetes status, systolic blood pressure, and body mass index.

3.2.c. Variables in the Index (See Appendix)

Male is a binary indicator for sex which is coded as 1 if the participant is male and 0 if the participant is female. Sex is self-reported at all waves. *Age* is a continuous value in years at the time of the Wave IV interview. All participants were aged 24-32 years by the Wave IV interview. *Systolic blood pressure (SBP)* is continuous and measured by the interviewer using a standard method and equipment. Briefly, each participant's right arm was measured using a standard tailor's measuring tape after bulky clothing was removed to select a proper blood pressure cuff. Resting blood pressure was taken with participants seated three times in 30 second intervals using a calibrated Microlife BP3MC1-PC-IB oscillometric blood pressure monitor (MicroLife USA, INC., Dunedin, FL).[532] The *use of anti-hypertensive medications* is a self-reported binary measure derived from lists of reported medication classifications. Participants reporting the following medication classifications were coded as taking anti-hypertensive medications: thiazide diuretics, angiotensin II inhibitors, antihypertensive combinations, vasodilators, calcium channel blocking agents, beta-adrenergic blocking agents, cardio-selective beta-blockers, non-cardio-selective beta-blockers, antiadrenergic agents (centrally and/or

peripherally acting), angiotensin converting enzyme inhibitors, and agents for hypertensive emergencies.

Smoking is defined as reporting at least one cigarette per day in the last 30 days. *Having diabetes* is based on several factors including: elevated blood glucose levels (fasting or non-fasting), elevated A1c levels, self-reporting a diabetes diagnosis, and/or self-reporting taking diabetes medications. Fasting glucose levels exceeding 126 mg/dL or non-fasting glucose exceeding 200 mg/dL and/or hemoglobin A1c levels exceeding 6.5% were considered diabetic.[533] Self-reported diabetes medication use was derived from a response to a question about taking diabetes medications in the past four weeks. *Body Mass Index (BMI)* is calculated as measured weight divided by measured height-squared. Height and Weight were measured in the field using standard procedures. Height is measured using a carpenter's square, a steel tape measure and a sticky note. Respondents were asked to stand on a smooth, non-carpeted flooring, the interviewer placed the carpenter square flush against the wall at the top of the respondent's head and placed a sticky note at the height. The tape measure was used to measure height in centimeters (cm) to the nearest 0.5 cm.[532] Weight was measured using a Health-o-meter 844 KL High Capacity Digital Bathroom Scale (Jarden Corporation, Rye, NY) with a maximum weight of 200 kg after being placed on a hard flat surface. Weight was recorded to the nearest 0.1 Kg.[532] Using the standard calculation BMI is a continuous variable in kg/m^2.

Using these variables from Add Health, after applying the Pencina and Williams SAS macro, the resulting two CVD indices are continuous measures ranging from 0-100% and interpreted as 30-year cardiovascular (CVD) risk levels for several cardiovascular outcomes including: coronary death, myocardial infarction, fatal and non-fatal stroke, coronary insufficiency, angina pectoris, stroke plus transient ischemic attack, intermittent claudication, and congestive heart failure.[491,534,535] The first 30-year CVD risk outcome generated predicts "hard" CVD outcomes including coronary death, myocardial infarction, and fatal and non-fatal stroke, while the second 30-year CVD risk outcome generated predicts "full" CVD outcomes including those in the hard outcomes in addition to coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure. As a sensitivity analysis, a few cut points were used to examine the robustness of the findings. Cut-points were defined as 30-year CVD

risk scores of 20% or higher and 10% or higher for each outcome. The cut-points were selected based on previously published research.[535]

3.2.d. Covariates

The covariates in this analysis are based on *a priori* existing knowledge from extensive literature reviews (Figure 3.1).[474,485,491,500,536-538] Age is derived by taking the number of days between the initial Wave I survey in which date of birth was collected and the date of the Wave III survey follow-up and dividing by 365.25. Early socioeconomic status is based on the participant's reporting of using or needing public assistance prior to age 18 in either Wave III or IV. Those who were either not asked this question or for whom there was no response to this question in Wave III were asked in Wave IV. Thus, those asked in Wave III were used and if a response was missing in Wave III, the Wave IV response was used.

Sex is derived from a question asked at Wave I. Interviewers are prompted to confirm the respondents' sex at the time of each interview and ask the respondent if necessary. Wave I reported gender is used in all analyses.

Race is self-reported and based on a series of yes/no questions: "What is your race?" with the following options: White, Black/African American, American Indian/Native American, Asian Pacific Islander, and Other. Additionally, ethnicity is derived from the question: "Are you of Hispanic or Latino origin?" Those who affirmatively answered that they were of Hispanic or Latino origin were coded as Hispanic regardless of their race, and each race was coded as 'yes' if they reported that race and were labeled as non-Hispanic. Since the social experience of mixed race persons, Black and White, historically reflect the experience of Black or African Americans, those reporting Black race with a combination of any other race are coded as Black. This is also true for other races such as Asian, American Indian or other race. Those reporting White are those who remain after non-white races were coded. Finally, this analysis is limited to those self-identifying as non-Hispanic Black or non-Hispanic White. Race is a hypothesized effect modifier.

Education is derived from the question "What is the highest level of education that you have achieved to date?" This question was asked of all participants in Wave III. From this question education levels are considered: less than high school, high school graduate, college degree or more. Current household income at Wave III is derived from the question "Thinking about your

income and the income of everyone who lives in your household and contributes to the household budget, what was the total household income before taxes and deductions in [2000/2001]? Include all sources of income, including non-legal sources." The responses for this question were a 12-level categorical variable ranging from less than $5000 to $150,000 or more. These categories were combined to a 4-level income variable (<$19,999, $19,999-$49,999, $50,000-$74,999, and ≥$75,000). The definition of urban and rural have changed substantially over time according to the US Census definitions. Add Health does not contain all of the needed variables to apply the definition of urban/rural in the same way as the US Census. Thus, population density is used as a proxy for urbanicity.[539]

3.2.e. Statistical Analysis

The analysis begins with an examination of each variable and its associated distributions. Means and standard errors were examined for continuous variables and proportions were examined for categorical variables. Each continuous variable, including both outcomes, was assessed for a normal distribution by using histograms, box plots, q-q plots and scatter plots and their associated indicators to establish normality. Both outcomes as calculated were substantially right skewed with long tails. After analyzing the data using regression models, the errors produced by both risk scores were non-normally distributed. Due to the non-normality of the errors in regression I subsequently log transformed both outcomes and re-evaluated their distributions. In both cases the distributions of the risk scores and the evaluation of the errors terms produced though regression were normally distributed after log transformation, an expected result based on prior research using Add Health data.[534,535,540] No other variables needed log transformation. Bivariate associations were examined and reported in Table 3.1. Each variable was stratified by both race and exposure status. All variables included in the models (described below) are included in Table 3.1. To determine the statistical difference between the groups, I used the t-test to examine group means for continuous variables and the modified Rao-Scott chi-square test to examine difference in proportions. A statistical significance level of 0.05 was considered significant.

To examine differential vulnerability by race to PEs, I employed the generalized least squares regression using the Taylor Series Linearization method to estimate regression coefficients and standard errors to test the association between PE*race interaction and each 30-year CVD risk (full and hard outcomes). I report a basic model which includes PEs and race (model 1). I report

a *covariate adjusted model* that includes PEs and race, plus the following covariates: sex, age, early SES, population density, and income (model 2). I report the *interaction model* that includes an interaction between PEs and race, plus all covariates included in Model 2 (model 3). Additionally, I report stratified models for non-Hispanic Blacks and non-Hispanic Whites for both models 1 and 2.

(1) $Y_{30-yr\ CVD\ risk} = \beta_0 + \beta_1 PE + \beta_2 race\ \varepsilon$

(2) $Y_{30-year\ CVD\ risk} = \beta_0 + \beta_1 PE + \beta_2 race + \beta_3 sex + \beta_4 early\ SES + \beta_5 education + \beta_6 population\ density + \beta_7 income + \beta_7 age + \varepsilon$

(3) $Y_{30-year\ CVD\ risk} = \beta_0 + \beta_1 PE + \beta_2 race + \beta_3 sex + \beta_4 early\ SES + \beta_5 education + \beta_6 population\ density + \beta_7 income + \beta_8 age + \beta_9 PE * race + \varepsilon$

Specifically, I hypothesize that the association between PEs on 30-year CVD risk will depend on race, such that the association between PEs and CVD risk will be greater for Blacks than for Whites.

In sensitivity analyses I examine binary outcomes using the cut-point values set at $\geq$20% (out of a continuum of 0-100% risk) and $\geq$10% using a logistic regression analysis. I also examined different exposure measurement with varying PE cut-points in separate analyses: 1 or more vs never, 2 or more vs <2 PEs, 4 or more vs <4 PEs, 6 or more PEs vs <6 Pes, the original coding scheme with never as the reference and with 6 or more as the reference, and sex- and race-stratified models with PEs set to the original coding, and sex-specific PEs stratified by gender.

All analyses are conducted using survey packages available in SAS 9.4 including survey cluster, strata and weight variables as directed by Add Health analytical guidelines.[530] All stratified analyses are done using domain analysis per the recommended analytic guidance.[530] Interpretation of the log transformed variables can be examined by taking the anti-log of log-transformed value produced in the models and will provide a geometric mean (similar to a median) value for a risk level.

3.2.f. Checking Regression Assumptions

Given the linear regression analysis, I examined the meeting of the standard assumptions of the regression: independence of errors, homoscedasticity, linearity, and a normal distribution of the residuals. The independence assumption was evaluated with the generalized Durbin-Watson statistic which may suggest autocorrelation, however due to the clustered nature of the data this statistic is less reliable to judge independence. Homoscedasticity (constant variance) is visually evaluated by a scatterplot of the residuals versus the predicted values. If no pattern is present then it can be assumed that this assumption has been met. Linearity is assessed by examining a scatterplot of residual versus each continuous variable (age). Finally, a q-q plot was used to examine the assumption of normal residuals. Goodness of fit was assessed using the R-square statistic. Influential points were examined by generating the studentized residuals, leverage, and the influence of observations on parameter estimates.

3.2.g. Missing Data

The amount of missingness ranged from 0% (race) to 4.6% (household income) for all variables in the analysis. Since income had about 5% of respondents with missing data, a missing category was created so that those could be retained in the analysis. After the missing values were accounted for using the missing category, all of the remaining missing data were excluded, thus a complete case analysis was conducted dropping n=759 respondents (8.2% of the overall analytic sample). This method is supported by the literature when missingness is low.[541,542]

3.3 Results

Table 3.1 displays the descriptive statistics by race and by exposure status. Blacks reported lower incomes ($p<0.0001$), were more likely to use public assistance before age 18 ($p=0.0028$), had higher unadjusted full CVD risk ($p=0.0002$) and a higher unadjusted hard CVD risk ($p<0.0001$) compared to Whites. There were no differences between Blacks and Whites for age, sex, educational attainment, population density, and exposure to the police. Those reporting high levels of PEs were slightly younger ($p=0.0012$), had less education ($p<0.0001$), lived in more densely populated areas ($p=0.0007$), and were more likely to use public assistance before 18 years old ($p=0.0062$) compared to those reporting low PEs. There were no differences between those reporting high PEs for race, sex, income, unadjusted full CVD risk, and an unadjusted hard CVD risk compared to those reporting a low number of PEs.

Table 3.2 displays the liner regression coefficients for the regression of CVD risk (full) on high vs low PEs. After controlling for covariates, there is a positive but non-significant association between high PEs and 30-year CVD risk (model 2, beta coefficient for PEs=0.004, p-value=0.9005). I observed a positive and statistically significant association between race and 30-year CVD (full), where Blacks have a higher risk for a full CVD event occurring in the next 30-years compared to Whites (model 2 beta coefficient for race=0.088, p<0.0001). I also observed a negative and statistically significant interaction between race and PEs (model 3 beta coefficient for interaction=-0.276, p=0.0061) suggesting a *lower* 30-year full CVD risk among Blacks compared to Whites among those reporting a higher number of PEs.

Table 3.3 displays linear regression coefficients for the regression of CVD risk (hard) on high vs low PEs. Similar results to those in Table 3.2 were found for the 30-Year (hard) CVD risk. After controlling for covariates, there is a positive but non-significant association between high PEs and 30-year CVD risk (model 2, beta coefficient for PEs=0.010, p=0.8136). I observed a positive and statistically significant association between race and 30-year CVD (hard), where Blacks have a higher risk for a hard CVD event occurring in the next 30-years compared to Whites (model 2 beta coefficient for race=0.109, p<0.0001). I also observed a negative and statistically significant interaction between race and PEs (model 3 beta coefficient for interaction term=-0.334, p=0.0044) suggesting a *lower* 30-year hard CVD risk among Blacks compared to Whites among those reporting a higher number of PEs.

In order to examine whether the associations of PEs with CVD risk is greater for Blacks compared to Whites, I next look to the stratified analysis by race. Table 3.4 displays the predicted values and group differences of CVD risk for both outcomes stratified by race and PEs. I observed that among Blacks, those with a high number of PEs have a lower 30-year CVD risk compared to those with a low number of PEs. This is observed for both the full and hard 30-year CVD outcomes (full CVD risk score difference for Blacks: -0.205, 95% CI: -0.347, -0.036, p=0.0178; Hard CVD risk score difference for Blacks: -0.249, 95% CI: -0.449, -0.049, p=0.0151). This finding was not observed for Whites. Among Whites, the difference between those reporting high numbers of PEs and those reporting low number of PEs was positive but not statistically significant for either outcome (Full CVD risk score difference for Whites: 0.051,

95% CI: -0.011,0.012, p=0.1074; Hard CVD risk score difference for Whites: 0.065, 95% CI: -0.007, 0.138, p=0.0772).

In an additional stratified analysis by sex (Table 3.5), I observed that among males, there is a negative and statistically significant difference between those reporting high vs. low PEs for both outcomes (full CVD risk score difference for males: -0.166, 95% CI: -0.282,-0.049, p=0.0057; hard CVD risk score difference for males:- 0.201, 95% CI: -0.335, -0.066, p=0.0037). A different picture emerges for females where there is a positive but not statistically significant difference between women reporting a high number of PEs compared to reporting a low number of PEs for both outcomes (full CVD risk score difference for females: 0.023, 95% CI: -0.124, 0.171, p=0.7549; Hard CVD risk score difference for females: 0.037, 95% CI: -0.140, 0.213, p=0.6814).

In the final stratified analysis, I examined the racial and sex predicted mean CVD risk scores and their within group differences (Table 3.6). I observed negative within group differences for Black males and females, and White males, but a positive within group difference for White women. The differences for Black men (-0.273, 95% CI: -0.476, -0.070, p=0.0089) and White women (0.137, 95% CI: 0.057, 0.216, p=0.0009) are statistically significant, indicating that high PEs are protective for Black men but increase CVD risk for White women.

3.3.a. Sensitivity Analyses

I conducted sensitivity analyses where I varied the threshold for high PEs at never vs. 1 or more, 2 or more vs <2, 4 or more vs <4, and 6 or more vs <6. The sensitivity analyses generally support the primary analysis where those with high PEs have lower risk for a cardiovascular disease event in the next 30 years (Supplementary Tables 3.1 – 3.20). The findings from the main analysis were similar for those with 1 or more PE (Supplementary Tables 3.2 and 3.3), but not for those with 2 or more PEs (Supplementary Tables 3.4 and 3.5). Additionally, when the number of PEs are higher (i.e., >=4 PEs or >=6 PEs), Supplementary Tables 3.6-3.10, the results were also similar. Consistent with the main findings, while Blacks have higher risk of cardiovascular disease in general (positive beta coefficients for the race term), the interaction between PEs and race resulted in a negative beta coefficient especially at higher PEs (>=6 PEs) where CVD risk is lower for Blacks than Whites (Supplementary Tables 3.8 - 3.13). For example, Blacks reporting 6 or more PEs have a 27% *lower* full CVD risk score [(exp(-0.039-

0.278)-1)*100] than Blacks reporting <6 PEs, while Whites reporting ≥6 PEs have a 4% *lower* risk for full CVD risk score [(exp(-0.039)-1)*100] compared to Whites with <6 PEs (Supplementary Tables 3.8). This difference is statistically significant (p for exposure*race interaction=0.0217). Additionally, Blacks reporting 6 or more PEs have a 32% *lower* hard CVD risk score [(exp(-0.041-0.339)-1)*100] compared to Blacks reporting <6 PEs, while Whites reporting ≥ 6 PEs have a 4% *lower* hard CVD risk score [(exp(-0.041)-1)*100] compared to Whites reporting < 6PEs (Supplementary Tables 3.9). This difference is statistically significant (p for exposure*race interaction=0.0044). Finally, sex-stratified analyses reveals that among men there are racial differences in CVD risk (full and hard) but only for those with 6 or more PEs (Supplementary Table 3.14 and 3.16), where White men have a higher CVD risk at higher levels of PEs compared to Black men. This finding is not observed among women (Supplementary Tables 3.15 and 3.17).

Additional sensitivity analyses were run examining cut-offs of 10% and 20% for hard and full 30-year CVD risk using logistic regression analysis. The sensitivity analyses support our main findings for the 30-year CVD risk (hard) score (Supplementary Tables 3.19 and 3.20) but not for the full risk score (Supplementary Table 3.18). With the full CVD index set at a cut-point of ≥20% (high risk) for a CVD (full) event in the next 30 years, the adjusted odds ratio is protective, but unlike the main findings, not statistically significant (aOR=0.803, 95% CI: 0.619, 1.043), see Supplementary Table 3.18. With the hard CVD index set at a cut-point of ≥20% (high risk) for a CVD (hard) event in the next 30 years, the adjusted odds ratio is protective (aOR=0.593, 95% CI: 0.359, 0.982), see Supplementary Table 3.19. When high risk is defined at ≥10% for the hard CVD risk score, the adjusted odds ratios is also protective (aOR=0.825, 95% CI: 0.690, 0.987), see Supplementary Table 3.20. Both of the results in Tables 3.19 and 3.20 are consistent with the main findings.

3.3.b. Variables in Index

Supplementary Table 3.21 displays the variables that make up the CVD indices by race and by exposure level. There were no statistically significant racial differences between age, sex, and the use of hypertension medication use. There was higher systolic blood pressure (p<0.0001), a higher prevalence of diabetes (p<0.0001), and a higher body mass index (p<0.0001) among Blacks compared to Whites. Whites were more likely to have recently smoked (p=0.0015) compared to Blacks.

Those with higher levels of PEs are slightly older (p=0.0004), more likely to have recently smoked (p<0.0001), and have a lower body mass index (p=0.0005) compared to those with lower PEs. There were no differences between those reporting high and low PEs with respect to sex, taking hypertension medications, systolic blood pressure, and prevalence of diabetes.

3.4 Discussion

This study examined a differential vulnerability hypothesis which states that an association between a high number of PEs and 30-year cardiovascular risk score depends on race, such that the effect of exposure to a high number of PEs on the risk for a CVD event occurring in the next 30 years will be higher for Blacks compared to Whites. I observed that the relationship between PEs and 30-year CVD risk score is dependent on race, but not as hypothesized. I observed a statistically significant interaction between race and PEs which suggests that the relationship between PEs and CVD risk differs by race for both outcomes. While examining the data stratified by race, I observed a statistically significant difference between 30-year CVD risk among Blacks in which Blacks reporting a high level of PEs have *lower* risk of having a future CVD event compared to Blacks reporting a low level of PEs; whereas among Whites, the risk was not statistically different for high compared to low PEs (Table 3.4). In the sex stratified analysis (Table 3.5), I observed statistically significant associations between PEs and CVD risk among men for both CVD risk scores: men with a high reported level of PEs had a statistically significant *lower* risk of a CVD event occurring in the next 30 years compared to men with low PEs. In contrast, among females, the risk level is in the hypothesized direction – high CVD risk at higher PEs--but not statistically significant. In the final stratified analysis by both sex and race I observed that there were statistically significant associations between PEs and CVD risk (full and hard) among Black men and White women, but the differences observed among Black women and White men were not statistically significant. Among Black men the CVD risk was lower among those reporting a high level of PEs, while among White women the CVD risk was higher with higher reported PEs.

The two hypotheses for this study were that (1) exposure to a high level of PEs would be associated with CVD risk and (2) the association would be stronger for Blacks than Whites. In general, but not always, studies report that Blacks have a higher level of exposure to the police compared to Whites.[73,161,440,451,472,543,544] In this study, I observed a similar level of police exposure between Whites and Black (Table 3.1). While seemingly counterintuitive, it is

somewhat consistent with some reports about police exposure by race at the time of the measurement (~2001). The US Department of Justice reported similar exposure to the police overall between 2001 and 2011 for Whites and Blacks (20% for Whites and 17% for Blacks).[172] However, the same study reported that the quality of the interactions differed by race where Blacks were 2.5 times more likely to experience a threat or use of force.[172] The question in Add Health refers to the quantity of encounters and not the quality of these encounters, which may have a stronger relationship with health outcomes.[545] Additionally, while Terry stops were legal in 2001, the use did not peak until about 10 years later.[163]

The main results of the first analysis revealed lower CVD risk among Blacks with high PEs compared to Blacks with Low PEs and Whites. Prior research suggests that direct exposure to the police is associated with poor health outcomes such as self-rated health, PTSD, injury, death, and biological markers of poor health.[73,133-136,138,161,474,499,546-550] While our findings that Blacks reporting a higher number of PEs have lower CVD risk compared to Blacks reporting a low number of PEs are counterintuitive to this line of reasoning, they are supported by prior research which observed either weaker or protective effects of major discrimination or stressful life events on poor health outcomes among Blacks compared to Whites.[551,552] This research is relevant to this study as police encounters are often perceived as discriminatory and in many encounters are considered major stressful live events.[172,553]

There are several potential explanations for this seemingly counterintuitive finding. First, while disadvantaged neighborhoods, or neighborhoods most likely to be surveilled by police,[35] on the surface seem to be constrained by limited resources, there are also several positive things that disadvantaged communities have that may alleviate the effects of stress through coping mechanisms.[381] Some coping mechanisms are health promoting (e.g., exercise) while others are health inhibiting (e.g., comfort food eating). These coping strategies can thus cause both immediate comfort and long-term damage to one's health. The Add Health population, who are, at the time of this study, entering into adulthood, may not have developed the behaviors that are detrimental to one's physical health yet. For example, in my study, the BMI for those with high PEs was significantly lower than those with low PEs (See Supplementary Table 3.21). Some research suggests that in adults with higher levels of PEs (i.e., the previously incarcerated), men with a history of incarceration had a lower likelihood of adult weight gain than those without an

incarceration history; there were no differences for women and within males there were no racial differences.[554] In this study, the BMI for those with high PEs was significantly lower than those with low PEs (See Supplementary Table 3.21). This suggests that perhaps, the police approach healthier appearing men with health promoting habits that place them at a lower risk for CVD later in life

Another potential explanation for the unexpected results of this study is called the "Black-White paradox in health" where despite the social and economic disadvantage faced by Black Americans, they tend to have better mental health than other groups.[555,556] This has been attributed to resilience, positive coping, and flourishing.[556] While others suggest that reporting major discrimination is associated with a higher risk of inflammation markers at older ages, this effect is weaker among Blacks compared to Whites, despite Blacks reporting more discrimination than Whites.[551] This may suggest a theory that has been coined "what is common becomes normal."[557] This line of research has demonstrated that in areas where children are obese or overweight, mothers perceive their overweight children as normal weight. This is thought to be the product of comparing the weight of their children to others and perceiving it as normal. This theory can be translated to PEs, because Blacks are more likely to experience PEs than Whites, thus it is a normative experience in many Black communities, while it is less normative in White communities.[35] Given Whites' privileged social status, there may be a larger effect of the experience of PEs because of a diminished capacity or ability to deal with this social adversity.[555,556,558] This perception of "normal" may be protective against the daily discrimination Blacks face by the hands of police as a result, while also being harmful to Whites, a phenomenon that has been observed numerous times in several studies examining racial disparities in health outcomes.[109,556,558-561] Perhaps this is most relevant to the race and sex stratified results which suggest that high PEs are associated with a lower CVD risk among Black men and a higher CVD risk among White women, a result also observed in other studies.[552] Compared to other racial and ethnic groups, Black men have higher exposure to the police both violently and non-violently while White women have the lowest.[141,170,562] Thus, PEs are common among Black men and not common among White women.

This study has several limitations. The PE exposure in this study was derived from one question that had limited options to categorize the number of PEs. The number of PEs went up to 6 or

more rather than allowing individuals to state the estimated number of times they had encountered the police. The findings when the PEs cut-point was set at a higher level (e.g., 6 or more) were generally supported by sensitivity analysis where those reporting more PEs have a lower risk of 30-year CVD risk scores which is most evident in Supplementary Tables 3.8 and 3.9. In this analysis, those with 6 or more PEs have a lower risk compared to those with fewer PEs and there was a significant interaction between PEs and race suggesting Blacks with high PEs have a significantly lower risk of having a CVD event in the next 30 years compared to both Blacks with low PEs and Whites with both high and low PEs. This general effect was observed regardless of how the exposure was classified, where there was a lower risk among Blacks at higher levels of PEs compared to both Blacks with lower levels of PEs and Whites with both higher and lower levels of PEs (Supplementary Tables 3.7-3.14b). Finally, logistic regression analysis revealed a protective significant odds ratio when the outcome cut-points were set at a 10% risk level for both the hard and full CVD risk scores (Supplementary Table 3.16 and 3.17). Thus, the question may not be an ideal one to use as the exposure. Indeed, while this question was asked during the height of the War on Drugs, Terry Stops, peaked later.[545] Additionally, it may also not be the only, or even the most prominent, source of stress among those who are socially disadvantaged.[109,296,348,381] Additionally, although the response rate for Wave III was high, there may have been differential attrition due to those who are more likely to display a proclivity for delinquent behavior potentially being less likely to participate in research studies.[563]

Urban residents were not oversampled in Add Health, although urban residents may have higher exposure to the police.[86,149,441,471] In fact, there appeared to be over-sampling of smaller communities across the US with about 25% of the population residing in an Urban Area (>1500 population per km^2), while about 67% of the US population resides in an urban area according to the US Census. The Add Health participants were between 24 to 32 years old at Wave IV (when the outcome was measured), thus despite having a high level of overweight (mean BMI = 28.9 kg/m^2, Supplementary Table 3.21) and a high prevalence of cigarette smoking,[564] there is a relatively low prevalence of diabetes,[565] an average systolic blood pressure,[566] and a low prevalence of taking antihypertensive medications compared to the US population.[567] With this lower prevalence of major CVD risk factors, there are few participants who may have reached the level to place them at a higher risk for CVD. Additionally, the outcome was measured 6

years after the exposure, many life events can occur in such a long time period such as marriage, children, completing education, among others which may influence one's CVD risk. As noted earlier, there is very little missing data. However, data are likely not missing at random, but rather are missing for reasons related to the exposure. At minimum there was a loss of power, and potentially some selection bias, by excluding those with missing data. The sample size is still quite large, thus conducting imputation would likely not result in any change of the results, but rather improve standard errors.[541,542,568]

This study includes data on an individual level; however, respondents live, work, and play in larger communities. Omitting the influence of group-level community factors makes this study subject to the psychologistic fallacy in which individual-level outcomes are exclusively explained by individual-level characteristics.[569,570] I did, however, control for the population density in the community to account for urbanicity. Finally, the assumptions for the linear regression analysis were met; however, after examining some of the influential points, removing these records increased the beta coefficient in Model 3 for the full outcomes by about 2% and for the hard outcome by about 5%, but the general result and interpretation did not change in any way.

While there are limitations, there are also strengths. Mainly, this study uses Add Health data, a longitudinal and nationally representative sample of American adolescents as they develop into adults. Each wave of Add Health has a considerably high response rate (near or above 80%). Additionally, there is a very large sample size of Blacks and Whites. The exposure is known to occur before the outcome as it was measured in Wave III based on encounters occurring before Wave III and was not repeated in Wave IV, thus the outcome occurs approximately 6 or more years later. While the risk score is calculated using an algorithm, it is based on several systematically measured factors such as height and weight and based on a relatively younger population than those affected by CVD.[491]

3.5 Conclusions

There have been calls by researchers to understand how interactions between the police and community members affect health.[73,161,168,169] Prior research that focuses on police-related deaths ignores the effects of police encounters that are not violent and occur much more regularly. While stories of excessive police violence are reported nearly daily in the media, we do not have

a mechanism to gather data on routine PEs and thus cannot quantify the number of times PEs occur. Indeed, our current federal surveillance systems for capturing violence by the hands of police are woefully inadequate, such as death records, FBIs Uniform Crime Reporting, and CDC's injury surveillance.[160,169,473,571] In fact, the CDC reports that between 2015 and 2018, 2,208 deaths occurred due to legal intervention (ICD-10 codes Y35.0-Y35.4, Y35.6-Y35.7) based on death records.[161,572] However, this is thought to be a substantial undercounting of the number of deaths, only one part of the interaction between the public and the police. Even with this undercount, crowdsourced databases have estimated that approximately 5,9,48 deaths have occurred between 2015 and 2020 (between 2015 and 2018 approximately 3,928 deaths have occurred).[164] Although deaths due to police intervention are terrible, they are relatively rare. However, Terry stops, stops that are legally allowed due to *Terry V Ohio* (1968), are more common. In fact, they are so common that some cities have created surveillance mechanisms to quantify such stops.[86] For example, New York City has tracked about 65,000 PEs since 2015.[163] This represents only one city, thus supporting the commonness of this exposure and an area of future research.

This study is among the first to examine non-fatal encounters with the police and CVD risk. I observed that among Black respondents with high reported PEs the 30-year CVD risk was lower than those with lower reported PEs, while there was no difference between CVD risk among Whites. National estimates of brutal encounters are valuable for understanding the problem overall, but we do not fully understand the health toll of everyday PEs which are substantially more common in minority communities. This chapter sheds light on racial disparities in PEs and begins to address a major gap in our understanding of routine, non-fatal PEs, a call that has been noted by several prominent criminal justice health and health researchers.[160,165-169,173,473,571]

3.6 Tables and Figures

Table 3. 1 Descriptive Statistics by Race and Police Encounter (PE) Exposure Level at Add Health Wave III (n=8,447).

	Total n=8,447	Non-Hispanic Black n=2,268	Non-Hispanic White n=6,179	*P-value*[a]	High PEs[b] n=332	Low PEs[b] n=8,115	*P-value*[a]
Race (W1), n (%)							
Non-Hispanic Black	2,268 (17.3)	---	---	---	82 (16.5)	2,186 (17.3)	0.1824
Non-Hispanic White	6,179 (82.7)	---	---		250 (83.5)	5,929 (82.7)	
Age (W3), Years, mean (SE)	21.7 (0.13)	22.0 (0.22)	21.7 (0.14)	0.3361	21.3 (0.16)	21.8 (0.12)	0.0012
Sex (W1), n (%)							
Male	3,734 (48.4)	904 (46.0)	2,830 (48.9)	0.2283	149 (50.7)	3,585 (48.3)	0.5820
Female	4,713 (51.6)	1,364 (54.0)	3,349 (51.1)		183 (49.3)	4,530 (51.7)	
Educational Attainment (W3), n (%)							
Less than High School	967 (13.0)	285 (17.7)	682 (12.0)		65 (21.3)	902 (12.6)	
High School Graduate	6,135 (72.1)	1,684 (70.7)	4,451 (72.4)	0.0845	240 (73.8)	5,895 (72.0)	<0.0001
College Degree or Higher	1,345 (14.9)	299 (11.6)	1,046 (15.6)		27 (4.9)	1,318 (15.4)	
Household Income (W3), n (%)							
<$20,000	4,447 (53.0)	1,252 (59.7)	3,222 (51.6)		172 (55.4)	4,302 (52.9)	
$20,000-$39,999	1,759 (19.8)	425 (18.4)	1,334 (20.1)		74 (17.0)	1,685 (20.0)	
$40,000-$74,999	1,018 (12.8)	245 (9.6)	773 (13.4)	<0.0001	38 (11.8)	980 (12.8)	0.7826
≥$75,000	865 (11.0)	174 (5.3)	691 (12.1)		36 (12.0)	829 (10.9)	
Unknown (flag)	331 (3.4)	172 (6.9)	159 (2.7)		12 (3.9)	319 (3.4)	
Population Density (W3), n (%)							
<180.2 pop./Km^2	2,554 (31.7)	583 (30.8)	1,971 (31.8)		81 (26.1)	2,473 (31.9)	
180.2 - 1,027.6 pop./Km^2	2,355 (28.9)	484 (20.8)	1,891 (30.6)	0.0875	82 (22.7)	2,273 (20.0)	0.0007
1,027.7 - 2,630.4 pop./Km^2	2,060 (25.8)	538 (25.3)	1,522 (25.9)		80 (24.6)	1,980 (25.8)	
>2,630.4 pop./Km^2	1,478 (13.6)	683 (21.0)	795 (10.2)		89 (26.5)	1,389 (13.0)	
Early Socioeconomic Status (W3), n (%)							
Public Assist. before 18 yrs	1,312 (15.3)	514 (27.0)	798 (12.8)	0.0028	73 (21.9)	1,239 (15.0)	0.0262
No Public Asst.	7,135 (84.7)	1,754 (73.0)	5,381 (87.2)		259 (78.1)	6,876 (85.0)	
Exposure (W3), n (%)							
High Police Encounters (PEs)	332 (4.7)	82 (4.5)	250 (4.7)	0.8028	---	---	---
Low Police Encounters (PEs)	8,115 (95.3)	2,186 (95.5)	5,929 (95.3)		---	---	
Outcomes (W4), mean (SE)							
30-year CVD Risk Score (Full)[c]	0.125 (0.002)	0.139 (0.004)	0.121 (0.002)	0.0002	0.121 (0.005)	0.125 (0.002)	0.4073
30-year CVD Risk Score (Hard)[c]	0.069 (0.002)	0.080 (0.003)	0.066 (0.002)	<0.0001	0.065 (0.004)	0.069 (0.002)	0.3256

[a] *P-values* generated with chi-square for differences in weighted percents and t-tests for difference in weighted group means

[b] High PEs is 6 or more PEs among men, 2 or more for women. Low PEs is <6 PEs among men, <2 for women
[c] 30-Year cardiovascular disease risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke (hard) + coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure (full). The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.
P-values <0.05 are statistically significant. CI=confidence interval, SE=standard error

Table 3. 2 Linear Regression Coefficients for Regression of 30-Year Full Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on Police Encounters at Wave III (n=8,447).

	Log Transformed 30-year Cardiovascular Risk (Full)[a]								
	Model 1[b,c] (R^2=0.004364)			Model 2[b,d] (R^2=0.4043)			Model 3[b,e] (R^2=0.4054)		
	Beta	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-2.302	0.019	<0.0001	-5.005	0.107	<0.0001	-5.015	0.106	<0.0001
Police Encounters (W3)[f]									
High PEs	0.001	0.041	0.9781	0.004	0.034	0.9005	0.050	0.031	0.1092
Low PEs	Ref	---	---	Ref	--	--	Ref	--	--
Race (W1)									
Black	0.110	0.032	0.0008	0.088	0.017	<0.0001	0.100	0.018	<0.0001
White	Ref	--	--	Ref	--	--	Ref	--	--
Sex (W1)									
Male	--	--	--	0.651	0.016	<0.0001	0.652	0.156	<0.0001
Female	--	--	--	Ref	--	--	Ref	--	--
Age (W3), years	--	--	--	0.105	0.005	<0.0001	0.105	0.005	<0.0001
Early SES (W3)									
Public Assist. before 18 yrs	--	--	--	0.061	0.019	0.0018	0.064	0.019	0.0009
No Public Asst.	--	--	--	Ref	--	--	Ref	--	--
Education (W3)									
<High School	--	--	--	0.361	0.027	<0.0001	0.360	0.027	<0.0001
High School Grad	--	--	--	0.169	0.020	<0.0001	0.169	0.020	<0.0001
College Grad	--	--	--	Ref	--	--	Ref	--	--
Population Density (W3)									
<180.2 pop./Km^2	--	--	--	Ref	--	--	Ref	--	--
180.2 - 1,027.6 pop./Km^2	--	--	--	-0.053	0.023	0.0218	-0.052	0.023	0.0252
1,027.7 - 2,630.4 pop./Km^2	--	--	--	-0.025	0.020	0.2190	-0.025	0.020	0.2173
>2,630.4 pop./Km^2	--	--	--	-0.112	0.026	<0.0001	-0.111	0.026	<0.0001
Income (W3)									
<$20,000	--	--	--	-0.034	0.025	0.1842	-0.034	0.025	0.1860
$20,000-$39,999	--	--	--	-0.035	0.026	0.1822	-0.036	0.026	0.1765
$40,000-$74,999	--	--	--	-0.057	0.032	0.0780	-0.058	0.032	0.0742
>=$75,000	--	--	--	Ref	--	--	Ref	--	--
Missing Flag	--	--	--	-0.040	0.047	0.3974	-0.037	0.045	0.4097
High Police PEs*Black	--	--	--	--	--	--	-0.276	0.099	0.0061

[a] 30-Year cardiovascular disease (full) risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke, coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for detailed methods.

[b] Beta coefficients are generated using a generalized least-squared regression analysis incorporating Add Health's complex research design.

[c] Model 1 includes police encounters (PEs) and race.
[d] Model 2 includes the PEs, race, sex, age, early socioeconomic status (SES), education level, population density, and income
[e] Model 3 includes the variables in the Model 2 plus an interaction between race and PEs.
[f] High PEs is 6 or more PEs among men, 2 or more for women. Low PEs is <6 PEs among men, <2 for women
P-values <0.05 are statistically significant. CI=confidence interval, SE=standard error, W1=Wave I, W3=Wave III

Table 3. 3 Linear Regression Coefficients for Regression of 30-Year Hard Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on Police Encounters (PEs) at Wave III (n=8,447).

	Log Transformed 30-year Cardiovascular Risk (Hard)[a]								
	Model 1[b,c] (R^2=0.004227)			Model 2[b,d] (R^2=0.4507)			Model 3[b,e] (R^2=0.4519)		
	Beta	**SE**	***P-value***	**Beta[b]**	**SE**	***P-value***	**Beta[b]**	**SE**	***P-value***
Intercept	-3.001	0.022	<0.0001	-6.178	0.124	<0.0001	-6.190	0.123	<0.0001
Police Encounters (W3)[f]									
High PEs	0.010	0.049	0.8329	0.010	0.040	0.8136	0.065	0.036	0.0771
Low PEs	Ref	--	--	Ref	--	--	Ref	--	--
Race (W1)									
Black	0.132	0.039	0.0010	0.109	0.020	<0.0001	0.124	0.021	<0.0001
White	Ref	--	--	Ref	--	--	Ref	--	--
Sex (W1)									
Male	--	--	--	0.870	0.019	<0.0001	0.871	0.018	<0.0001
Female	--	--	--	Ref	--	--	Ref	--	--
Age (W3), years	--	--	--	0.121	0.006	<0.0001	0.121	0.005	<0.0001
Early SES (W3)									
Public Assist. before 18 yrs	--	--	--	0.070	0.023	0.0023	0.074	0.023	0.0013
No Public Asst.	--	--	--	Ref	--	--	Ref	--	--
Education (W3)									
<High School	--	--	--	0.433	0.032	<0.0001	0.432	0.031	<0.0001
High School Grad	--	--	--	0.201	0.024	<0.0001	0.200	0.024	<0.0001
College Grad	--	--	--	Ref	--	--	Ref	--	--
Population Density (W3)									
<180.2 pop./Km^2	--	--	--	Ref	--	--	Ref	--	--
180.2 - 1,027.6 pop./Km^2				-0.059	0.026	0.0262	-0.058	0.027	0.0304
1,027.7 - 2,630.4 pop./Km^2	--	--	--	-0.026	0.023	0.2595	-0.027	0.023	0.2576
>2,630.4 pop./Km^2	--	--	--	-0.128	0.031	<0.0001	-0.127	0.031	<0.0001
Income (W3)									
<$20,000	--	--	--	-0.039	0.030	0.1887	-0.039	0.030	0.1905
$20,000-$39,999	--	--	--	-0.038	0.031	0.2211	-0.039	0.031	0.2141
$40,000-$74,999	--	--	--	-0.067	0.038	0.0786	-0.068	0.038	0.0748
>=$75,000	--	--	--	Ref	--	--	Ref	--	--
Missing Flag	--	--	--	-0.049	0.054	0.3689	-0.045	0.051	0.3805
High PEs*Black	--	--	--	--	--	--	-0.334	0.115	0.0044

[a] 30-Year cardiovascular disease (hard) risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for detailed methods.
[b] Beta coefficients are generated using a generalized least-squared regression analysis incorporating Add Health's complex research design.
[c] Model 1 includes police encounters (PEs) and race.
[d] Model 2 includes the PEs, race, sex, age, early socioeconomic status (SES), education level, population density, and income
[e] Model 3 includes the variables in the Model 2 plus an interaction between race and PEs.
[f] High PEs is 6 or more PEs among men, 2 or more for women. Low PEs is <6 PEs among men, <2 for women.
P-values <0.05 are statistically significant. CI=confidence interval, SE=standard error

Table 3. 4 Adjusted Mean 30-Year Cardiovascular Risk Scores (Full and Hard Outcomes) at Add Health Wave IV by Police Encounter Status at Wave III Stratified by Race (n=8,447).

	High Police Encounters Reported[a] N = 332		Low Police Encounters Reported[a] N = 8,115		Black High-Low Difference[b] 95% CI *P-value*	White High-Low Difference[b] 95% CI *P-value*
	Non-Hispanic Black N = 82	Non-Hispanic White N = 250	Non-Hispanic Black N = 2,186	Non-Hispanic White N = 5,929		
Log 30-year Cardiovascular Risk Score – Full (95% CI)[f]						
Model 1[c]	-2.241 (-2.443,-2.040)	-2.291 (-2.391,-2.191)	-2.190 (-2.246,-2.134)	-2.303 (-2.340,-2.265)	-0.052 (-0.243,0.140)	0.012 (-0.080,0.103)
anti-log[e]	0.106	0.101	0.112	0.100	p=0.5952	p=0.8021
Model 2[d]	-2.379 (-2.555,-2.203)	-2.200 (-2.268,-2.132)	-2.174 (-2.240,-2.108)	-2.251 (-2.287,-2.215)	-0.205 (-0.374,-0.036)	0.051 (-0.011,0.112)
anti-log[e]	0.093	0.111	0.114	0.105	p=0.0178	p=0.1074
Log 30-year Cardiovascular Risk Score - Hard (95% CI)[f]						
Model 1[c]	-2.910 (-3.144,-2.675)	-2.981 (-3.102,-2.860)	-2.867 (-2.936,-2.799)	-3.002 (-3.046,-2.958)	-0.042 (-0.265,0.180)	0.021 (-0.091,0.133)
anti-log[e]	0.054	0.051	0.057	0.050	p=0.7071	p=0.7120
Model 2[d]	-3.091 (-3.297,-2.885)	-2.875 (-2.955,-2.796)	-2.842 (-2.920,-2.763)	-2.941 (-2.982,-2.899)	-0.249 (-0.449,-0.049)	0.065 (-0.007,0.138)
anti-log[e]	0.045	0.056	0.058	0.053	p=0.0151	p=0.0772

[a] High PEs is 6 or more PEs among men, 2 or more for women. Low PEs is <6 PEs among men, <2 for women.
[b] Negative differences are interpreted as higher CVD risk in the low PE group.
[c] Model 1 includes police encounters (PEs).
[d] Model 2 includes the PEs, sex, age, early socioeconomic status (SES), education level, population density, and income
[e] Anti-log is the exponent of the log-transformed mean risk score. It is interpreted as a geometric mean proportion, ie., 0.106=10.6% risk.
[f] 30-Year cardiovascular disease risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke (hard) + coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure (full). The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for detailed methods. *P-values* <0.05 are statistically significant, CI=confidence interval.

Table 3. 5 Adjusted Mean 30-Year Cardiovascular Risk Scores (Full and Hard Outcomes) at Add Health Wave IV by Police Encounter Status at Wave III Stratified by Sex (n=8,447).

	High Police Encounters Reported[a] N = 332		Low Police Encounters Reported[a] N = 8,115		Male High-Low Difference[b] 95% CI *P-value*	Female High-Low Difference[b] 95% CI *P-value*
	Male N = 149	Female N = 183	Male N = 3,585	Female N = 4,530		
Log 30-year Cardiovascular Risk Score - Full (95% CI)[g]						
Model 1[c]	-2.013 (-2.135, -1.891)	-2.529 (-2.621, -2.437)	-1.917 (-1.950, -1.885)	-2.600 (-2.640, -2.560)	-0.096 (-0.212, 0.020)	0.071 (-0.019, 0.161)
anti-log[f]	0.134	0.080	0.147	0.074	p=0.1033	p=0.1196
Model 2[d]	-1.977 (-2.091, -1.864)	-2.432 (-2.514, -2.350)	-1.883 (-1.926, -1.840)	-2.542 (-2.587, -2.497)	-0.095 (-0.190, 0.001)	0.110 (0.030, 0.189)
anti-log[f]	0.138	0.088	0.152	0.079	p=0.0520	p=0.0070
Model 3[e]	-2.041 (-2.170, -1.912)	-2.516 (-2.662, -2.371)	-1.875 (-1.915, -1.836)	-2.540 (-2.585, -2.494)	-0.166 (-0.282, -0.049)	0.023 (-0.124, 0.171)
anti-log[f]	0.130	0.081	0.153	0.079	p=0.0057	p=0.7549
Log 30-year Cardiovascular Risk Score - Hard (95% CI)[g]						
Model 1[c]	-2.602 (-2.746, -2.458)	-3.310 (-3.417, -3.204)	-2.493 (-2.532, -2.455)	-3.402 (-3.448, -3.356)	-0.109 (-0.246, 0.028)	0.092 (-0.013, 0.196)
anti-log[f]	0.074	0.037	0.083	0.033	p=0.1185	p=0.0843
Model 2[d]	-2.559 (-2.693, -2.424)	-3.199 (-3.295, -3.103)	-2.449 (-2.500, -2.397)	-3.334 (-3.387, -3.281)	-0.110 (-0.224, 0.004)	0.135 (0.042, 0.228)
anti-log[f]	0.077	0.041	0.086	0.036	p=0.0588	p=0.0047
Model 3[e]	-2.640 (-2.788, -2.492)	-3.295 (-3.469, -3.121)	-2.439 (-2.487, -2.391)	-3.331 (-3.385, -3.278)	-0.201 (-0.335, -0.066)	0.037 (-0.140, 0.213)
anti-log[f]	0.071	0.037	0.087	0.036	p=0.0037	p=0.6814

[a] High PEs is 6 or more PEs among men, 2 or more for women. Low PEs is <6 PEs among men, <2 for women.
[b] Negative differences are interpreted as higher CVD risk in the low PE group.
[c] Model 1 includes police encounters (PEs) and race.
[d] Model 2 includes the PEs, race, age, early socioeconomic status (SES), education level, population density, and income
[e] Model 3 includes the variables in the Model 2 plus an interaction between race and PEs.
[f] Anti-log is the exponent of the log-transformed mean risk score. It is interpreted as a geometric mean proportion, ie., 0.134=13.4% risk.
[g] 30-Year cardiovascular disease risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke (hard) + coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure (full). The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods. *P-values* <0.05 are statistically significant, CI=confidence interval.

Table 3. 6 Adjusted Mean 30-Year Cardiovascular Risk Scores (Full and Hard Outcomes) at Add Health Wave IV by Police Encounter Status at Wave III Stratified by Race and Sex (n=8,447).

	High Police Encounters Reported[a] N = 322				Low Police Encounters Reported[a] N = 8,115			
	Non-Hispanic Black		Non-Hispanic White		Non-Hispanic Black		Non-Hispanic White	
	Male	Female	Male	Female	Male	Female	Male	Female
	N = 46	N = 36	N = 103	N = 147	N = 858	N = 1,328	N = 2,727	N = 3,202
Log 30-year Cardiovascular Risk Score – Full (95% CI)[b]								
Model 1[c]	-2.072 (-2.336,-1.809)	-2.560 (-2.864, -2.257)	-2.001 (-2.134,-1.868)	-2.556 (-2.651, -2.461)	-1.832 (-1.889, -1.775)	-2.484 (-2.540, -2.427)	-1.944 (-1.979, -1.908)	-2.647 (-2.694,-2.600)
anti-log[e]	0.126	0.077	0.135	0.078	0.160	0.083	0.143	0.071
Model 2[d]	-2.111 (-2.328, -1.893)	-2.584 (-2.876, -2.291)	-1.952 (-2.056, -1.848)	-2.455 (-2.537, -2.372)	-1.838 (-1.922, -1.754)	-2.502 (-2.582, -2.423)	-1.908 (-1.950, -1.865)	-2.591 (-2.643, -2.539)
anti-log[e]	0.121	0.075	0.142	0.086	0.159	0.082	0.148	0.075
Differences[f] (High –Low)		**Difference**	***Model 1[c]* 95% CI**	***P-value***		**Difference**	***Model 2[d]* 95% CI**	***P-value***
Black Males		-0.241	(-0.496, 0.015)	0.0649		-0.273	(-0.476, -0.070)	0.0089
Black Females		-0.077	(-0.376, 0.222)	0.6116		-0.081	(-0.368, 0.206)	0.5756
White Males		-0.057	(-0.182, 0.067)	0.3636		-0.044	(-0.139, 0.051)	0.3590
White Females		0.091	(-0.003, 0.185)	0.0566		0.137	(0.057, 0.216)	0.0009
Log 30-year Cardiovascular Risk Score – Hard (95% CI)[b]								
Model 1[c]	-2.679 (-2.982, -2.377)	-3.344 (-3.700, -3.989)	-2.586 (-2.740, -2.432)	-3.341 (-3.452, -3.231)	-2.378 (-2.447, -2.310)	-3.269 (-3.335, -3.202)	-2.528 (-2.570, -2.487)	-3.456 (-3.510, -3.402)
anti-log[e]	0.069	0.035	0.075	0.035	0.093	0.038	0.080	0.032
Model 2[d]	-2.731 (-2.980, -2.482)	-3.371 (-3.722, -3.021)	-2.533 (-2.656, -2.410)	-3.225 (-3.321, -3.129)	-2.388 (-2.490, -2.287)	-3.288 (-3.381, -3.194)	-2.487 (-2.538,-2.437)	-3.390 (-3.451, -3.330)
anti-log[e]	0.065	0.034	0.079	0.040	0.092	0.037	0.083	0.034
Differences[f] (High –Low)		**Difference**	***Model 1[c]* 95% CI**	***P-value***		**Difference**	***Model 2[d]* 95% CI**	***P-value***

Black Males	-0.301	(-0.595, -0.008)	0.0444	-0.343	(-0.578, -0.108)	0.0045
Black Females	-0.076	(-0.427, 0.275)	0.6699	-0.083	(-0.429, 0.262)	0.6336
White Males	-0.058	(-0.203, 0.088)	0.4340	-0.046	(-0.158, 0.066)	0.4209
White Females	0.114	(0.006, 0.223)	0.0394	0.165	(0.073, 0.258)	0.0006

[a] High PEs is 6 or more PEs among men, 2 or more for women. Low PEs is <6 PEs among men, <2 for women.
[b] 30-Year cardiovascular disease risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke (hard) + coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure (full). The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.
[c] Model 1 includes police encounters (PEs).
[d] Model 2 includes the PEs, age, early socioeconomic status (SES), education level, population density, and income
[e] Anti-log is the exponent of the log-transformed mean risk score. It is interpreted as a geometric mean proportion, ie., 0.077=7.7% risk.
[f] Negative differences are interpreted as higher CVD risk in the reference group.
P-values <0.05 are statistically significant, CI=confidence interval.

Supplementary Table 3. 1 Adjusted Mean 30-Year Cardiovascular Risk Scores (Full and Hard Outcomes) at Add Health Wave IV by Police Encounter Status (6 or more PEs vs <6 PEs) at Wave III Stratified by Race and Sex

	>=6 Police Stops N = 160				<6 Police Stops N = 8,287			
	Non-Hispanic Black		Non-Hispanic White		Non-Hispanic Black		Non-Hispanic White	
	Male N = 46	Female N = 4	Male N = 103	Female N = 7	Male N = 858	Female N = 1,360	Male N = 2,727	Female N = 3,342
30-year Cardiovascular Risk Score – Full (95% CI)[a]								
Model 1[b]	-2.072 (-2.336,-1.809)	-2.225 (-2.853,-1.597)	-2.001 (-2.134,-1.868)	-2.750 (-2.894,-2.605)	-1.832 (-1.899,-1.775)	-2.486 (-2.543,-2.428)	-1.944 (-1.979,-1.908)	-2.642 (-2.688,-2.597)
Anti-log[d]	0.126	0.108	0.135	0.064	0.160	0.083	0.143	0.071
Model 2[c]	-2.111 (-2.328,-1.893)	-2.133 (-2.783,-1.484)	-1.952 (-2.056,-1.848)	-2.610 (-2.857,-2.363)	-1.838 (-1.922,-1.754)	-2.505 (-2.584,-2.426)	-1.908 (-1.950,-1.865)	-2.582 (-2.633,-2.532)
Anti-log[d]	0.121	0.118	0.142	0.074	0.159	0.082	0.148	0.076
Differences 6 + PEs - <6 PEs			**Difference[e]**	**Model 1 95% CI**	***P-value***	**Difference[e]**	**Model 2 95% CI**	***P-value***
Black Males			-0.241	(-0.496, 0.015)	0.0649	-0.273	(-0.476, -0.070)	0.0089
Black Females			0.261	(-0.368, 0.890)	0.4129	0.372	(-0.275, 1.018)	0.2576
White Males			-0.057	(-0.182, 0.067)	0.3636	-0.044	(-0.139, 0.051)	0.3590
White Females			-0.107	(-0.260, 0.046)	0.1674	-0.028	(-0.278, 0.223)	0.8275
	Non-Hispanic Black		**Non-Hispanic White**		**Non-Hispanic Black**		**Non-Hispanic White**	
	Male	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**
30-year Cardiovascular Risk Score – Hard (95% CI)[a]								
Model 1[b]	-2.679 (-2.982,-2.377)	-2.952 (-3.718,-2.187)	-2.586 (-2.740,-2.432)	-3.574 (-3.749,-3.400)	-2.378 (-2.447,-2.310)	-3.271 (-3.337,-3.204)	-2.528 (-2.570,-2.487)	-3.450 (-3.503,-3.398)
Anti-log[d]	0.069	0.052	0.075	0.028	0.093	0.038	0.080	0.032
Model 2[c]	-2.731 (-2.980,-2.482)	-2.843 (-3.635,-2.051)	-2.533 (-2.656,-2.410)	-3.416 (-3.707,-3.125)	-2.388 (-2.490,-2.287)	-3.291 (-3.384,-3.198)	-2.487 (-2.538,-2.437)	-3.380 (-3.439,-3.321)
Anti-log[d]	0.065	0.058	0.079	0.033	0.092	0.037	0.083	0.034
Differences 6 + PEs - <6 PEs			**Difference[e]**	**Model 1 95% CI**	***P-value***	**Difference[e]**	**Model 2 95% CI**	***P-value***
Black Males			-0.301	(-0.595, -0.008)	0.0444	-0.343	(-0.578, -0.108)	0.0045
Black Females			0.318	(-0.447, 1.084)	0.4120	0.448	(-0.314, 1.237)	0.2637
White Males			-0.058	(-0.203, 0.088)	0.4340	-0.046	(-0.158, 0.066)	0.4209
White Females			-0.124	(-0.308, 0.060)	0.1852	-0.036	(-0.330, 0.259)	0.8101

(n=8,447).

[a] 30-Year cardiovascular disease risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke (Hard) + coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure (Full). The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.
[b] Model 1 includes police encounters (PEs).
[c] Model 2 includes the PEs, age, early socioeconomic status (SES), education level, population density, and income
[d] Anti-log is the exponent of the log-transformed mean risk score. It is interpreted as a geometric mean proportion, ie., 0.077=7.7% risk.
[e] Negative differences are interpreted as higher CVD risk in the reference group.
P-values <0.05 are statistically significant, CI=confidence interval.

Supplementary Table 3. 2 Linear Regression Coefficients for Regression of 30-Year Full Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on Police Encounters (PEs) (Never vs. 1 or More) at Wave III (n=8,447).

	Log Transformed 30-year Cardiovascular Risk (Full)[a]								
	Model 1[b,c] (R^2=0.02065)			Model 2 [b,d] (R^2=0.4044)			Model 3 [b,e] (R^2=0.4050)		
	Beta	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-2.344	0.020	<0.0001	-5.014	0.106	<0.0001	-5.026	0.105	<0.0001
Police Encounters (W3)									
1 or More PEs	0.198	0.026	<0.0001	0.021	0.018	0.2489	0.037	0.019	0.0505
Never	Ref	--	--	Ref	--	--	Ref	--	--
Race (W1)									
Black	0.117	0.032	0.0004	0.089	0.017	<0.0001	0.108	0.020	<0.0001
White	Ref	--	--	Ref	--	--	Ref	--	--
Sex (W1)									
Male	--	--	--	0.646	0.016	<0.0001	0.648	0.016	<0.0001
Female	--	--	--	Ref	--	--	Ref	--	--
Age (W3), years	--	--	--	0.105	0.005	<0.0001	0.106	0.005	<0.0001
Early SES (W3)									
Public Assist. before 18 yrs	--	--	--	0.060	0.019	0.0023	0.062	0.020	0.0020
No Public Asst.	--	--	--	Ref	--	--	Ref	--	--
Education (W3)									
<High School	--	--	--	0.360	0.027	<0.0001	0.361	0.027	<0.0001
High School Grad	--	--	--	0.169	0.020	<0.0001	0.169	0.020	<0.0001
College Grad	--	--	--	Ref	--	--	Ref	--	--
Population Density (W3)									
<180.2 pop./Km^2	--	--	--	Ref	---	---	Ref	--	--
180.2 - 1,027.6 pop./Km^2	--	--	--	-0.053	0.023	0.0220	-0.052	0.023	0.0248
1,027.7 - 2,630.4 pop./Km^2	--	--	--	-0.026	0.020	0.2020	-0.026	0.020	0.1997
>2,630.4 pop./Km^2	--	--	--	-0.114	0.026	<0.0001	Ref	--	--
Income (W3)									
<$20,000	--	--	--	-0.033	0.026	0.1984	-0.032	0.025	0.2093
$20,000-$39,999	--	--	--	-0.034	0.026	0.1956	-0.330	0.027	0.2106
$40,000-$74,999	--	--	--	-0.056	0.032	0.0834	-0.055	0.032	0.0858
>=$75,000	--	--	--	Ref	--	--	Ref	--	--
Missing Flag	--	--	--	-0.038	0.047	0.4284	-0.036	0.047	0.4418
1 or More PEs*Black	--	--	--	--	--	--	-0.105	0.044	0.0202

[a] 30-Year cardiovascular disease (full) risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke, coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[b] Beta coefficients are generated using a generalized least-squared regression analysis incorporating Add Health's complex research design.
[c] Model 1 includes police encounters (PEs) and race.
[d] Model 2 includes the PEs, race, sex, age, early socioeconomic status (SES), education level, population density, and income
[e] Model 3 includes the variables in the Model 2 plus an interaction between race and PEs.
P-values <0.05 are statistically significant. CI=confidence interval, SE=standard error

Supplementary Table 3. 3 Linear Regression Coefficients for Regression of 30-Year Hard Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on Police Encounters (PEs) (Never vs 1 or more) at Wave III (n=8,447).

	Log Transformed 30-year Cardiovascular Risk (Hard)[a]								
	Model 1[b,c] (R^2=0.02439)			Model 2[b,d] (R^2=0.4509)			Model 3[b,e] (R^2=0.4515)		
	Beta	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-3.058	0.024	<0.0001	-6.189	0.124	<0.0001	-6.203	0.122	<0.0001
Police Encounters (W3)									
1 or More PEs	0.268	0.032	<0.0001	0.029	0.022	0.1838	0.047	0.022	0.0329
Never	Ref	--	--	Ref	--	--	Ref	--	--
Race (W1)									
Black	0.141	0.389	0.0004	0.110	0.020	<0.0001	0.132	0.024	<0.0001
White	Ref	--	--	Ref	--	--	Ref	--	--
Sex (W1)									
Male	--	--	--	0.864	0.019	<0.0001	0.866	0.019	<0.0001
Female	--	--	--	Ref	--	--	Ref	--	--
Age (W3), years	--	--	--	0.121	0.005	<0.0001	0.121	0.005	<0.0001
Early SES (W3)									
Public Assist. before 18 yrs	--	--	--	0.069	0.023	0.0031	0.071	0.023	0.0028
No Public Asst.	--	--	--	Ref	--	--	Ref	--	--
Education (W3)									
<High School	--	--	--	0.432	0.032	<0.0001	0.433	0.031	<0.0001
High School Grad	--	--	--	0.201	0.023	<0.0001	0.201	0.023	<0.0001
College Grad	--	--	--	Ref	--	--	Ref	--	--
Population Density (W3)									
<180.2 pop./Km^2	--	--	--	Ref	---	---	Ref	--	--
180.2 - 1,027.6 pop./Km^2	--	--	--	-0.059	0.026	0.0264	-0.058	0.027	0.0298
1,027.7 - 2,630.4 pop./Km^2	--	--	--	-0.280	0.024	0.2365	-0.028	0.023	0.2342
>2,630.4 pop./Km^2	--	--	--	-0.130	0.031	<0.0001	-0.128	0.031	<0.0001
Income (W3)									
<$20,000	--	--	--	-0.038	0.030	0.2048	-0.037	0.030	0.2158
$20,000-$39,999	--	--	--	-0.037	0.031	0.2377	-0.036	0.031	0.2544
$40,000-$74,999	--	--	--	-0.066	0.038	0.0846	-0.065	0.038	0.0871
>=$75,000	--	--	--	Ref	--	--	Ref	--	--
Missing Flag	--	--	--	-0.046	0.055	0.4036	-0.044	0.053	0.4159
1 or More PEs*Black	--	--	--	--	--	--	-0.120	0.055	0.0304

[a] 30-Year cardiovascular disease (hard) risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[b] Beta coefficients are generated using a generalized least-squared regression analysis incorporating Add Health's complex research design.

[c] Model 1 includes police encounters (PEs) and race.
[d] Model 2 includes the PEs, race, sex, age, early socioeconomic status (SES), education level, population density, and income
[e] Model 3 includes the variables in the Model 2 plus an interaction between race and PEs.
P-values <0.05 are statistically significant. CI=confidence interval, SE=standard error

Supplementary Table 3. 4 Linear Regression Coefficients for Regression of 30-Year Full Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on Police Encounters (PEs) (<2 vs 2 or more) at Wave III (n=8,447).

	Log Transformed 30-year Cardiovascular Risk (Full)[a]								
	Model 1[b,c] (R^2=0.02229)			Model 2[b,d] (R^2=0.4045)			Model 3[b,e] (R^2=0.4050)		
	Beta	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-2.332	0.020	<0.0001	-5.010	0.107	<0.0001	-5.019	0.106	<0.0001
Police Encounters (W3)									
2 or More PEs	0.264	0.027	<0.0001	0.032	0.023	0.1651	0.052	0.023	0.0235
<2 PEs	Ref	--	--	Ref	--	--	Ref	--	--
Race (W1)									
Black	0.110	0.032	0.0008	0.089	0.017	<0.0001	0.101	0.020	<0.0001
White	Ref	--	--	Ref	--	--	Ref	--	--
Sex (W1)									
Male	--	--	--	0.646	0.016	<0.0001	0.647	0.015	<0.0001
Female	--	--	--	Ref	--	--	Ref	--	--
Age (W3), years	--	--	--	0.105	0.005	<0.0001	0.105	0.005	<0.0001
Early SES (W3)									
Public Assist. before 18 yrs	--	--	--	0.059	0.019	0.0024	0.061	0.019	0.0019
No Public Asst.	--	--	--	Ref	--	--	Ref	--	--
Education (W3)									
<High School	--	--	--	0.360	0.028	<0.0001	0.359	0.027	<0.0001
High School Grad	--	--	--	0.169	0.020	<0.0001	0.168	0.020	<0.0001
College Grad	--	--	--	Ref	--	--	Ref	--	--
Population Density (W3)									
<180.2 pop./Km^2	--	--	--	Ref	--	--	Ref	--	--
180.2 - 1,027.6 pop./Km^2	--	--	--	-0.052	0.023	0.0225	-0.051	0.023	0.0261
1,027.7 - 2,630.4 pop./Km^2				-0.026	0.020	0.2017	-0.026	0.020	0.2048
>2,630.4 pop./Km^2	--	--	--	-0.114	0.026	<0.0001	-0.113	0.026	<0.0001
Income (W3)									
<$20,000	--	--	--	-0.033	0.026	0.1922	-0.033	0.025	0.1938
$20,000-$39,999	--	--	--	-0.035	0.026	0.1817	-0.036	0.026	0.1782
$40,000-$74,999	--	--	--	-0.056	0.032	0.0814	-0.057	0.032	0.0790
>=$75,000	--	--	--	Ref	--	--	Ref	--	--
Missing Flag	--	--	--	-0.038	0.048	0.4222	-0.037	0.046	0.4297
2 or More PEs*Black	--	--	--	--	--	--	-0.117	0.065	0.0760

[a] 30-Year cardiovascular disease (full) risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke, coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[b] Beta coefficients are generated using a generalized least-squared regression analysis incorporating Add Health's complex research design.
[c] Model 1 includes police encounters (PEs) and race.
[d] Model 2 includes the PEs, race, sex, age, early socioeconomic status (SES), education level, population density, and income
[e] Model 3 includes the variables in the Model 2 plus an interaction between race and PEs.
P-values <0.05 are statistically significant. CI=confidence interval, SE=standard error

Supplementary Table 3. 5 Linear Regression Coefficients for Regression of 30-Year Hard Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on Police Encounters (PEs) (<2 vs 2 or more) at Wave III (n=8,447).

	Log Transformed 30-year Cardiovascular Risk (Hard)[a]								
	Model 1[b,c] (R^2=0.02582)			Model 2[b,d] (R^2=0.4510)			Model 3[b,e] (R^2=0.4514)		
	Beta	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-3.042	0.023	<0.0001	-6.185	0.124	<0.0001	-6.195	0.123	<0.0001
Police Encounters (W3)									
2 or More PEs	0.353	0.033	<0.0001	0.042	0.027	0.1302	0.064	0.027	0.0168
<2 PEs	Ref	--	--	Ref	--	--	Ref	--	--
Race (W1)									
Black	0.132	0.039	0.0010	0.110	0.020	<0.0001	0.125	0.024	<0.0001
White	Ref	--	--	Ref	--	--	Ref	--	--
Sex (W1)									
Male	--	--	--	0.864	0.018	<0.0001	0.865	0.018	<0.0001
Female	--	--	--	Ref	--	--	Ref	--	--
Age (W3), years	--	--	--	0.121	0.005	<0.0001	0.121	0.005	<0.0001
Early SES (W3)									
Public Assist. before 18 yrs	--	--	--	0.068	0.023	0.0032	0.070	0.023	0.0027
No Public Asst.	--	--	--	Ref	--	--	Ref	--	--
Education (W3)									
<High School	--	--	--	0.432	0.032	<0.0001	0.431	0.032	<0.0001
High School Grad	--	--	--	0.200	0.024	<0.0001	0.200	0.024	<0.0001
College Grad	--	--	--	Ref	--	--	Ref	--	--
Population Density (W3)									
<180.2 pop./Km^2	--	--	--	Ref	--	--	Ref	--	--
180.2 - 1,027.6 pop./Km^2	--	--	--	-0.059	0.026	0.0271	-0.058	0.027	0.0314
1,027.7 - 2,630.4 pop./Km^2	--	--	--	-0.028	0.023	0.2382	-0.028	0.023	0.2418
>2,630.4 pop./Km^2	--	--	--	-0.130	0.031	<0.0001	-0.128	0.031	<0.0001
Income (W3)									
<$20,000	--	--	--	-0.039	0.030	0.1972	-0.039	0.030	0.1989
$20,000-$39,999	--	--	--	-0.038	0.031	0.2197	-0.038	0.031	0.2159
$40,000-$74,999	--	--	--	-0.066	0.038	0.0822	-0.067	0.038	0.0799
>=$75,000	--	--	--	Ref	--	--	Ref	--	--
Missing Flag	--	--	--	-0.047	0.055	0.3950	-0.045	0.053	0.4019
2 or More PEs*Black	--	--	--	--	--	--	-0.134	0.079	0.0916

[a] 30-Year cardiovascular disease (hard) risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[b]Beta coefficients are generated using a generalized least-squared regression analysis incorporating Add Health's complex research design.

[c] Model 1 includes police encounters (PEs) and race.
[d] Model 2 includes the PEs, race, sex, age, early socioeconomic status (SES), education level, population density, and income
[e] Model 3 includes the variables in the Model 2 plus an interaction between race and PEs.
P-values <0.05 are statistically significant. CI=confidence interval, SE=standard error

Supplementary Table 3. 6 Linear Regression Coefficients for Regression of 30-Year Full Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on Police Encounters (PEs) (<4 vs 4 or more) at Wave III (n=8,447).

	Log Transformed 30-year Cardiovascular Risk (Full)[a]								
	Model 1[b,c] (R^2=0.01396)			Model 2[b,d] (R^2=0.4044)			Model 3[b,e] (R^2=0.4047)		
	Beta	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-2.315	0.019	<0.0001	-5.004	0.106	<0.0001	-5.009	0.105	<0.0001
Police Encounters (W3)									
4 or More PEs	0.295	0.042	<0.0001	-0.030	0.037	0.4142	-0.004	0.034	0.9063
<4 PEs	Ref	--	--	Ref	--	--	Ref	--	--
Race (W1)									
Black	0.109	0.032	0.0009	0.088	0.017	<0.0001	0.095	0.018	<0.0001
White	Ref	--	--	Ref	--	--	Ref	--	--
Sex (W1)									
Male	--	--	--	0.653	0.016	<0.0001	0.653	0.015	<0.0001
Female	--	--	--	Ref	--	--	Ref	--	--
Age (W3), years	--	--	--	0.105	0.005	<0.0001	0.105	0.005	<0.0001
Early SES (W3)									
Public Assist. before 18 yrs	--	--	--	0.062	0.019	0.0014	0.063	0.019	0.0012
No Public Asst.	--	--	--	Ref	--	--	Ref	--	--
Education (W3)									
<High School	--	--	--	0.363	0.028	<0.0001	0.363	0.027	<0.0001
High School Grad	--	--	--	0.170	0.020	<0.0001	0.170	0.020	<0.0001
College Grad	--	--	--	Ref	--	--	Ref	--	--
Population Density (W3)									
<180.2 pop./Km^2	--	--	--	Ref	--	--	Ref	--	--
180.2 - 1,027.6 pop./Km^2	--	--	--	-0.053	0.023	0.0216	-0.052	0.023	0.0240
1,027.7 - 2,630.4 pop./Km^2				-0.025	0.020	0.2223	-0.024	0.020	0.2273
>2,630.4 pop./Km^2	--	--	--	-0.111	0.026	<0.0001	-0.109	0.026	<0.0001
Income (W3)									
<$20,000	--	--	--	-0.034	0.025	0.1803	-0.034	0.025	0.1831
$20,000-$39,999	--	--	--	-0.036	0.026	0.1786	-0.036	0.026	0.1766
$40,000-$74,999	--	--	--	-0.057	0.032	0.0752	-0.057	0.032	0.0750
>=$75,000	--	--	--	Ref	--	--	Ref	--	--
Missing Flag	--	--	--	-0.040	0.047	0.3907	-0.038	0.045	0.4037
4 or More PEs*Black	--	--	--	--	--	--	-0.147	0.102	0.1502

[a] 30-Year cardiovascular disease (full) risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke, coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[b] Beta coefficients are generated using a generalized least-squared regression analysis incorporating Add Health's complex research design.
[c] Model 1 includes police encounters (PEs) and race.
[d] Model 2 includes the PEs, race, sex, age, early socioeconomic status (SES), education level, population density, and income
[e] Model 3 includes the variables in the Model 2 plus an interaction between race and PEs.
P-values <0.05 are statistically significant. CI=confidence interval, SE=standard error

Supplementary Table 3. 7 Linear Regression Coefficients for Regression of 30-Year Hard Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on Police Encounters (PEs) (<4 vs 4 or more) at Wave III (n=8,447).

	Log Transformed 30-year Cardiovascular Risk (Hard)[a]								
	Model 1[b,c] (R^2=0.01602)			Model 2[b,d] (R^2=0.4508)			Model 3[b,e] (R^2=0.4512)		
	Beta	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-3.019	0.023	<0.0001	-6.176	0.123	<0.0001	-6.182	0.122	<0.0001
Police Encounters (W3)									
4 or More PEs	0.398	0.050	<0.0001	-0.036	0.043	0.4112	-0.033	0.039	0.9316
<4 PEs	Ref	--	--	Ref	--	--	Ref	--	--
Race (W1)									
Black	0.131	0.039	0.0011	0.109	0.020	<0.0001	0.117	0.021	<0.0001
White	Ref	--	--	Ref	--	--	Ref	--	--
Sex (W1)									
Male	--	--	--	0.873	0.018	<0.0001	0.873	0.018	<0.0001
Female	--	--	--	Ref	--	--	Ref	--	--
Age (W3), years	--	--	--	0.121	0.005	<0.0001	0.121	0.005	<0.0001
Early SES (W3)									
Public Assist. before 18 yrs	--	--	--	0.071	0.022	0.0019	0.073	0.023	0.0016
No Public Asst.	--	--	--	Ref	--	--	Ref	--	--
Education (W3)									
<High School	--	--	--	0.436	0.032	<0.0001	0.435	0.032	<0.0001
High School Grad	--	--	--	0.202	0.023	<0.0001	0.202	0.023	<0.0001
College Grad	--	--	--	Ref	--	--	Ref	--	--
Population Density (W3)									
<180.2 pop./Km^2	--	--	--	Ref	--	--	Ref	--	--
180.2 - 1,027.6 pop./Km^2	--	--	--	-0.059	0.026	0.0260	-0.058	0.026	0.0290
1,027.7 - 2,630.4 pop./Km^2	--	--	--	-0.026	0.023	0.2641	-0.026	0.023	0.2702
>2,630.4 pop./Km^2	--	--	--	-0.126	0.031	<0.0001	-0.124	0.030	<0.0001
Income (W3)									
<$20,000	--	--	--	-0.040	0.030	0.1841	-0.040	0.030	0.1871
$20,000-$39,999	--	--	--	-0.038	0.031	0.2161	-0.039	0.031	0.2137
$40,000-$74,999	--	--	--	-0.068	0.038	0.0754	-0.068	0.038	0.0752
>=$75,000	--	--	--	Ref	--	--	Ref	--	--
Missing Flag	--	--	--	-0.049	0.054	0.3614	-0.046	0.052	0.3746
4 or More PEs*Black	--	--	--	--	--	--	-0.182	0.119	0.1269

[a] 30-Year cardiovascular disease (hard) risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[b] Beta coefficients are generated using a generalized least-squared regression analysis incorporating Add Health's complex research design.

[c] Model 1 includes police encounters (PEs) and race.
[d] Model 2 includes the PEs, race, sex, age, early socioeconomic status (SES), education level, population density, and income
[e] Model 3 includes the variables in the Model 2 plus an interaction between race and PEs.
P-values <0.05 are statistically significant. CI=confidence interval, SE=standard error

Supplementary Table 3. 8 Linear Regression Coefficients for Regression of 30-Year Full Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on Police Encounters (PEs) (<6 vs 6 or more) at Wave III (n=8,447).

	Log Transformed 30-year Cardiovascular Risk (Full)[a]								
	Model 1[b,c] (R^2=0.008156)			Model 2[b,d](R^2=0.4048)			Model 3[b,e](R^2=0.4055)		
	Beta	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-2.308	0.019	<0.0001	-5.003	0.106	<0.0001	-5.011	0.106	<0.0001
Police Encounters (W3)									
6 or More PEs	0.253	0.057	<0.0001	-0.096	0.049	0.0497	-0.039	0.046	0.3970
<6 PEs	Ref	--	--	Ref	--	--	Ref	--	--
Race (W1)									
Black	0.108	0.032	0.0010	0.088	0.017	<0.0001	0.095	0.017	<0.0001
White	Ref	--	--	Ref	--	--	Ref	--	--
Sex (W1)									
Male	--	--	--	0.655	0.016	<0.0001	0.655	0.016	<0.0001
Female	--	--	--	Ref	--	--	Ref	--	--
Age (W3), years	--	--	--	0.105	0.005	<0.0001	0.105	0.005	<0.0001
Early SES (W3)									
Public Assist. before 18 yrs	--	--	--	0.062	0.019	0.0013	0.065	0.019	0.0008
No Public Asst.	--	--	--	Ref	--	--	Ref	--	--
Education (W3)									
<High School	--	--	--	0.366	0.027	<0.0001	0.364	0.027	<0.0001
High School Grad	--	--	--	0.171	0.020	<0.0001	0.171	0.020	<0.0001
College Grad	--	--	--	Ref	--	--	Ref	--	--
Population Density (W3)									
<180.2 pop./Km^2	--	--	--	Ref	--	--	Ref	--	--
180.2 - 1,027.6 pop./Km^2	--	--	--	-0.053	0.023	0.0211	-0.052	0.023	0.0224
1,027.7 - 2,630.4 pop./Km^2				-0.025	0.020	0.2151	-0.025	0.020	0.2146
>2,630.4 pop./Km^2	--	--	--	-0.109	0.026	<0.0001	-0.107	0.026	<0.0001
Income (W3)									
<$20,000	--	--	--	-0.035	0.025	0.1700	-0.034	0.025	0.1751
$20,000-$39,999	--	--	--	-0.037	0.026	0.1640	-0.037	0.026	0.1666
$40,000-$74,999	--	--	--	-0.059	0.032	0.0673	-0.059	0.032	0.0673
>=$75,000	--	--	--	Ref	--	--	Ref	--	--
Missing Flag	--	--	--	-0.041	0.046	0.3779	-0.036	0.044	0.4124
6 or More PEs*Black	--	--	--	--	--	--	-0.278	0.120	0.0217

[a] 30-Year cardiovascular disease (full) risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke, coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[b] Beta coefficients are generated using a generalized least-squared regression analysis incorporating Add Health's complex research design.
[c] Model 1 includes police encounters (PEs) and race.
[d] Model 2 includes the PEs, race, sex, age, early socioeconomic status (SES), education level, population density, and income
[e] Model 3 includes the variables in the Model 2 plus an interaction between race and PEs.
P-values <0.05 are statistically significant. CI=confidence interval, SE=standard error

Supplementary Table 3. 9 Linear Regression Coefficients for Regression of 30-Year Hard Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on Police Encounters (PEs) (<6 vs 6 or more) at Wave III (n=8,447).

	Log Transformed 30-year Cardiovascular Risk (Hard)[a]								
	Model 1[b,c] (R^2=0.009251)			Model 2[b,d] (R^2=0.4512)			Model 3[b,e] (R^2=0.4520)		
	Beta	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-3.009	0.023	<0.0001	-6.176	0.123	<0.0001	-6.185	0.123	<0.0001
Police Encounters (W3)									
6 or More PEs	0.354	0.068	<0.0001	-0.111	0.058	0.0569	-0.041	0.054	0.4478
<6 PEs	Ref	--	--	Ref	--	--	Ref	--	--
Race (W1)									
Black	0.129	0.039	0.0012	0.109	0.020	<0.0001	0.118	0.021	<0.0001
White	Ref	--	--	Ref	--	--	Ref	--	--
Sex (W1)									
Male	--	--	--	0.875	0.018	<0.0001	0.875	0.018	<0.0001
Female	--	--	--	Ref	--	--	Ref	--	--
Age (W3), years	--	--	--	0.121	0.005	<0.0001	0.121	0.005	<0.0001
Early SES (W3)									
Public Assist. before 18 yrs	--	--	--	0.071	0.022	0.0017	0.075	0.022	0.0011
No Public Asst.	--	--	--	Ref	--	--	Ref	--	--
Education (W3)									
<High School	--	--	--	0.439	0.032	<0.0001	0.437	0.031	<0.0001
High School Grad	--	--	--	0.203	0.023	<0.0001	0.203	0.023	<0.0001
College Grad	--	--	--	Ref	--	--	Ref	--	--
Population Density (W3)									
<180.2 pop./Km^2	--	--	--	Ref	--	--	Ref	--	--
180.2 - 1,027.6 pop./Km^2				-0.059	0.026	0.0254	-0.059	0.026	0.0271
1,027.7 - 2,630.4 pop./Km^2	--	--	--	-0.027	0.023	0.2560	-0.027	0.023	0.2554
>2,630.4 pop./Km^2	--	--	--	-0.124	0.030	<0.0001	-0.122	0.030	<0.0001
Income (W3)									
<$20,000	--	--	--	-0.040	0.030	0.1741	-0.040	0.030	0.1793
$20,000-$39,999	--	--	--	-0.040	0.031	0.1998	-0.040	0.031	0.2026
$40,000-$74,999	--	--	--	-0.069	0.037	0.0675	-0.069	0.038	0.0675
>=$75,000	--	--	--	Ref	--	--	Ref	--	--
Missing Flag	--	--	--	-0.050	0.053	0.3489	-0.044	0.050	0.3839
6 or More PEs*Black	--	--	--	--	--	--	-0.339	0.137	0.0145

[a] 30-Year cardiovascular disease (hard) risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[b] Beta coefficients are generated using a generalized least-squared regression analysis incorporating Add Health's complex research design.

[c] Model 1 includes police encounters (PEs) and race.
[d] Model 2 includes the PEs, race, sex, age, early socioeconomic status (SES), education level, population density, and income
[e] Model 3 includes the variables in the Model 2 plus an interaction between race and PEs.
P-values <0.05 are statistically significant. CI=confidence interval, SE=standard error

Supplementary Table 3. 10 Linear Regression Coefficients for Regression of 30-Year Full Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on Police Encounters (PEs) (All Categories, Never as Reference) at Wave III (n=8,447).

	Log Transformed 30-year Cardiovascular Risk (Full)[a]								
	Model 1[b,c] (R^2=0.02510)			Model 2[b,d] (R^2=0.4056)			Model[b,e] (R^2=0.4067)		
	Beta	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-2.344	0.020	<0.0001	-5.016	0.107	<0.0001	-5.030	0.106	<0.0001
Police Encounters (W3)									
Never	Ref	--	--	Ref	--	--	Ref	--	--
1 PE	0.103	0.037	0.0059	0.007	0.024	0.7887	0.017	0.025	0.4895
2-3 PEs	0.243	0.031	<0.0001	0.067	0.024	0.0060	0.083	0.026	0.0021
4-5 PEs	0.361	0.054	<0.0001	0.048	0.050	0.3327	0.039	0.054	0.4658
6 or more PEs	0.287	0.058	<0.0001	-0.086	0.049	0.0831	-0.027	0.046	0.5628
Race (W1)									
Black	0.114	0.032	0.0005	0.089	0.017	<0.0001	0.107	0.020	<0.0001
White	Ref	--	--	Ref	--	--	Ref	--	--
Sex (W1)									
Male	--	--	--	0.648	0.016	<0.0001	0.649	0.016	<0.0001
Female	--	--	--	Ref	--	--	Ref	--	--
Age (W3), years	--	--	--	0.105	0.005	<0.0001	0.106	0.005	<0.0001
Early SES (W3)									
Public Assist. before 18 yrs	--	--	--	0.060	0.019	0.0020	0.063	0.019	0.0013
No Public Asst.	--	--	--	Ref	--	--	Ref	--	--
Education (W3)									
<High School	--	--	--	0.365	0.027	<.0001	0.365	0.027	<0.0001
High School Grad	--	--	--	0.171	0.020	<.0001	0.171	0.020	<0.0001
College Grad	--	--	--	Ref	--	--	Ref	--	--
Population Density (W3)									
<180.2 pop./Km^2	--	--	--	-0.052	0.023	0.0221	-0.052	0.023	0.0241
180.2 - 1,027.6 pop./Km^2	--	--	--	-0.027	0.020	0.1775	-0.028	0.020	0.1708
1,027.7 - 2,630.4 pop./Km^2				-0.011	0.026	<0.0001	-0.110	0.025	<0.0001
>2,630.4 pop./Km^2	--	--	--	Ref	--	--	Ref	--	--
Income (W3)									
<$20,000	--	--	--	-0.034	0.025	0.1801	-0.033	0.025	0.1909
$20,000-$39,999	--	--	--	-0.037	0.026	0.1591	-0.036	0.026	0.1731
$40,000-$74,999	--	--	--	-0.058	0.032	0.0689	-0.058	0.032	0.0698
>=$75,000	--	--	--	Ref	--	--	Ref	--	--
Missing Flag	--	--	--	-0.037	0.046	0.4209	-0.032	0.044	0.4649
Interactions									

Never*Black	--	--	--	--	--	--	---	--	--
1 PE*Black	--	--	--	--	--	--	-0.081	0.056	0.1511
2-3 PEs*Black	--	--	--	--	--	--	-0.096	0.069	0.1677
4-5 PEs*Black	--	--	--	--	--	--	0.062	0.107	0.5615
6 or More PEs*Black	--	--	--	--	--	--	-0.290	0.119	0.0167

[a] 30-Year cardiovascular disease (full) risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke, coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[b] Beta coefficients are generated using a generalized least-squared regression analysis incorporating Add Health's complex research design.

[c] Model 1 includes police encounters (PEs) and race.

[d] Model 2 includes the PEs, race, sex, age, early socioeconomic status (SES), education level, population density, and income

[e] Model 3 includes the variables in the Model 2 plus an interaction between race and PEs.

P-values <0.05 are statistically significant. CI=confidence interval, SE=standard error

Supplementary Table 3. 11 Linear Regression Coefficients for Regression of 30-Year Hard Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on Police Encounters (PEs) (All Categories, Never as Reference) at Wave III (n=8,447).

	Log Transformed 30-year Cardiovascular Risk (Hard)[a]								
	Model 1[b,c] (R^2=0.02947)			Model 2[b,d] (R^2=0.4520)			Model 3[b,e] (R^2=0.4531)		
	Beta	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-3.058	0.024	<0.0001	-6.192	0.124	<0.0001	-6.209	0.123	<0.0001
Police Encounters (W3)									
Never	Ref	--	--	Ref	--	--	Ref	--	--
1 PE	0.144	0.045	0.0015	0.011	0.028	0.6942	0.024	0.029	0.4205
2-3 PEs	0.323	0.038	<0.0001	0.086	0.029	0.0033	0.103	0.031	0.0013
4-5 PEs	0.474	0.063	<0.0001	0.055	0.057	0.3360	0.046	0.061	0.4598
6 or more PEs	0.401	0.069	<0.0001	-0.098	0.059	0.0979	-0.026	0.054	0.6359
Race (W1)									
Black	0.137	0.039	0.0006	0.110	0.020	<0.0001	0.131	0.024	<0.0001
White	Ref	--	--	Ref	--	--	Ref	--	--
Sex (W1)									
Male	--	--	--	0.866	0.019	<0.0001	0.867	0.019	<0.0001
Female	--	--	--	Ref	--	--	Ref	--	--
Age (W3), years	--	--	--	0.121	0.005	<0.0001	0.122	0.005	<0.0001
Early SES (W3)									
Public Assist. before 18 yrs	--	--	--	0.068	0.022	0.0027	0.072	0.023	0.0019
No Public Asst.	--	--	--	Ref	--	--	Ref	--	--
Education (W3)									
<High School	--	--	--	0.438	0.032	<0.0001	0.437	0.031	<0.0001
High School Grad	--	--	--	0.203	0.023	<0.0001	0.203	0.023	<0.0001
College Grad	--	--	--	Ref	--	--	Ref	--	--
Population Density (W3)									
<180.2 pop./Km^2	--	--	--	Ref	--	--	Ref	--	--
180.2 - 1,027.6 pop./Km^2	--	--	--	-0.059	0.026	0.0266	-0.058	0.026	0.0291
1,027.7 - 2,630.4 pop./Km^2				-0.030	0.024	0.2081	-0.030	0.023	0.2010
>2,630.4 pop./Km^2	--	--	--	-0.126	0.030	<0.0001	-0.125	0.030	<0.0001
Income (W3)									
<$20,000	--	--	--	-0.039	0.030	0.1864	-0.038	0.030	0.1976
$20,000-$39,999	--	--	--	-0.040	0.031	0.1957	-0.039	0.031	0.2114
$40,000-$74,999	--	--	--	-0.068	0.037	0.0697	-0.068	0.037	0.0709
>=$75,000	--	--	--	Ref	---	---	Ref	--	--
Missing Flag	--	--	--	-0.045	0.053	0.3964	-0.039	0.051	0.4412
Interactions									

Never*Black	--	--	--	--	--	--	Ref	--	--
1 PE*Black	--	--	--	--	--	--	-0.092	0.065	0.1607
2-3 PEs*Black	--	--	--	--	--	--	-0.101	0.084	0.2322
4-5 PEs*Black	--	--	--	--	--	--	0.068	0.126	0.5929
6 or More PEs*Black	--	--	--	--	--	--	-0.352	0.137	0.0114

[a] 30-Year cardiovascular disease (hard) risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[b] Beta coefficients are generated using a generalized least-squared regression analysis incorporating Add Health's complex research design.

[c] Model 1 includes police encounters (PEs) and race.

[d] Model 2 includes the PEs, race, sex, age, early socioeconomic status (SES), education level, population density, and income

[e] Model 3 includes the variables in the Model 2 plus an interaction between race and PEs.

P-values <0.05 are statistically significant. CI=confidence interval, SE=standard error

Supplementary Table 3. 12 Linear Regression Coefficients for Regression of 30-Year Full Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on Police Encounters (PEs) (All Categories, 6 or More as Reference) at Wave III (n=8,447).

	Log Transformed 30-year Cardiovascular Risk (Full)[a]								
	Model 1[b,c] (R^2=0.02510)			Model 2[b,d] (R^2=0.4056)			Model 3[b,e] (R^2=0.4067)		
	Beta	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-2.056	0.062	<0.0001	-5.101	0.106	<0.0001	-5.057	0.106	<0.0001
Police Encounters (W3)									
Never	-0.287	0.058	<0.0001	0.086	0.049	0.0831	0.027	0.046	0.5628
1 PE	-0.185	0.064	0.0045	0.092	0.054	0.0889	0.044	0.052	0.3951
2-3 PEs	-0.044	0.060	0.4599	0.153	0.049	0.0024	0.110	0.048	0.0246
4-5 PEs	0.074	0.075	0.3277	0.134	0.069	0.0527	0.066	0.074	0.3740
6 or more PEs	Ref	--	--	Ref	--	--	Ref	--	--
Race (W1)									
Black	0.114	0.032	0.0005	0.089	0.017	<0.0001	-0.183	0.120	0.1313
White	Ref	--	--	Ref	--	--	Ref	--	--
Sex (W1)									
Male	--	--	--	0.648	0.016	<0.0001	0.649	0.016	<0.0001
Female	--	--	--	Ref	--	--	Ref	--	--
Age (W3), years	--	--	--	0.105	0.005	<0.0001	0.106	0.005	<0.0001
Early SES (W3)									
Public Assist. before 18 yrs	--	--	--	0.060	0.019	0.0020	0.063	0.019	0.0013
No Public Asst.	--	--	--	Ref	--	--	Ref	--	--
Education (W3)									
<High School	--	--	--	0.365	0.027	<0.0001	0.365	0.027	<0.0001
High School Grad	--	--	--	0.171	0.020	<0.0001	0.171	0.020	<0.0001
College Grad	--	--	--	Ref	--	--	Ref	--	--
Population Density (W3)									
<180.2 pop./Km^2	--	--	--	Ref	--	--	Ref	--	--
180.2 - 1,027.6 pop./Km^2	--	--	--	-0.052	0.023	0.0221	-0.052	0.023	0.0241
1,027.7 - 2,630.4 pop./Km^2	--	--	--	-0.027	0.020	0.1775	-0.028	0.020	0.1708
>2,630.4 pop./Km^2	--	--	--	-0.111	0.026	<0.0001	-0.110	0.025	<0.0001
Income (W3)									
<$20,000	--	--	--	-0.034	0.025	0.1801	-0.033	0.025	0.1909
$20,000-$39,999	--	--	--	-0.037	0.026	0.1591	-0.036	0.026	0.1731
$40,000-$74,999	--	--	--	-0.058	0.032	0.0689	-0.058	0.032	0.0698
>=$75,000	--	--	--	Ref	--	--	Ref	--	--
Missing Flag	--	--	--	-0.037	0.046	0.4209	-0.032	0.044	0.4649
Interactions									

Never*Black	--	--	--	--	--	--	0.290	0.119	0.0167
1 PE*Black	--	--	--	--	--	--	0.209	0.141	0.1406
2-3 PEs*Black	--	--	--	--	--	--	0.194	0.130	0.1381
4-5 PEs*Black	--	--	--	--	--	--	0.352	0.158	0.0277
6 or More PEs*Black	--	--	--	--	--	--	Ref	--	--

[a] 30-Year cardiovascular disease (full) risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke, coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[b] Beta coefficients are generated using a generalized least-squared regression analysis incorporating Add Health's complex research design.

[c] Model 1 includes police encounters (PEs) and race.

[d] Model 2 includes the PEs, race, sex, age, early socioeconomic status (SES), education level, population density, and income

[e] Model 3 includes the variables in the Model 2 plus an interaction between race and PEs.

P-values <0.05 are statistically significant. CI=confidence interval, SE=standard error

Supplementary Table 3. 13 Linear Regression Coefficients for Regression of 30-Year Hard Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on Police Encounters (PEs) (All Categories, 6 or More as Reference) at Wave III (n=8,447).

	Log Transformed 30-year Cardiovascular Risk (Hard)[a]								
	Model 1[b,c] (R^2=0.02947)			Model 2[b,d] (R^2=0.4520)			Model 3[b,e] (R^2=0.4531)		
	Beta	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-2.656	0.073	<0.0001	-6.289	0.121	<0.0001	-6.235	0.121	<0.0001
Police Encounters (W3)									
Never	-0.401	0.069	<0.0001	0.098	0.059	0.0979	0.026	0.054	0.6359
1 PE	-0.257	0.076	0.0010	0.109	0.063	0.0882	0.049	0.061	0.4183
2-3 PEs	-0.078	0.071	0.2756	0.184	0.059	0.0022	0.128	0.057	0.0265
4-5 PEs	0.072	0.088	0.4103	0.153	0.080	0.0573	0.071	0.085	0.4043
6 or more PEs	Ref	--	--	Ref	--	--	Ref	---	--
Race (W1)									
Black	0.137	0.039	0.0006	0.110	0.020	<0.0001	-0.221	0.137	0.1107
White	Ref	--	--	Ref	--	--	Ref	--	--
Sex (W1)									
Male	--	--	--	0.866	0.019	<0.0001	0.867	0.019	<0.0001
Female	--	--	--	Ref	--	--	Ref	--	--
Age (W3), years	--	--	--	0.121	0.005	<0.0001	0.122	0.005	<0.0001
Early SES (W3)									
Public Assist. before 18 yrs	--	--	--	0.068	0.022	0.0027	0.072	0.023	0.0019
No Public Asst.	--	--	--	Ref	--	--	Ref	--	--
Education (W3)									
<High School	--	--	--	0.438	0.032	<0.0001	0.437	0.031	<0.0001
High School Grad	--	--	--	0.203	0.023	<0.0001	0.203	0.023	<0.0001
College Grad	--	--	--	Ref	--	--	Ref	--	--
Population Density (W3)									
<180.2 pop./Km^2	--	--	--	Ref	--	--	Ref	--	--
180.2 - 1,027.6 pop./Km^2	--	--	--	-0.059	0.026	0.0266	-0.058	0.026	0.0291
1,027.7 - 2,630.4 pop./Km^2				-0.030	0.024	0.2081	-0.030	0.023	0.2010
>2,630.4 pop./Km^2	--	--	--	-0.126	0.030	<0.0001	-0.125	0.030	<0.0001
Income (W3)									
<$20,000	--	--	--	-0.039	0.030	0.1864	-0.038	0.030	0.1976
$20,000-$39,999	--	--	--	-0.040	0.031	0.1957	-0.039	0.031	0.2114
$40,000-$74,999	--	--	--	-0.068	0.037	0.0697	-0.068	0.037	0.0709
>=$75,000	--	--	--	Ref	---	---	Ref	--	--
Missing Flag	--	--	--	-0.045	0.053	0.3964	-0.039	0.051	0.4412
Interactions									

Never*Black	--	--	--	--	--	--	0.352	0.137	0.0114
1 PE*Black	--	--	--	--	--	--	0.259	0.158	0.1037
2-3 PEs*Black	--	--	--	--	--	--	0.251	0.149	0.0949
4-5 PEs*Black	--	--	--	--	--	--	0.419	0.182	0.0226
6 or More PEs*Black	--	--	--	--	--	--	Ref	---	---

[a] 30-Year cardiovascular disease (hard) risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[b] Beta coefficients are generated using a generalized least-squared regression analysis incorporating Add Health's complex research design.

[c] Model 1 includes police encounters (PEs) and race.

[d] Model 2 includes the PEs, race, sex, age, early socioeconomic status (SES), education level, population density, and income

[e] Model 3 includes the variables in the Model 2 plus an interaction between race and PEs.

P-values <0.05 are statistically significant. CI=confidence interval, SE=standard error

Supplementary Table 3. 14 Linear Regression Coefficients for Regression of 30-Year Full Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on Police Encounters (PEs) (Sex Specific Categories, Never as Reference) at Wave III among Men (n=3,734).

	Log Transformed 30-year Cardiovascular Risk (Full)[a]								
	Model 1[b,c] (R^2=0.009194)			Model 2[b,d] (R^2=0.1521)			Model 3[b,e] (R^2=0.1547)		
	Beta	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-1.941	0.019	<0.0001	-4.021	0.144	<0.0001	-4.037	0.144	<0.0001
Police Encounters (W3)									
Never	Ref	---	---	Ref	---	---	Ref	---	---
1 PE	-0.040	0.029	0.1719	-0.008	0.026	0.7718	-0.001	0.027	0.9702
2-3 PEs	0.041	0.029	0.1553	0.057	0.028	0.0457	0.060	0.032	0.0603
4-5 PEs	0.011	0.059	0.8476	0.020	0.053	0.7011	-0.003	0.055	0.9590
6 or more PEs	-0.096	0.059	0.1080	-0.088	0.048	0.0700	-0.037	0.048	0.4350
Race (W1)									
Black	0.099	0.033	0.0030	0.057	0.024	0.0187	0.070	0.028	0.0140
White	Ref	---	---	Ref	---	---	Ref	---	---
Age (W3), years	---	---	---	0.094	0.006	<0.0001	0.094	0.006	<0.0001
Early SES (W3)									
Public Assist. before 18 yrs	---	---	---	0.013	0.029	0.6525	0.018	0.029	0.5355
No Public Asst.	---	---	---	Ref	---	---	Ref	---	---
Education (W3)									
<High School	---	---	---	0.274	0.038	<0.0001	0.273	0.037	<0.0001
High School Grad	---	---	---	0.102	0.034	0.0030	0.102	0.034	0.0029
College Grad	---	---	---	Ref	---	---	Ref	---	---
Population Density (W3)									
<180.2 pop./Km2	---	---	---	Ref	---	---	Ref	---	---
180.2 - 1,027.6 pop./Km^2	---	---	---	-0.082	0.026	0.0022	-0.082	0.026	0.0022
1,027.7 - 2,630.4 pop./Km^2	---	---	---	-0.067	0.025	0.0077	-0.068	0.025	0.0069
>2,630.4 pop./Km^2	---	---	---	-0.083	0.037	0.0262	-0.082	0.037	0.0274
Income (W3)									
<$20,000	---	---	---	-0.036	0.032	0.2664	-0.036	0.033	0.2749
$20,000-$39,999	---	---	---	-0.017	0.032	0.5908	-0.017	0.032	0.6025
$40,000-$74,999	---	---	---	-0.076	0.035	0.0303	-0.077	0.035	0.0289
>=$75,000	---	---	---	Ref	---	---	Ref	---	---
Missing Flag	---	---	---	-0.026	0.064	0.6849	-0.018	0.059	0.7629
Interactions									
Never*Black	---	---	---	---	---	---	---	---	---
1 PE*Black	---	---	---	---	---	---	-0.043	0.078	0.5793
2-3 PEs*Black	---	---	---	---	---	---	-0.020	0.070	0.7793

4-5 PEs*Black	---	---	---	---	---	---	0.152	0.110	0.1700
6 or More PEs*Black	---	---	---	---	---	---	-0.244	0.125	0.0543

[a] 30-Year cardiovascular disease (full) risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke, coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[b] Beta coefficients are generated using a generalized least-squared regression analysis incorporating Add Health's complex research design.

[c] Model 1 includes police encounters (PEs) and race.

[d] Model 2 includes the PEs, race, sex, age, early socioeconomic status (SES), education level, population density, and income

[e] Model 3 includes the variables in the Model 2 plus an interaction between race and PEs.

P-values <0.05 are statistically significant. CI=confidence interval, SE=standard error

Supplementary Table 3. 15 Linear Regression Coefficients for Regression of 30-Year Full Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on Police Encounters (PEs) (Sex Specific Categories, Never as Reference) at Wave III among Women (n=4,713).

	Log Transformed 30-year Cardiovascular Risk (Full)[a]								
	Model 1[b,c] (R^2=0.01194)			Model 2[b,d] (R^2=0.1713)			Model 3[b,e] (R^2=0.1722)		
	Beta	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-2.646	0.023	<0.0001	-5.357	0.152	<0.0001	-5.362	0.152	<0.0001
Police Encounters (PEs)									
Never	Ref	---	---	Ref	---	---	Ref	---	---
1 PE	-0.00005	0.050	0.9993	0.018	0.041	0.6570	0.030	0.044	0.5012
2+ PEs	0.071	0.045	0.1182	0.111	0.040	0.0066	0.139	0.040	0.0007
Race (W1)									
Black	0.158	0.034	<0.0001	0.118	0.023	<0.0001	0.130	0.025	<0.0001
White	Ref	---	---	Ref	---	---	Ref	---	---
Age (W3), years	---	---	---	0.117	0.007	<0.0001	0.117	0.007	<0.0001
Early SES (W3)									
Public Assist. before 18 yrs	---	---	---	0.099	0.025	<0.0001	0.100	0.025	<0.0001
No Public Asst.	---	---	---	Ref	---	---	Ref	---	---
Education (W3)									
<High School	---	---	---	0.442	0.042	<0.0001	0.442	0.042	<0.0001
High School Grad	---	---	---	0.226	0.031	<0.0001	0.226	0.031	<0.0001
College Grad	---	---	---	Ref	---	---	Ref	---	---
Population Density (W3)									
<180.2 pop./Km2	---	---	---	Ref	---	---	Ref	---	---
180.2 - 1,027.6 pop./Km^2	---	---	---	-0.029	0.029	0.3192	-0.027	0.029	0.3396
1,027.7 - 2,630.4 pop./Km^2	---	---	---	0.008	0.029	0.7730	0.009	0.029	0.7619
>2,630.4 pop./Km^2	---	---	---	-0.136	0.034	0.0001	-0.137	0.034	<0.0001
Income (W3)									
<$20,000	---	---	---	-0.030	0.037	0.4144	-0.030	0.037	0.4190
$20,000-$39,999	---	---	---	-0.048	0.042	0.2562	-0.048	0.043	0.2672
$40,000-$74,999	---	---	---	-0.021	0.050	0.6734	-0.021	0.050	0.6745
>=$75,000	---	---	---	Ref	---	---	Ref	---	---
Missing Flag	---	---	---	-0.060	0.064	0.3459	-0.063	0.064	0.3273
Interactions									
Never*Black	---	---	---	---	---	---	---	---	---
1 PE*Black	---	---	---	---	---	---	-0.100	0.113	0.3785
2 or More PEs*Black	---	---	---	---	---	---	-0.232	0.145	0.1117

[a] 30-Year cardiovascular disease (full) risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke, coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure. The risk score is calculated using a Cox-

Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[b] Beta coefficients are generated using a generalized least-squared regression analysis incorporating Add Health's complex research design.

[c] Model 1 includes police encounters (PEs) and race.

[d] Model 2 includes the PEs, race, sex, age, early socioeconomic status (SES), education level, population density, and income

[e] Model 3 includes the variables in the Model 2 plus an interaction between race and PEs.

P-values <0.05 are statistically significant. CI=confidence interval, SE=standard error

Supplementary Table 3. 16 Linear Regression Coefficients for Regression of 30-Year Hard Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on Police Encounters (PEs) (Sex Specific Categories, Never as Reference) at Wave III among Men (n=3,734).

	Log Transformed 30-year Cardiovascular Risk (Hard)[a]								
	Model 1[b,c] (R^2=0.01076)			Model 2[b,d] (R^2=0.1502)			Model 3[b,e] (R^2=0.1530)		
	Beta	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-2.527	0.023	<0.0001	-4.942	0.172	<0.0001	-4.962	0.171	<0.0001
Police Encounters (W3)									
Never	Ref	---	---	Ref	---	---	Ref	---	---
1 PE	-0.041	0.034	0.2335	-0.004	0.031	0.9055	0.004	0.032	0.8942
2-3 PEs	0.054	0.035	0.1189	0.072	0.034	0.0357	0.076	0.038	0.0482
4-5 PEs	0.015	0.068	0.8276	0.024	0.061	0.6987	-0.0005	0.063	0.9940
6 or more PEs	-0.108	0.070	0.1288	-0.101	0.058	0.0825	-0.036	0.056	0.5236
Race (W1)									
Black	0.134	0.039	0.0008	0.084	0.028	0.0037	0.102	0.034	0.0036
White	Ref	---	---	Ref	---	---	Ref	---	---
Age (W3), years	---	---	---	0.108	0.007	<0.0001	0.109	0.007	<0.0001
Early SES (W3)									
Public Assist. before 18 yrs	---	---	---	0.014	0.035	0.6960	0.020	0.035	0.5717
No Public Asst.	---	---	---	Ref	---	---	Ref	---	---
Education (W3)									
<High School	---	---	---	0.337	0.046	<0.0001	0.336	0.045	<0.0001
High School Grad	---	---	---	0.126	0.040	0.0020	0.126	0.040	0.0020
College Grad	---	---	---	Ref	---	---	Ref	---	---
Population Density (W3)									
<180.2 pop./Km^2	---	---	---	Ref	---	---	Ref	---	---
180.2 - 1,027.6 pop./Km^2	---	---	---	-0.096	0.031	0.0025	-0.096	0.031	0.0026
1,027.7 - 2,630.4 pop./Km^2	---	---	---	-0.076	0.029	0.0103	-0.077	0.029	0.0095
>2,630.4 pop./Km^2	---	---	---	-0.096	0.044	0.0326	-0.095	0.044	0.0352
Income (W3)									
<$20,000	---	---	---	-0.044	0.038	0.2476	-0.044	0.039	0.2562
$20,000-$39,999	---	---	---	-0.018	0.038	0.6282	-0.018	0.038	0.6381
$40,000-$74,999	---	---	---	-0.093	0.041	0.0247	-0.094	0.041	0.0235
>=$75,000	---	---	---	Ref	---	---	Ref	---	---
Missing Flag	---	---	---	-0.036	0.075	0.6304	-0.026	0.069	0.7097
Interactions									
Never*Black	---	---	---	---	---	---	---	---	---
1 PE*Black	---	---	---	---	---	---	-0.051	0.093	0.5796
2-3 PEs*Black	---	---	---	---	---	---	-0.022	0.087	0.7997

4-5 PEs*Black	---	---	---	---	---	---	0.158	0.131	0.2288
6 or More PEs*Black	---	---	---	---	---	---	-0.312	0.144	0.0317

[a] 30-Year cardiovascular disease (hard) risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[b] Beta coefficients are generated using a generalized least-squared regression analysis incorporating Add Health's complex research design.

[c] Model 1 includes police encounters (PEs) and race.

[d] Model 2 includes the PEs, race, sex, age, early socioeconomic status (SES), education level, population density, and income

[e] Model 3 includes the variables in the Model 2 plus an interaction between race and PEs.

P-values <0.05 are statistically significant. CI=confidence interval, SE=standard error

Supplementary Table 3. 17 Linear Regression Coefficients for Regression of 30-Year Hard Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on Police Encounters (PEs) (Sex Specific Categories, Never as Reference) at Wave III, Women (n=4,713).

	Log Transformed 30-year Cardiovascular Risk (Hard)[a]								
	Model 1[b,c] (R^2=0.01186)			Model 2[b,d] (R^2=0.1698)			Model 3[b,e] (R^2=0.1707)		
	Beta	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-3.455	0.027	<0.0001	-6.571	0.178	<0.0001	-6.578	0.178	<0.0001
Police Encounters (W3)									
Never	Ref	---	---	Ref	---	---	Ref	---	---
1 PE	0.002	0.058	0.9739	0.022	0.047	0.6396	0.036	0.050	0.4720
2+ PEs	0.092	0.053	0.0827	0.137	0.047	0.0044	0.169	0.047	0.0004
Race (W1)									
Black	0.181	0.039	<0.0001	0.135	0.027	<0.0001	0.148	0.029	<0.0001
White	Ref	---	---	Ref	---	---	Ref	---	---
Age (W3), years	---	---	---	0.134	0.008	<0.0001	0.135	0.008	<0.0001
Early SES (W3)									
Public Assist. before 18 yrs	---	---	---	0.114	0.029	0.0001	0.116	0.029	0.0001
No Public Asst.	---	---	---	Ref	---	---	Ref	---	---
Education (W3)									
<High School	---	---	---	0.523	0.048	<0.0001	0.523	0.048	<0.0001
High School Grad	---	---	---	0.264	0.036	<0.0001	0.264	0.035	<0.0001
College Grad	---	---	---	Ref	---	---	Ref	---	---
Population Density (W3)									
<180.2 pop./Km2	---	---	---	Ref	---	---	Ref	---	---
180.2 - 1,027.6 pop./Km^2	---	---	---	-0.029	0.032	0.3859	-0.027	0.033	0.4091
1,027.7 - 2,630.4 pop./Km^2	---	---	---	0.013	0.033	0.7011	0.013	0.033	0.6897
>2,630.4 pop./Km^2	---	---	---	-0.153	0.039	0.0001	-0.154	0.039	0.0001
Income (W3)									
<$20,000	---	---	---	-0.032	0.043	0.4624	-0.032	0.043	0.4677
$20,000-$39,999	---	---	---	-0.051	0.048	0.2895	-0.050	0.048	0.3024
$40,000-$74,999	---	---	---	-0.021	0.059	0.7249	-0.021	0.059	0.7265
>=$75,000	---	---	---	Ref	---	---	Ref	---	---
Missing Flag	---	---	---	-0.067	0.073	0.3654	-0.069	0.073	0.3463
Interactions									
Never*Black	---	---	---	---	---	---	---	---	---
1 PE*Black	---	---	---	---	---	---	-0.126	0.127	0.3244
2 or More PEs*Black	---	---	---	---	---	---	-0.264	0.173	0.1295

[a] 30-Year cardiovascular disease (hard) risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[b] Beta coefficients are generated using a generalized least-squared regression analysis incorporating Add Health's complex research design.
[c] Model 1 includes police encounters (PEs) and race.
[d] Model 2 includes the PEs, race, sex, age, early socioeconomic status (SES), education level, population density, and income
[e] Model 3 includes the variables in the Model 2 plus an interaction between race and PEs.
P-values <0.05 are statistically significant. CI=confidence interval, SE=standard error

Supplementary Table 3. 18 Logistic Regression Coefficients for Regression of 30-Year Full Cardiovascular Risk Score (High Risk at >20%) at Add Health Wave IV on Police Encounters (PEs) at Wave III (n=8,447).

	30-year Cardiovascular Risk (Full): High Risk (20% or more) vs. Low Risk (Less than 20%)[a]											
	Model 1[b,c]				Model 2[b,d]				Model 3[b,e]			
	Beta	**SE**	***P-value***	**OR (95% CI)**	**Beta**	**SE**	***P-value***	**OR (95% CI)**	**Beta**	**SE**	***P-value***	**OR (95% CI)**
Intercept	-1.657	0.116	<0.0001	--	-10.395	0.648	<0.0001	--	-10.541	0.662	<0.0001	--
Police Encounters (W3)[f]												
High PEs	-0.022	0.098	0.8235	0.957 (0.648, 1.412)	-0.048	0.109	0.6578	0.908 (0.591, 1.396)	-0.173	0.128	0.1779	0.841 (0.654, 1.083)
Low PEs	Ref	--	--	--	Ref	--	--	--	Ref	--	--	--
Race (W1)												
Black	0.205	0.059	0.0007	1.506 (1.193, 1.901)	0.202	0.053	0.0002	1.498 (1.217, 1.844)	0.002	0.142	0.9871	1.002 (0.757, 1.328)
White	Ref	--	--	--	Ref	--	--	--	Ref	--	--	--
Sex (W1)												
Male	--	--	--	--	0.957	0.058	<0.0001	6.786 (5.399, 8.530)	0.961	0.058	<0.0001	6.834 (5.438, 8.588)
Female	--	--	--	--	Ref	--	--	--	Ref	--	--	--
Age (W3), years	--	--	--	--	0.379	0.029	<0.0001	1.461 (1.380, 1.547)	0.381	0.029	<0.0001	1.463 (1.382, 1.549)
Early SES (W3)												
Public Assist. before 18	--	--	--	--	-0.014	0.129	0.9152	0.986 (0.765, 1.272)	-0.0004	0.130	0.9976	1.000 (0.772, 1.294)
No Public Asst.	--	--	--	--	Ref	--	--	--	Ref	--	--	--
Education (W3)												
<High School	--	--	--	--	0.639	0.092	<0.0001	3.495 (2.488, 4.909)	0.636	0.091	<0.0001	3.476 (2.483, 4.866)
High School Grad	--	--	--	--	-0.026	0.075	0.7235	1.797 (1.352, 2.388)	-0.025	0.074	0.7324	1.795 (1.352, 2.384)
College Grad	--	--	--	--	Ref	--	--	--	Ref	--	--	--
Population Density (W3)												
<180.2 pop./Km^2	--	--	--	--	Ref	--	--	--	Ref	--	--	--

180.2 - 1,027.6 pop./Km^2	--	--	--	--	-0.020	0.089	0.8202	0.858 (0.676, 1.088)	-0.020	0.089	0.8213	0.860 (0.676, 1.093)
1,027.7 - 2,630.4 pop./Km^2	--	--	--	--	-0.004	0.074	0.9584	0.872 (0.704, 1.080)	-0.006	0.075	0.9326	0.872 (0.703, 1.082)
>2,630.4 pop./Km^2	--	--	--	--	-0.109	0.119	0.3617	0.785 (0.571, 1.078)	-0.104	0.118	0.3769	0.790 (0.577, 1.083)
Income (W3)												
<$20,000	--	--	--	--	-0.004	0.079	0.9621	0.860 (0.653, 1.132)	-0.005	0.078	0.9501	0.861 (0.653, 1.135)
$20,000-$39,999	--	--	--	--	-0.007	0.097	0.9417	0.857 (0.631, 1.162)	-0.012	0.097	0.9048	0.855 (0.629, 1.162)
$40,000-$74,999	--	--	--	--	-0.028	0.128	0.8284	0.839 (0.576, 1.222)	-0.034	0.129	0.7916	0.836 (0.572, 1.221)
>=$75,000	--	--	--	--	Ref	--	--	--	Ref	--	--	--
Missing Flag	--	--	--	--	-0.109	0.161	0.4998	0.774 (0.509, 1.175)	-0.095	0.157	0.5483	0.787 (0.523, 1.186)
High PEs*Black	--	--	--	--	--	--	--	--	-0.219	0.132	0.0998	0.803 (0.619, 1.043)
Model Fit			**Value**				**Value**				**Value**	
-2 Log L			11165644				9202206.7				9194591.5	
AIC			11165650				9202236.7				9194623.5	

[a] 30-Year cardiovascular disease (full) risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke, coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[b] Beta coefficients are generated using a logistic regression analysis incorporating Add Health's complex research design.

[c] Model 1 includes police encounters (PEs) and race.

[d] Model 2 includes the PEs, race, sex, age, early socioeconomic status (SES), education level, population density, and income

[e] Model 3 includes the variables in the Model 2 plus an interaction between race and PEs.

[f] High PEs is 6 or more PEs among men, 2 or more for women. Low PEs is <6 PEs among men, <2 for women

P-values <0.05 are statistically significant. CI=confidence interval, SE=standard error

Supplementary Table 3. 19 Logistic Regression Coefficients for Regression of 30-Year Hard Cardiovascular Risk Score (High Risk at >20%) at Add Health Wave IV on Police Encounters (PEs) at Wave III (n=8,447).

	30-year Cardiovascular Risk (Hard): High Risk (20% or more) vs. Low Risk (Less than 20%)[a]											
	Model 1[b,c]				Model 2[b,d]				Model 3[b,e]			
	Beta	**SE**	***P-value***	**OR (95% CI)**	**Beta**	**SE**	***P-value***	**OR (95% CI)**	**Beta**	**SE**	***P-value***	**OR (95% CI)**
Intercept	-3.537	0.259	<0.0001	--	-12.081	1.272	<0.0001	--	-12.393	1.266	<0.0001	--
Police Encounters (W3)[f]												
High PEs	-0.509	0.260	0.0519	0.361 (0.129, 1.008)	-0.535	0.268	0.0481	0.343 (0.119, 0.991)	-0.812	0.247	0.0013	0.444 (0.272, 0.724)
Low PEs	Ref	--	--	--	Ref	--	--	--	Ref	--	--	--
Race (W1)												
Black	0.475	0.093	<0.0001	2.586 (1.793, 3.729)	0.484	0.084	<0.0001	2.635 (1.890, 3.673)	-0.024	0.247	0.9242	0.977 (0.599, 1.593)
White	Ref	--	--	--	Ref	--	--	--	Ref	--	--	--
Sex (W1)												
Male	--	--	--	--	0.871	0.101	<0.0001	5.709 (3.823, 8.524)	0.874	0.102	<0.0001	5.746 (3.846, 8.585)
Female	--	--	--	--	Ref	--	--	--	Ref	--	--	--
Age (W3), years	--	--	--	--	0.374	0.053	<0.0001	1.453 (1.309, 1.614)	0.376	0.053	<0.0001	1.456 (1.311, 1.617)
Early SES (W3)												
Public Assist. before 18	--	--	--	--	-0.241	0.224	0.2828	0.786 (0.504, 1.223)	-0.226	0.226	0.3178	0.798 (0.510, 1.246)
No Public Asst.	--	--	--	--	Ref	--	--	--	Ref	--	--	--
Education (W3)												
<High School	--	--	--	--	0.542	0.157	0.0008	2.320 (1.275, 4.221)	0.537	0.157	0.0008	2.301 (1.265, 4.187)
High School Grad	--	--	--	--	-0.242	0.110	0.0288	1.059 (0.670, 1.674)	-0.241	0.109	0.0295	1.057 (0.668, 1.673)
College Grad	--	--	--	--	Ref	--	--	--	Ref	--	--	--

Population Density (W3)												
<180.2 pop./Km^2	--	--	--	--	Ref	--	--	--	Ref	--	--	--
180.2 - 1,027.6 pop./Km^2	--	--	--	--	-0.059	0.147	0.6901	0.732 (0.479, 1.118)	-0.060	0.146	0.6828	0.732 (0.479, 1.118)
1,027.7 - 2,630.4 pop./Km^2					0.079	0.118	0.5022	0.840 (0.570, 1.238)	0.076	0.118	0.5182	0.839 (0.569, 1.236)
>2,630.4 pop./Km^2	--	--	--	--	-0.274	0.206	0.1855	0.590 (0.328, 1.061)	-0.268	0.205	0.1933	0.594 (0.331, 1.067)
Income (W3)												
<$20,000	--	--	--	--	-0.098	0.147	0.5056	0.676 (0.359, 1.273)	-0.096	0.147	0.5162	0.678 (0.359, 1.279)
$20,000-$39,999	--	--	--	--	0.017	0.151	0.9068	0.758 (0.400, 1.439)	0.014	0.151	0.9271	0.756 (0.398, 1.436)
$40,000-$74,999	--	--	--	--	-0.484	0.251	0.0561	0.460 (0.206, 1.025)	-0.488	0.251	0.0539	0.458 (0.205, 1.021)
>=$75,000	--	--	--	--	Ref	--	--	--	Ref	--	--	--
Missing Flag	--	--	--	--	0.271	0.284	0.3429	0.977 (0.379, 2.520)	0.277	0.286	0.3346	0.984 (0.380, 2.548)
High PEs*Black	--	--	--	--	--	--	--	--	-0.522	0.255	0.0423	0.593 (0.359, 0.982)
Model Fit			**Value**				**Value**				**Value**	
-2 Log L			4042912				3512062.9				3507057.2	
AIC			4042918				3512092.9				3507089.2	

[a] 30-Year cardiovascular disease (hard) risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[b] Beta coefficients are generated using a logistic regression analysis incorporating Add Health's complex research design.

[c] Model 1 includes police encounters (PEs) and race.

[d] Model 2 includes the PEs, race, sex, age, early socioeconomic status (SES), education level, population density, and income

[e] Model 3 includes the variables in the Model 2 plus an interaction between race and PEs.

[f] High PEs is 6 or more PEs among men, 2 or more for women. Low PEs is <6 PEs among men, <2 for women

P-values <0.05 are statistically significant. CI=confidence interval, SE=standard error

Supplementary Table 3. 20 Logistic Regression Coefficients for Regression of 30-Year Hard Cardiovascular Risk Score (High Risk at >10%) at Add Health Wave IV on Police Encounters (PEs) at Wave III (n=8,447).

	30-year Cardiovascular Risk (Hard): High Risk (10% or more) vs. Low Risk (Less than 10%)[a]											
	Model 1[b,c]				Model 2[b,d]				Model 3[b,e]			
	Beta	**SE**	***P-value***	**OR (95% CI)**	**Beta**	**SE**	***P-value***	**OR (95% CI)**	**Beta**	**SE**	***P-value***	**OR (95% CI)**
Intercept	-1.322	0.099	<0.0001	--	-9.454	0.621	<0.0001	--	-9.583	0.620	<0.0001	--
Police Encounters (W3)[f]												
High PEs	0.024	0.084	0.7734	1.049 (0.754, 1.461)	-0.003	0.094	0.9759	0.994 (0.687, 1.440)	-0.116	0.092	0.2087	0.891 (0.743, 1.068)
Low PEs	Ref	--	--	--	Ref	--	--	--	Ref	--	--	--
Race (W1)												
Black	0.170	0.053	0.0017	1.405 (1.139, 1.732)	0.181	0.049	0.0004	1.435 (1.180, 1.744)	0.007	0.106	0.9509	1.007 (0.816, 1.241)
White	Ref	--	--	--	Ref	--	--	--	Ref	--	--	--
Sex (W1)												
Male	--	--	--	--	1.112	0.058	<0.0001	9.245 (7.359, 11.614)	1.115	0.057	<0.0001	9.303 (7.415, 11.672)
Female	--	--	--	--	Ref	--	--	--	Ref	--	--	--
Age (W3), years	--	--	--	--	0.350	0.027	<0.0001	1.419 (1.344, 1.498)	0.351	0.027	<0.0001	1.421 (1.346, 1.499)
Early SES (W3)												
Public Assist. before 18	--	--	--	--	0.0001	0.103	0.9992	1.000 (0.817, 1.225)	0.012	0.103	0.9084	1.012 (0.826, 1.240)
No Public Asst.	--	--	--	--	Ref	--	--	--	Ref	--	--	--
Education (W3)												
<High School	--	--	--	--	0.636	0.081	<0.0001	3.400 (2.477, 4.668)	0.634	0.080	<0.0001	3.385 (2.472, 4.637)
High School Grad	--	--	--	--	-0.049	0.066	0.4570	1.713 (1.305, 2.249)	-0.048	0.065	0.4620	1.712 (1.304, 2.247)
College Grad	--	--	--	--	Ref	--	--	--	Ref	--	--	--
Population Density (W3)												

<180.2 pop./Km^2	--	--	--	--	Ref	--	--	--	-0.063	0.087	0.4688	--
180.2 - 1,027.6 pop./Km^2	--	--	--	--	-0.063	0.087	0.4660	0.824 (0.652, 1.041)	-0.007	0.068	0.9153	0.826 (0.653, 1.045)
1,027.7 - 2,630.4 pop./Km^2	--	--	--	--	-0.005	0.068	0.9389	0.873 (0.711, 1.072)	-0.058	0.112	0.6040	0.873 (0.710, 1.074)
>2,630.4 pop./Km^2	--	--	--	--	-0.062	0.113	0.5845	0.825 (0.603, 1.129)	Ref	--	--	0.830 (0.608, 1.132)
Income (W3)												
<$20,000	--	--	--	--	0.049	0.073	0.4986	0.885 (0.667, 1.175)	0.074	0.072	0.5114	0.886 (0.666, 1.178)
$20,000-$39,999	--	--	--	--	0.128	0.088	0.1481	0.957 (0.722, 1.270)	0.124	0.088	0.1594	0.956 (0.720, 1.271)
$40,000-$74,999	--	--	--	--	-0.034	0.122	0.7781	0.814 (0.563, 1.177	-0.041	0.121	0.7385	0.811 (0.561, 1.174)
>=$75,000	--	--	--	--	Ref	--	--	--	Ref	--	--	--
Missing Flag	--	--	--	--	-0.314	0.147	0.0340	0.615 (0.406, 0.932)	-0.299	0.141	0.0351	0.626 (0.418, 0.938)
High PEs*Black	--	--	--	--	--	--	--	--	-0.192	0.090	0.0355	0.825 (0.690, 0.987)
Model Fit		**Value**				**Value**				**Value**		
-2 Log L		13046735				10367344				10360305		
AIC		13046741				10367374				10360337		

[a] 30-Year cardiovascular disease (hard) risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[b] Beta coefficients are generated using a logistic regression analysis incorporating Add Health's complex research design.

[c] Model 1 includes police encounters (PEs) and race.

[d] Model 2 includes the PEs, race, sex, age, early socioeconomic status (SES), education level, population density, and income

[e] Model 3 includes the variables in the Model 2 plus an interaction between race and PEs.

[f] High PEs is 6 or more PEs among men, 2 or more for women. Low PEs is <6 PEs among men, <2 for women

P-values <0.05 are statistically significant. CI=confidence interval, SE=standard error

Supplementary Table 3. 21 Descriptive Statistics by Race and Police Encounters (PEs) Exposure Level at Add Health Wave III for Variables in the Framingham 30-year Cardiovascular Risk Scores at Wave IV (n=8,447).

	Total	**Non-Hispanic Black**	**Non-Hispanic White**	***P-value*[b]**	**High PEs[c]**	**Low PEs[c]**	***P-value*[b]**
	n=8,447	**n=2,268**	**n=6,179**		**n=332**	**n=8,115**	
Age, years, mean (SE)	28.2 (0.13)	28.4 (0.16)	28.2 (0.14)	0.2645	27.8 (0.19)	28.3 (0.12)	0.0004
Sex[d], n (%)							
Male	3,734 (48.4)	904 (46.0)	2,830 (48.9)	0.1550	149 (50.7)	3,585 (48.3)	0.5817
Female	4,713 (51.6)	1,364 (54.0)	3,349 (51.1)		183 (49.3)	4,530 (51.7)	
Taking Hypertension Medications, n (%)							
Yes	303 (3.8)	87 (4.4)	216 (3.7)	0.1703	7 (2.0)	296 (3.9)	0.2266
Systolic Blood Pressure, mm Hg, mean (SE)	125.0 (0.23)	126.6 (0.43)	124.6 (0.25)	<0.0001	124.1 (0.94)	125.0 (0.24)	0.3638
Diabetic, n (%)							
Yes	545 (5.8)	285 (13.8)	260 (4.1)	<0.0001	16 (3.5)	529 (5.9)	0.1431
Smoked in Last 30 Days, n (%)							
Yes	3,068 (39.2)	642 (31.7)	2,426 (40.9)	0.0015	185 (57.9)	2,883 (38.4)	<0.0001
Body Mass Index, mean (SE)	28.9 (0.15)	30.7 (0.25)	28.5 (0.17)	<0.0001	27.3 (0.46)	29.0 (0.15)	0.0005

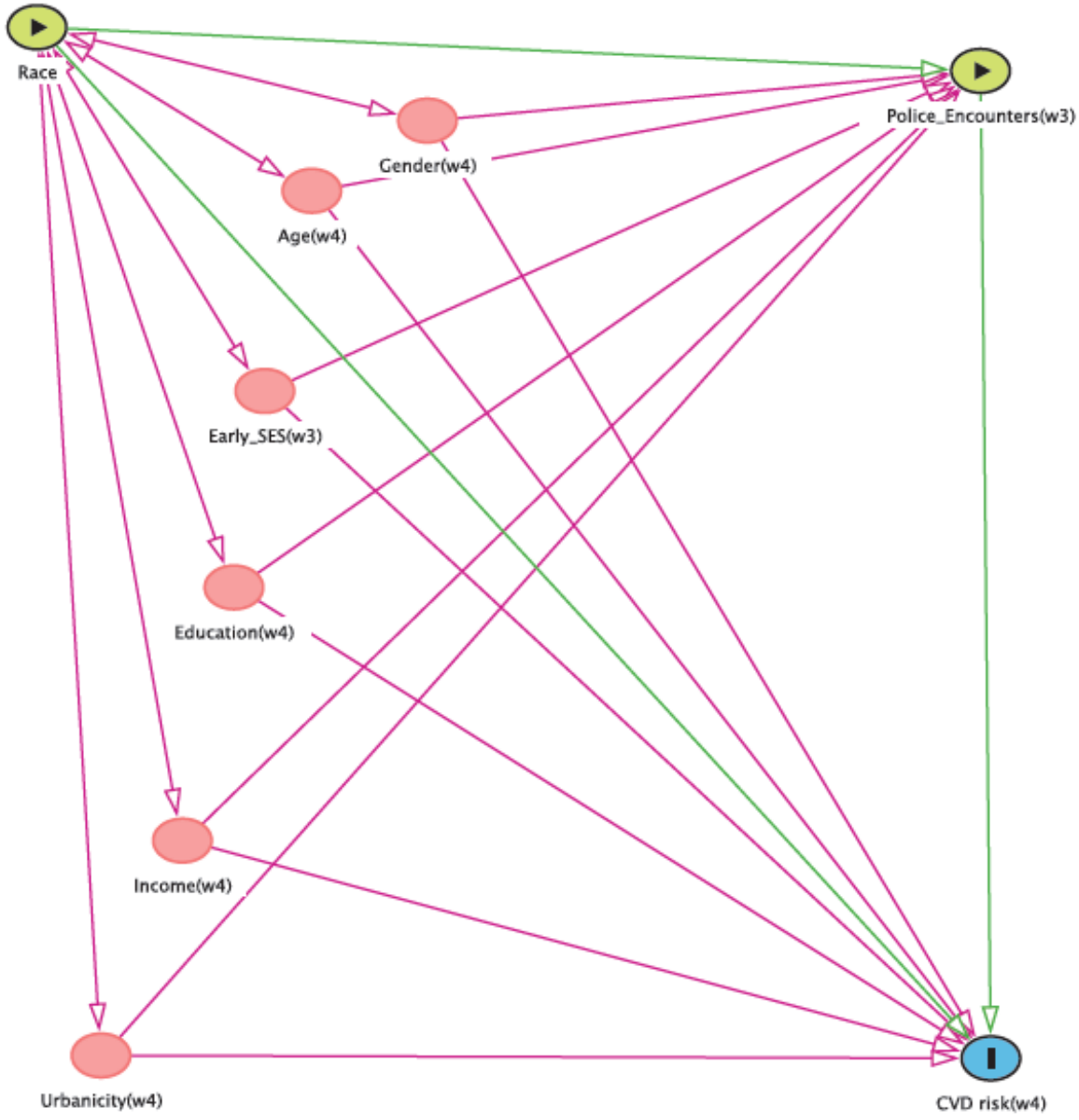
[a] See Pencina et. al., 2009 for methods

[b] *P-values* generated with chi-square for differences in weighted percents and t-tests for difference in weighted group means

[c] High PEs is 6 or more PEs among men, 2 or more for women. Low PEs is <6 PEs among men, <2 for women

[d] Based on Sex reported at Wave I

Figure 3. 1 Directed Acyclic Graph (DAG) of the Hypothesized Relationship Between Police Encounters and 30-Year CVD Risk Score.



Chapter 4 Community-Level Structural Racism and Individual 30-Year Cardiovascular Risk in the United States: Residential and School Racial Segregation

"All persons shall be entitled to the full and equal enjoyment of the goods, services, facilities, privileges, advantages, and accommodations of any place of public accommodation, as defined in this section without discrimination or segregation on the ground of race, color, religion, or national origin."

~ Title II, Sec. 201 (a), Civil Rights Act, 1964

4.1 Background

Despite substantial declines in cardiovascular disease (CVD) mortality rates over time for all racial and ethnic groups in the United States (US), disparities have widened (Figure 4.1). In 1950, the Black – White CVD mortality rate ratio was 1.0, indicating that Blacks died at a rate equal to that of Whites (Figure 4.1). Although the rates for both Blacks and Whites have declined over time, the mortality gap between Blacks and Whites has increased, with a Black-White racial disparity in CVD mortality of 1.3 in 2016.[483] The Black – White racial disparities in CVD mortality resulted in approximately 19,448 excess deaths in 2009.[3] CVD racial disparities are larger in some large cities while smaller in others. For example, Washington, D.C. had the largest racial disparity in CVD (rate ratio=1.90) while Tuscon, AZ had the lowest (rate ratio: 0.86).[3] CVD mortality disparities that widely vary across cities provide some evidence that disparities in CVD are not related solely to some biological reason. Indeed, these disparities hold steady even after accounting for individual factors such as age, gender and socioeconomic status, and traditional risk factors like diabetes, smoking, and hypertension.[500,573] Given that mortality is a marker of the health of a nation and its citizens, identifying risk factors for mortality and premature mortality outcomes provide opportunities for interventions at the population level. Many researchers have argued that major factors affecting the health and well-being of racial minorities are structural racism and discrimination.[7,10,11]

Here I will focus on structural racism and resulting discrimination in housing. More specifically, segregation is the result of several US policies and court rulings such as: Plessy v Ferguson or Jim Crow (1896), Corrigan v Buckley (1926), the Home Owner's Loan Act (1934), Shelley v Kraemer (1948), the Fair Housing Act (1968) which ended legal segregation in housing, and the Community Reinvestment Act (1977) which ended mortgage discrimination. Many of the overtly racist policies have been eliminated (e.g., slavery, Jim Crow, redlining), while others, through color-blind wording, continue to victimize racial and ethnic minorities (e.g., disenfranchisement policies, residential zoning, and New Deal economic policies that exclude certain professions from benefits) deeming the construct of structural racism extremely difficult to measure.[35,36,235]

4.1.a. Housing Policies and Residential Segregation

US housing policies have resulted in residential segregation, income inequality, and segregation in education limiting opportunity for Blacks while privileging Whites. Jim Crow was a form of segregation, not only with housing but also with other public and private businesses and services, that was made legal in 1896 by the Supreme Court Case of *Plessey v Ferguson*.[122] While this ruling made segregation legal in the US, it was not honored in Northern states. Thus, the North created segregation in a different way.

Local communities were solidifying and protecting the whiteness of certain communities through specialized zoning policies, such as prohibiting rental properties and racially patterned commercial property zones.[36] Zoning laws have been used as a means to racially segregate communities, despite zoning based on race alone being deemed unconstitutional in the *Buchanan v Warley* Supreme Court ruling in 1917.[257] Rules defining certain properties for commercial versus personal use, or limiting or banning rental properties in communities, ensures that lower income individuals are not welcome in a community, thereby maintaining residential segregation through color-blind means.[36] This practice is still used today.

Another common tool used to segregate communities was racially restrictive covenants, or language written into the deeds of properties. The language in contracts abided by through the sale process to ensure the property values would grow, as there was a strong belief that having an

integrated neighborhood would reduce the value of one's properties.[36] While this policy was deemed unconstitutional in 1948 many property deeds contained the racist language that indicated what the future buyer *could* look like.[36,264] Being unconstitutional, these racially restrictive deeds were made *de facto*, or followed by custom, and they were often followed, until the Fair Housing Act of 1968 and the Community Reinvestment Act of 1977 together banned the discriminatory practice of redlining.[265,271]

The US government took an active role in producing segregation that extended far beyond the Jim Crow South. Much of the literature examining structural level factors leading to residential segregation is attributed to the federal policy nicknamed "redlining." As a means to stimulate the depressed economy after the Great Depression, politicians created federally backed mortgages as a way to build wealth through homeownership. Many of the New Deal policies also subtly reflected the racist view of the time period by incorporating color-blind language that differentially affected Blacks in the US, a departure from Reconstruction Era Jim Crow laws which were explicitly racist.[36,235] In 1934, the US Congress passed the Home Owner's Loan Act, a color-blind policy as written.[574] The execution of the act, however, formed legal segregation based on race or redlining using explicit language about race and socioeconomic status that affected communities nationwide.[121,575] This 1934 New Deal policy allowed the banks to deny mortgages to Blacks by using maps to determine areas where the residents were at high risk for defaulting on repayment of the loan. High risk areas were coded as red, and nearly all predominantly segregated Black communities were coded red.[36] Redlining made banks much less likely to provide a mortgage to families both living in and wishing to purchase homes in redlined areas.[120,121]

Additionally, realtors capitalized on these segregation laws and the belief that Blacks moving into White neighborhoods would threaten property values and thus used tricks like "Blockbusting" as a custom, where Black women were hired to push baby carriages to signal that the neighborhood was changing. Realtors then benefitted from both the purchases and sales of these homes.[36]

A few tangentially related policies also affected how Americans lived and interacted with one another. One example is the National Securities Recourses Act of 1947, which pressured major

companies to move out of urban centers to reduce the chance of being targets of nuclear attack. This moved decent jobs out of cities. Additionally, the Federal-Aid Highway Act of 1944 created the US highway system, which linked cities and smaller towns, while also producing "slum-clearance" in which predominantly over-crowded Black communities were labeled as "slums" and selected for demolition to build highways and other infrastructure projects, further segregating communities, by building literal barriers between people who were already segregated.[36] Additionally, as US cities became overcrowded, suburban areas were built up. After WWII, the GI Bill (Servicemen's Readjustment Act 1944) aimed to help those who fought in the war to return to improved housing access, educational attainment, and better financial possibility. This bill favored Whites, not because it was a racist bill, per se, but rather because the laws and distribution of the associated resources that intersected with the GI Bill were racist. For example, Black GIs could not access mortgages because of discriminatory lending policies; nor could they take advantage of the educational benefit as most US colleges did not admit Blacks.[36] The point of this history recap is that racism is cyclical, meaning, as one form of racism declines or is outlawed, it is replaced by another form, over and over again, consistent with the Fundamental Causes Theory which suggests that we need to eliminate the fundamental cause (i.e., racism) before the cycle has a chance to end.[6]

4.1.b School Segregation

While segregated communities are the result of both legal and *de facto* policies, segregated neighborhoods produce segregated schools.[36] With the Supreme Court ruling of *Brown v Board of Education of Topeka, Kansas* (1955) the segregated schools in the South were ordered to desegregate, but the ruling made no mention of schools in the North, as such schools and communities in the North remained more segregated than those in the South.[36,239,240] Additionally, the 1964 Civil Rights Act did not further improve school segregation, as a portion of this law did not include means to reduce school segregation. For example, the Civil Rights Act stated that schools could not fund bussing or a means to bring racial and ethnic minority students to predominately White schools.[116,157]

In recent years, despite major educational improvements for Blacks in the US, the Supreme Court has loosened the oversight that was in place to ensure that schools integrated, and schools began re-segregating, quality of education declined, and some suggest that educational quality is

worse than it was prior to the Brown decision.[239] According to the American Community Survey 5-year estimates (2011-2015), there is substantial state-level variation in racial disparities in educational attainment where in Idaho there is virtually no difference in the percentages of Blacks and Whites with college degrees or higher, whereas in Washington, DC, Whites are 3.4 times more likely to attain a bachelor's degree or higher than Blacks. Thus far, there has been little research regarding racial segregation in schools. Dudovitz et. al., 2021 identified a relationship between school segregation, measured by the percent of White students in a school, and depression, substance use, and lower self-rated health later life.[576] Many of the outcomes examined in the Dudovitz et. al. study are associated with CVD.[576]

4.1.c. Discrimination and Health

Racism research has largely focused on interpersonal discrimination, or overt acts of racism that are highly visible, but are dependent on variable perceptions of the interactions. This body of literature suggests that interpersonal discrimination is detrimental to health.[7,10,17,18] For example, a recent meta-analysis of nearly 300 studies between 1983 and 2013 reported that there were significant effects of interpersonal discrimination for both mental and physical health, with a stronger association for mental health.[18] Research has demonstrated that discrimination is a psychosocial stressor that can lead to unhealthy behaviors, internalized racism, and physiological responses that can lead to both poor mental and physical health.[10,56] Essentially, racism gets absorbed into the body from the environment and results in a biological response (i.e., embodiment).[7,8] Despite the overwhelming evidence that discrimination is harmful to health, discrimination is a measurement of individually perceived racism. Thus, an individual must experience a racist encounter and perceive that the encounter is racially discriminatory (i.e., micro-level interpersonal discrimination). Additionally, the standard measures of interpersonal racism do not capture the macro-forces that are more common because they are part of everyday life in the US.[15,16] In a society where overt discrimination is largely deemed unacceptable,[12] health researchers must consider other methods to understand the effects of macro-level discrimination born out of legal, or *de jure,* and customary, *or de facto,* often subtle discrimination that are written into our laws, policies, and customs.[35,36,235,239]

Phelan and colleagues (2015) reported that macro-level racism, referred to as structural racism, is a "Fundamental Cause" that limits opportunities for racial minorities to access resources, such as

knowledge, power, social connections, goods, and services that can be used to avoid risks for poor health.[6] Understanding the effects of structural racism is an emerging area of study in public health and thus more work is needed to understand what outcomes are most affected by this macro form of racism, herein referred to as structural racism.

Structural racism is defined as a structure or barrier that is created by policies, laws or customs that produce racially disparate outcomes where racial minorities are negatively affected and Whites are privileged.[6,7,10,12,13,15,16,44] Some policies were created with discriminatory intent and produced structures which explicitly excluded or isolated racial and ethnic minority groups. Some examples of this are Jim Crow (1896-1964), which legalized segregation based on race in many US states, as well as the Home Owners Loan Act of 1933, which legalized discriminatory mortgage lending nationwide.[120-122,574] Some policies are not explicitly discriminatory or racist; however, they produce racist structures and thus a racially "disparate impact" or policy outcomes that differ by race (e.g., educational attainment, incarceration, or homeownership). These policies are generally crafted in a way that is "color-blind" or race neutral but produce a racially disparate outcome. An example of a color-blind policy with a discriminatory effect is the War on Drugs. This "war," which began in the 1960's, has persisted to the present with "tough on crime" race-neutral policies and court rulings, including mandatory minimum sentencing, stop-and-frisk, and felony disenfranchisement.[35] These policies and rulings are responsible for the increased rates of incarceration, of which a higher proportion are among Black men.[35] For example, the mandatory minimum sentencing laws are written so that they are about the type of drug and not about those who uses it (e.g., rock cocaine versus powder cocaine). However, rock cocaine (crack) was predominantly used by Blacks whereas powder cocaine was predominantly used by Whites.[577] Despite the drug being chemically the same, there have been different sentencing penalties imposed on these similar drugs, with harsher sentencing for rock cocaine or crack.[35] Thus, color-blind "War on Drugs" policy skirts around race as written. Yet it has successfully built a prison industry that disproportionality locks-up People of Color.

4.1.d. Residential Segregation and Health

Residential segregation research is measuring the downstream effects of segregation policies, proxy measures of structural racism, or disparate impact of policies. Additionally, oftentimes structural racism is conceptualized or defined in the study, however the reference to a specific

policy that caused the disparate impact under study is only tangentially referenced, if mentioned at all.[91,578] Why does this matter? If a disparate impact cannot be traced back to a policy or if the researchers do not understand the history of that disparate impact, how can we begin to dismantle it? Or how can the results be attributed or linked to structural racism? Research provides a set of foundational information needed to couple with the historic context of policies and court rulings. However, understanding how or why that policy was formed, the effects of the policy, and the effects those policies have on health can help us begin to identify ways to either change the policy more thoughtfully or eliminate it altogether.

Segregation literature suggests that multiple measures may be needed to understand the impact of residential and school segregation.[118,266] Some researchers have begun combining redlining maps from the 1930s, current Home Mortgage Disclosure Act data (mortgage amounts or denials by race), and health outcomes. These studies have observed that there are strong spatial associations with past residential segregation, present residential segregation and, thus, available community resources and health outcomes.[68,96-100] A novel measure that has been seen more frequently in the literature is the Index of Concentration of Extremes (ICE). Social epidemiologists and other social science researchers have been using the ICE measure to reflect the concentration of privilege or disadvantage based on race/ethnicity, economic issues, and their intersection to better understand the causes of health disparities.[177,178,521,579] This small, but growing, body of literature suggests that ICE for race and/or ICE for income, or higher level of racial concentration in an area, is associated with hypertension[177], mortality (premature, childhood, and cause specific mortality including CVD)[579], and fatal and non-fatal assaults.[178] While seemingly unrelated, residential segregation leads to segregated local schools.[36]

Despite the issues noted above, the residential segregation and health literature has overwhelmingly suggested that residential segregation is associated with adverse birth outcomes[87,88,90,91,580-594], cancer outcomes[595-597], HIV mortality[598], both fatal and non-fatal cardiovascular outcomes[126,507], hypertension[177], survival[72], fatal police shootings[89], and community violence.[178,599,600] This large body of work suggests that the effects of residential segregation are harmful to the health of both Black and White Americans, however this is not always the case.[595,601] For example, Warner and colleagues report a higher breast cancer survival

among Blacks women when they reside in a community with a higher concentration of Black residents, but lower survival for Whites under the same community racial composition.[595]

4.1.e. Policies and Practices that Create, Reinforce, or Perpetuate Racism

A common misconception is that discrimination is solely based on racist acts of racist individuals, and only people involved with a particular exchange are impacted. However, the last several years notwithstanding (2016-2020), research has suggested that overt interpersonal forms of discrimination are decreasing and have been replaced with more subtle forms of discrimination and implicit biases.[10] For example, Williams and Mohammed (2013) report that since 1964 there has been increasing support among Whites for the government's work place anti-discrimination efforts. While there is a general decreasing trend of overt racism, there is an increased interest in examining implicit/unconscious bias. Review studies report that about 70% of Americans have implicit biases favoring Whites over Blacks.[10] Despite this declining trend, racial disparities in many health outcomes persist and are not explained completely by disease risk factors, behaviors, or socioeconomic status.[10]

The importance of housing policy, and thus residential segregation, cannot not be understated. As noted earlier segregation is associated with a myriad of health outcomes.[94] Home-ownership is also a path to wealth accumulation and is a major source of the large wealth gap between Blacks and Whites in the US.[36,457] Furthermore, residential segregation also leads to segregation in other areas such as education.[239,602] Educational segregation is commensurate with residential segregation because where people live dictates where they or their children learn. This was certainly understood with the Supreme Court ruling of Brown *v* Board of Education of Topeka, Kansas which began the desegregation of schools, a policy created through Plessy *v* Ferguson or "Separate but Equal."[239,240,456] There were other Supreme Court cases that furthered the desegregation process by restricting "Freedom of Choice" student assignment plans, mandating strict racial quotas, bussing, and court-ordered oversight.[239,242,603] Scholars suggest that these were effective methods to desegregate schools, improving the education of racial minorities while also improving education quality for Whites.[239,457] However in the early 1990's the Supreme Court ruled to release school districts from oversight, and the schools quickly began to re-segregate.[239]

Although there are racial disparities in educational achievement favoring Whites, especially at the higher education level, many argue that differential quality of education is driving this disparity, at least partially.[555,604] Additionally, residential segregation creates concentrated poverty and as a result, schools are segregated by both race and poverty status, which can prevent educators from providing a quality education because teachers then must focus on issues in the classroom such as hunger, fear, stress, crime, behavioral issues, and neglect.[36] This differential quality of basic education results in fewer racial minorities being able to attend college thereby creating another barrier to economic success.[36]

Measuring the policy implicated in producing a racial disparity is challenging, however the downstream effects or disparate impacts that can be traced back to policies can be measured. For example, residential segregation did not occur naturally; as noted earlier it resulted from several local zoning ordinances, racially restrictive covenants though language in deeds, racist federal mortgage lending laws, and a few tangentially related policies that built up the suburban areas due to urban overcrowding (e.g., National Securities Resource Act of 1947, and Federal-Aid Highway Act 1944).[36,120,575] More specifically, the downstream consequences of racist policies that limit access to decent housing for Blacks and other racial and ethnic minorities while providing freedom to live where they choose to Whites has been a mainstay for most of the history of the US.[36] While most studies examining structural racism through housing cite redlining as the policy that produced segregated communities in the US, it is not the only one.

Additionally, policies, laws and customs have lasting effects. Historic policies that target racial and ethnic minorities have morphed into color-blind policies that, through very specific language, have resulted in persistent racial disparities in health outcomes but also in access to resources including informational, financial, and network related resources.[6,10,12,16,35,36,235,239,457] This study will focus on residential and school segregation, which is a policy area that has substantially shaped the lives of racial minorities in the US for generations. This policy area has had consequences in other sectors as well, including employment, education and economic, among others.

4.1.f. Hypotheses

The present study will examine racial differences in the association between Census tract-level racial residential segregation and school-level racial segregation on individual-level 30-year CVD risk among US Blacks and Whites. The study uses data from the National Longitudinal Study of Adolescent to Adult Health (Add Health). I hypothesize that the association between community-level and/or school-level segregation at Wave I when respondents were between 7th and 12th grade and 30-year CVD risk will differ between Blacks and Whites at Wave IV when respondents were between 24 and 23 years old. I hypothesize that among Blacks, residing in communities and attending selected schools with higher racial segregation will be associated with an increased 30-year CVD risk score, whereas among Whites, residing in communities and attending schools with higher levels of segregation will be not be associated with an increased 30-year CVD risk score. This hypothesis stems from the very definition of structural racism where policies, norms and customs create barriers to progress for Blacks and other racial and ethnic minorities while simultaneously privileging Whites and is consistent with prior research.[6,7,10,12,13,15,16,44,77,177,579]

This study fills a void in the literature by focusing on 30-year CVD risk at a point in time for the respondents where an intervention could make a difference in the development of CVD. The only study that has examined the role of structural racism on CVD examined associations between state-level structural racism and reporting a non-fatal MI in the past year.[77] Other studies have examined some diagnosed risk factors associated with CVD such as hypertension[177], obesity[70,605], and diabetes[606] as well as outcomes of CVD such as premature mortality[92] and CVD mortality[507]. The present study builds on this research by adding additional dimensions of structural racism through residential and school segregation, examining the associations in a nationally representative longitudinal study, and includes predicted risk for both CVD mortality and other cardiovascular diseases.

4.2 Methods

The design of this study is a population-based, multi-level, cross-sectional study using nationally representative Add Health data. The main exposures are residential and school segregation, which are the result of racist policies.[36] The operationalization of structural racism is through the Census tract-level measures related to segregation (index of dissimilarity, the isolation index, index of the concentration of extremes for race) and school segregation using the index of

concentration of extremes based on race in schools. The outcome of this study is the Framingham 30-year CVD risk score.

4.2.a. Add Health Data

The primary source of data for this chapter is the Add Health dataset. The Add Health methodology has been extensively published elsewhere.[529-531] Briefly, Add Health is a nationally representative school-based prospective cohort study of adolescents that examines behavioral, emotional, social, educational, and contextual factors as respondents transition to adulthood. The baseline sample was gathered beginning in 1995 when participants were in 7^{th} – 12^{th} grade using a complex clustered sampling design. The initial sampling frame was based on a list of schools from the Quality of Education Data, Inc. Eighty high schools and their accompanying feeder middle schools were selected using probability proportionate to size. Students in selected schools were stratified based on grade and sex, and 17 students within each stratum were selected with additional samples taken for ethnic minorities, Black children with at least one parent with a college degree, students with disabilities, and siblings. Wave I included 20,745 adolescents that were representative of American adolescents with respect to region, urbanicity, school features, and ethnicity. Subsequent Waves II-IV followed up with those who completed Wave I. Field interviews were gathered in the participants' homes using Computer Assisted Personal Interviewing (CAPI) for non-sensitive questions or Audio Computer Assisted Self-Interview (ACASI) technology for sensitive questions. Topics included demographics, socioeconomic status, physical health, and risk behaviors. In addition to data collected through interviews, objective measurements (i.e., blood pressure, measured height and weight, etc.) were also taken after the interview using systematic data collection protocols.

Data for this study are from the In-School survey, the Wave I survey, the Wave I contextual data file, and the Wave IV survey. Residential segregation is measured at Wave I while school-level racial segregation is measured before Wave I as part of the primary sampling frames of all students in selected schools. The outcome was measured at Wave IV, when the cohort was between 24 and 32 years old. By Wave IV, personal habits and chronic diseases are beginning to affect Add Health participants. Eighty percent of respondents who completed Wave I completed the Wave IV survey.

4.2.b. Community-Level Measurement of Structural Racism

The main exposure in this study is community-level structural racism, defined as racial residential segregation and racial school segregation. Each exposure measure is described below (Table 4.1).

Residential segregation is defined in three ways: First is the Index of Dissimilarity (IoD), a widely used measure of the percentage of a group's population that would have to change residence in a geographic location to achieve an equitable racial composition.

The formula for the Index of Dissimilarity (IoD)[178] is as follows:

$$(1)\ \text{IoD} = \left[0.5\ \sum_{i=1}^{n} \left|\left(\frac{b_i}{B_i}\right) - \left(\frac{w_i}{W_i}\right)\right|\right]*100$$

Where, i=block group within a Census tract; b_i=number of Blacks in the block group, B_i=number of Blacks in the Census tract, w_i=number Whites in block group, and W_i=number of Whites in the Census tract. In the event that there are 0 Black or White people in a Census tract, 1 was substituted for the 0 (i.e., in the B_i and/or W_i position in equation (1)).

The second measure of residential segregation is the Index of Isolation (IoI) which measures the extent to which minority members are exposed to one another within a Census tract.[118,266] The IoI values range from 0-1, where higher values indicate higher levels of segregation.

The formula for the Index of Isolation (IoI)[118] is as follows:

$$(2)\ \text{IoI} = \sum_{i=1}^{n} \left[\frac{x_i}{X}\right]\left[\frac{x_i}{t_i}\right]$$

Where, i=block group within the Census tract, x_i=number of Blacks in the block group i, X=number of Blacks in the Census tract, t_i=number of people in the block group i.

Both the IoD and the IoI are derived from variables available in the Add Health Wave I contextual data file, which was merged with the final dataset available to the public through an extensive data use agreement process. The contextual data used in this study is from the US Census of Population and Housing from 1990 using the Summary Tape File 3A. The contextual

data was calculated by the Add Health team and provided to the public as de-identified data at the Census tract and Block Group level.

The final residential segregation measure is called the Index of Concentration of Extremes (ICE) which measures the extent to which a community is over-populated by extremes in racial concentration.

The general formula for the Index of Concentration of Extremes (ICE)[177,178] is as follows:

$$(3)\ ICE = \frac{(Number\ of\ Whites\ in\ an\ area) - (Number\ of\ Blacks\ in\ an\ area)}{Total\ Population\ in\ the\ Area\ with\ Information\ on\ race}$$

ICE is further defined by race where the most privileged was defined as non-Hispanic Whites, the least privileged was defined as non-Hispanic Blacks, and the denominator reflects those from which race data is available for all races. The "area" is defined in two ways: Census tract and school.

To calculate ICE_{tract}, counts of total population and percent of the tract that were Black, White, Latinx/Hispanic, and other race was provided in the contextual dataset at Wave I. The numbers of non-Hispanic Black and Non-Hispanic White people were calculated using the following steps:

a. The tract population count was multiplied by percent Black, percent Latinx/Hispanic, and percent White to obtain the number of Black, Latinx/Hispanic, and White persons in the tract, respectively.
b. The number of Latinx/Hispanic persons in the tract was subtracted from the number of Blacks in the tract to calculate the number of non-Hispanic Blacks in the Tract. Similarly, the number of Latinx/Hispanic persons were subtracted from the number of Whites to calculate the numbers of non-Hispanic Whites in the tract.
c. These race and ethnicity specific population counts were inserted into the ICE equation (3) to calculate the ICE value for each tract.
d. Finally, the ICE values were remerged into the individual data so that each individual had the tract level ICE value for their respective tract.

The final ICE value ranges from -1 to 1, where negative numbers indicate a higher concentration of Blacks and a positive number indicates a higher concentration of the Whites. Numbers closer to -1 or +1 indicate a higher level of segregation by racial concentration.

The second ICE value was generated using the populations at the schools using the initial sampling frame for the individuals that were selected into the Add Health study. The ICE_{school} variable was calculated similarly to the ICE_{tract} with the steps noted earlier. The in-school survey served as the primary sampling frame for the individuals who ultimately were sampled for the study. All students in selected schools were provided a survey which was conducted during a school day (n=90,118). This survey included questions about demographics and eligibility for the primary and ancillary Add Health studies. For the purpose of this study, only data on student's race and ethnicity was used. In the school data, race and ethnicity were collected as a series of binary (yes/no) questions including Black, White, Latinx/Hispanic and other races. In order to get a count of students by race for each school, an overall race variable was created by combining all race and ethnicity questions provided into one variable where those reporting Latinx/Hispanic were coded in one category (regardless of reported race), then those reporting their race as Black as a second category, and those reporting a race other than White as another category, and finally those reporting White as a final category. This created a race/ethnicity variable of Latinx/Hispanic, non-Hispanic Black, non-Hispanic other race, and non-Hispanic White groups where each student included only one race value. Then each racial group was further recoded into binary (0, 1) where if a student reported a race non-Hispanic Black or non-Hispanic White they were coded as 1. These binary race variables were summed up to the school level to calculate the number of non-Hispanic Black and non-Hispanic White students within each school. The total number of students per school was the sample size for the school. The denominator included all students in the school with non-missing information on race (n=2,760 had missing data on race in the school survey).

4.2.c. Individual Level Framingham Cardiovascular Risk Score

The outcome for this study is the Framingham 30-year Cardiovascular Risk Score (CVD) calculated from variables collected at Wave IV. The risk score predicts the risk of CVD events occurring within the next 30 years accounting for specific characteristics of the individual. CVD risk scores are useful clinical tools that provide a patient with a general risk for CVD events

assuming nothing changes. A CVD risk score can be considered a point of intervention where those with high risk scores can be targeted for therapeutic or behavioral interventions that can reduce a patient's risk for fatal or non-fatal CVD event.[490,491] The SAS macro code used to generate the individual risk score was provided by Dr. Pencina and Mr. Williams from Duke University and Kenanco Biostatistics, respectively. The code provided included macros which predicted cardiovascular risk in 30 years using a Cox proportional hazards model that accounts for competing causes of death based on data from the Framingham Heart Study. The Cox model included the following covariates: age, sex, use of antihypertensive medications, smoking status, diabetes status, systolic blood pressure, and body mass index.

Using these variables from Add Health, after applying the Pencina and Williams SAS macro, the resulting CVD index is a continuous measure ranging from 0-100% and interpreted as a 30-year CVD risk level for several cardiovascular outcomes including: coronary death, myocardial infarction, fatal and non-fatal stroke, coronary insufficiency, angina pectoris, stroke plus transient ischemic attack, intermittent claudication, and congestive heart failure.[491,534,535] There are two 30-year CVD risk variables generated. The hard outcomes predicted by the CVD risk score are coronary death, myocardial infarction, and fatal and non-fatal stroke, while the full outcomes include those in the hard outcomes as well as coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure. See Appendix A for additional details on the 30-year CVD risk score.

4.2.d. Covariates

Covariates were selected *a priori* and are associated with both structural racism and 30-year CVD risk (Figure 4.2). The following individual covariates were included: age, sex, race, parental education and parental income. Additionally, the following tract-level hypothesized confounders were included in the models measured at Wave I (1994-1995) using the 1990 US Census data: the overall percent of those living below the federal poverty level, percent Latinx/Hispanic, percent Black, percent with less than a high school diploma, urbanicity, and income inequality. This study also examined race as an effect modifier to determine whether or not the relationship between residential or school segregation and CVD risk are dependent on race.

Several individual-level covariates have been included in the models. *Age* is based on the age of the respondent at Wave IV. This variable was provided in the Add Health dataset. *Sex* and *race* are self-reported at Wave I. Each respondent was allowed to report multiple races and/or ethnicities. Following the US Office of Management and Budget[607] which defined racial categories for the US Census[e], anyone reporting being Latinx/Hispanic was coded as Latinx/Hispanic regardless of what race they reported. Then, those reporting their race as Black were defined as non-Hispanic Black. Those reporting Asian, Native or Other race were coded as non-Hispanic other race. Finally, those reporting their race as White were defined as non-Hispanic White. Parental education and parental income were variables provided by the Add Health dataset and were based on responses from the parents at Wave I. Parental variables had many missing values. In order to maintain the sample size, an unknown category was included. Parental education was defined as the highest level of education for the respondent's most highly educated parent; categories include less than high school, high school graduate, college graduate, and unknown. Parental income was defined in the following categories <$20,000, $20,000-$39,999, $40,000-$74,999, $\geq$ $75,000, and unknown and is based on parental income at Wave I.

Hypothesized tract-level confounders include percent Latinx/Hispanic, percent Black, percent with less than a high school diploma, urbanicity, and the percent of residents below the federal poverty level (FPL) all measured at Wave I (1994-1995). The final hypothesized confounding variable is income inequality. Income inequality was defined as the ICE by tract level median household income (ICE_{Income}) as defined by Krieger et. al. and Feldman et. al.[177,178] ICE_{income} was calculated using income levels to determine financial privilege and similar methods as described in the $ICE_{race\ at\ tract}$ and $ICE_{race\ at\ school}$. In this formula, the most privileged group was defined as those with incomes at or above the 80th percentile of all incomes in 1990, which was $55,205, and the least privileged was defined as those with incomes at or below the 20th percentile of all incomes in 1990, which was $12,500.[f] These exact income values were not available in the Add Health dataset, thus those in the tract earning $50,000 or more were defined as the most privileged and those in the tract earning $15,000 or less were defined as the least privileged. These dollar amount values available in the Add Health contextual dataset were the

[e] https://www.census.gov/topics/population/race/about.html
[f] https://www2.census.gov/programs-surveys/cps/tables/time-series/historical-income-households/h01ar.xlsx

closest to those identified as the 80th and 20th percentiles of incomes in 1990. The denominator includes all persons in which income was available for the tract.

4.2.e. Analytic Sample

After combining all of the datasets and deleting those records that did not have a Wave I weight value, per the analytic guidelines, there were 12,888 individuals included in the dataset. Of this, there were 9,206 individuals who were non-Hispanic Black or non-Hispanic White. These racial groups were selected because they are the two predominant races that have been most affected by the legacy of slavery in the United States, a legacy that has contributed to the disadvantage of non-Hispanic Blacks relative to non-Hispanic Whites in the US. Finally, participants without complete data on all variables included were excluded from the analysis. The final sample for this study is n=6,835. The sample size difference is made up of those missing tract level information because their addresses could not be geocoded (0.9%), or missing race at the schools survey (22%), and the remaining is related to missing data on covariates or the outcome (4%). While these records were excluded from the analyses presented in this study, they were not deleted from the dataset. As such, a domain analysis was conducted per the recommended analytic guidelines provided by Add Health for analyzing subgroups.

4.2.f. Statistical Analysis

The Add Health data is a complex and clustered design. The well documented sampling methodology states that schools were the primary sampling units (clustering unit).[529,530] A survey was provided to each student within selected schools which served as the overall sampling frame for the primary Add Health longitudinal study noting that students are clustered in schools and neighborhoods. Since this study examines neighborhood- and school-level predictors of CVD risk, a general estimating equation (GEE) analytic strategy was employed to account for the clustering at the school-level to obtain a population average cardiovascular risk score.

The analysis begins with an examination of each variable and its associated distribution. Means and standard errors were examined for continuous variables and proportions were examined for categorical variables. Each continuous variable, including both outcomes, was assessed for a normal distribution by using histograms, box plots, q-q plots and scatter plots and their

associated indicators to establish normality. Both outcomes as calculated were substantially right skewed with long tails. After analyzing the data using regression models, the errors produced by both risk scores were non-normally distributed. Due to the non-normality of the errors in regression the outcomes were subsequently log transformed and their distributions were re-evaluated. In both cases the distributions of the risk scores and the evaluation of the error terms were normally distributed after log transformation. Bivariate associations were examined and reported in Table 4.2. Each variable was stratified by race. All variables included in the models (described below) are included in Table 4.2. To determine the statistical difference between the groups, the t-test was used to examine group means for continuous variables and the modified Rao-Scott chi-square test to examine difference in proportions.

To examine the population average effect racial segregation has on 30-year CVD risk a GEE analytic approach was used. GEE was performed using PROC GENMOD with an identity link (and normal distribution) and a repeated statement that accounts for clustering at the school level and strata (region) variables (also class variables). Grand sampling survey weights were employed to account for the complex survey design, non-response, and post-stratification. An exchangeable covariance matrix was assumed after comparing the QIC values produced with an independent correlation structure, noting that the unstructured correlation structure would not converge. GEE is well suited for this research question which focuses on a population average CVD risk and results in robust Huber-White sandwich parameter and standard error estimators which provides forgiving estimates even if the correlation structure is misspecified.[608]

Interactions were incorporated into the model to assess differences in the association between segregation and CVD risk by race. Statistical significance was estimated using a Wald test that approximates Z-statistics to determine if mean 30-year CVD risk score statistically differs from 0. The SAS GENMOD procedure does not have an option to produce a domain analysis, thus for those participants who reported their race as something other than non-Hispanic Black or non-Hispanic White the grand sampling weight was recoded to 0.0001 per the analytic instructions.[530] This allows for the complex structure of the data to remain intact, while also excluding the groups who are outside of the study population.

For the analytic strategy, the population average 30-year CVD risk score for a 1-unit increase in residential segregation with only race included in the model was calculated (Model 1). Then, the individual and area-level covariates were added into Model 2. Then to determine if the association between segregation and population average 30-year CVD risk score varied by race, an interaction between the structural racism exposure and race was added (Model 3) using the following models:

(1) $$Y_{ij} = \beta_0 + \beta_{1j} community\ SR + \beta_{2i} race + \varepsilon_{ij}$$

(2) $$Yj_i = \beta_0 + \beta_{1j} community\ SR + \beta_{2i} race + \beta_{3i} individual\ level\ covariates + \beta_{4j} community\ level\ covariates + \varepsilon_{ij}$$

(3) $$Y_{ij} = \beta_0 + \beta_{1ij} community\ SR + \beta_{2i} Race + \beta_{3i} individual\ level\ covariates + \beta_{4ij} community\ level\ covariates + \beta_{5ij} race * community\ SR + \varepsilon_i$$

In these models, Y_{ij} represents the population average value of the log transformed 30-year CVD risk score predicting both full and hard outcomes for a 1-unit increase in structural racism defined as residential segregation (i.e., IoD, IoI, ICE_{tract}, and ICE_{school}), where i represents the individual. Community structural racism is defined as the three residential and one school segregation measures, each modeled separately, and j represents the Census tract or the school. Race is limited to those reporting non-Hispanic Black or non-Hispanic White. The individual level covariates include: age at Wave IV, sex reported at Wave I, parental income and parental education reported at Wave I. The community level covariates include percent of residents in the Census tract who are Latinx/Hispanic, Black, less than high school educated, living below the federal poverty level, and residing in an urbanized area, as well as a measure of income inequality at the tract level all measured at Wave I.

All analyses are conducted using survey packages available in SAS 9.4 including survey cluster, strata and weight variables as directed by Add Health analytical guidelines.[530] Interpretation of the log transformed variables can be examined by taking the anti-log of the coefficients produced in the models, which result in a geometric mean (similar to a median) value for that variable. The interpretation for the anti-log of the interaction term is the increased or decreased risk using the following equation $[(\exp^{coefficient})-1]*100$, where an anti-log >1 suggests an increased 30-year

CVD risk and an anti-log <1 suggests a decreased risk. Statistical significance was determined at the p=0.05 level.

4.2.g. Checking Regression Assumptions

Similar to linear regression analysis, the standard assumptions of the regression were examined: independence of errors, homoscedasticity, linearity, and a normal distribution of the residuals. Since Add Health data is clustered in nature the linear regression assumption of independence is violated in clustered data. Additionally, the exposure is at the tract or school level. As such, a GEE analytic strategy allows for accounting for both the group-level exposure and the clustering. The assumption of linearity was assessed by examining a scatterplot of residual versus each continuous variable in the model. Finally, a q-q plot was used to examine the assumption of normal residuals. There is not a standard goodness of fit value provided as GEE uses a quasi-likelihood, but rather the QIC was used to examine the proper assumed correlation structure. Additionally, the model-based and the empirical-based standard errors for both the exchangeable and independent correlation structures were examined to ensure a proper selection of a correlation structure. While the GEE model is a forgiving model to a misspecified correlation structure, it is still more efficient for the correlation structure to closely approximate the unknown population correlation structure. Influential points were examined by generating the Cook's distance, leverage, and the influence of observations on parameter estimates.

4.3 Results

Table 4.2 displays the demographic characteristics of the study sample overall and by race. There are differences in the sex distribution by race where among Blacks there is a higher proportion of females than males and among Whites, there is a nearly equal proportion for males to females. (p=0.0099). A high proportion of respondents with parents had at least a high school diploma (35% overall). Whites have higher incomes compared to Blacks. Blacks have higher unadjusted cardiovascular disease risk compared to Whites (p=0.0026 for the full CVD outcome, and p=0.0018 for the hard CVD outcome). Finally, Blacks generally reside in communities with higher segregation based on the IoI, the ICE measures for residential, school, and income as well as percent Black in tract, while Whites reside in more segregated communities based on the index of dissimilarity. The mean IoD was 14% with a range of 0 to 89% and the mean value for the IoI was 9% with a range of 0 to 100%. The ICE_{tract} was 0.53 which indicates that there is a

high level of racial segregation in the communities represented (i.e., positive values of ICE indicate a higher concentration of White residents relative to Black residents whereas negative values indicate a higher concentration of Black residents relative to White residents). This is similar for the ICE_{school} measure. The average income inequality was -0.026 which indicates that there was more income disadvantage relative to income advantage in the tracts represented in the Add Health study. Finally, Table 4.3 displays the means of area level variables at the tract-level rather than at the individual level.

4.3.a. Full 30-Year CVD Risk

Tables 4.3 to 4.6 display the general estimating equation (GEE) coefficients by each indicator of residential segregation. In Table 4.4, Model 1 demonstrates that the log-30 Year full CVD risk is not associated with the IoD, but Blacks have a higher CVD risk compared to Whites (coefficient for race=0.0814, p=0.0033). After controlling for hypothesized confounders (Model 2), the IoD remains non-significant and the association between CVD risk and race is attenuated and no longer statistically significant. Additionally, the relationship between log 30-year CVD risk and IoD is not dependent on race (Model 3) as evidenced by the non-significant race*IoD interaction (coefficient for interaction=0.0137, p=0.9813). Table 4.5 displays the GEE model coefficients for the IoI. In Model 1, the IoI is marginally significant (coefficient for IoI=0.1100, p=0.0999) and race is not significant. After controlling for hypothesized confounders, the coefficients for both IoI and race were attenuated and not statistically significant (Model 2). Model 3 includes the race*IoI interaction, which is marginally significant (coefficient for interaction=-0.1632, p=0.0732). This interaction is graphically displayed in Figure 4.3, which demonstrates opposite directions of the association of IoI on the log 30-year full CVD risk score by race, where Blacks have an approximate 4% *lower* risk in 30-year CVD risk scores for each unit increase in IoI, while Whites have an approximate 13% *higher* risk in 30-year CVD risk scores for each unit increase in IoI.

The results for ICE measures are displayed in Tables 4.6 and 4.7. For the ICE_{tract} measure, Table 4.6 demonstrated that there were non-significant coefficients for ICE_{tract} before (Model 1) and after (Model 2) controlling for hypothesized confounders; however there was a significant race*ICE_{tract} interaction (Model 3 coefficient for interaction=0.1289, p=0.0181). This interaction is observable in Figure 4.4, noting that 30-Year CVD risk increases for both Blacks and Whites

as ICE_{tract} increases, but the increase is greater in Blacks than Whites. Indeed, Blacks have an approximate 18% *higher* risk in 30-year CVD risk scores for each unit increase in ICE_{tract}, while Whites have an approximate 4% *higher* risk in 30-year CVD risk scores for each unit increase in ICE_{tract}. In Model 1 (Table 4.7) the coefficient for ICE_{school} is not statistically significant, but the coefficient for race is positively associated with the Full 30-Year CVD risk score (coefficient for race=0.1008, p=0.0026) or Black respondents have a higher CVD risk score compared to Whites. After controlling for hypothesized confounding variables, the coefficient for ICE_{school} becomes negative but remains not statistically significant, while the coefficient for race is attenuated and becomes non-significant (Model 2). Model 3 reveals a statistically significant race*ICE_{school} interaction (coefficient for interaction=0.1575, p=0.0375). The interaction can be observed in Figure 4.5 where CVD risk increases for Blacks but decreases for Whites as the level of segregation in schools increases. Indeed, Blacks have an approximate 11% *higher* risk in 30-year CVD risk scores for each unit increase in ICE_{school}, while Whites have an approximate 5% *lower* risk in 30-year CVD risk scores for each unit increase in ICE_{school}.

4.3.b. Hard 30-Year CVD Risk

Tables 4.8 to 4.11 display the GEE coefficients for each indicator of residential segregation. In Table 4.7, Model 1 suggests that the log 30-year hard CVD risk score is not associated with the IoD, but Blacks have a higher CVD risk compared to Whites (coefficient for race=0.0963, p=0.0054). After controlling for hypothesized confounders (Model 2), the coefficient for IoD is attenuated and remains non-significant while the coefficient for race is attenuated and becomes non-significant. Additionally, the relationship between log 30-year hard CVD risk and IoD is not dependent on race (Model 3) as evidenced by the non-significant race*IoD interaction (coefficient for interaction=0.0036, p=0.9760). Table 4.9 displays the GEE model coefficients for the IoI. In Model 1 the coefficients for both IoI and race are not significant. After controlling for hypothesized confounders, the coefficients for both IoI and race remain not statistically significant (Model 2). Model 3 includes the race*IoI interaction, which is marginally significant (coefficient for interaction=-0.1937, p=0.0682). This interaction is graphically displayed in Figure 4.6, which demonstrates an association in opposite directions for the relationship between IoI and the log of the 30-year hard CVD risk score by race, where Blacks have an approximate 7% *lower* risk in 30-year hard CVD risk for each unit increase in

IoI, while Whites have an approximate 13% *higher* risk in 30-year hard CVD risk for each unit increase in IoI.

Results for the ICE measures are displayed in Tables 4.10 and 4.11 and Figures 4.7 and 4. 8. There were non-significant coefficients for ICE_{tract} and race before (Model 1) and after controlling for hypothesized confounders (Model 2), however there was a significant race*ICE_{tract} interaction (Table 4.10, coefficient for interaction=0.1540, p=0.0181). This interaction is observable in Figure 4.7, noting that 30-year CVD risk increases for both Blacks and Whites as ICE_{tract} increases, with greater increase in risk for Blacks relative to Whites. Indeed, Blacks have an approximate 19% *higher* risk in 30-year CVD risk scores for each unit increase in ICE_{tract}, while Whites have an approximate 2% higher risk in 30-year CVD risk scores for each unit increase in ICE_{tract}. Finally, in Model 1 (Table 4.11) the coefficient for ICE_{school} is not significant but the coefficient for race is (coefficient for race=0.1224, p=0.0032). After controlling for hypothesized confounding variables, the coefficient for race becomes non-significant (Model 2). Model 3 reveals a statistically significant race*ICE_{school} interaction (coefficient for interaction=0.1834, p=0.0491). The interaction can be observed in Figure 4.8 where CVD risk increases for Blacks but decreases for Whites with higher levels of segregation in schools. Indeed, Blacks have an approximate 13% *higher* risk in 30-year CVD risk scores for each unit increase in ICE_{school}, while Whites have an approximate 6% *lower* risk in 30-year CVD risk scores for each unit increase in ICE_{school}.

4.4 Discussion

This study examined the relationship between residential and school segregation as measures of structural racism stemming from a multitude of federal and local policies restricting Black homeownership, and 30-year CVD risk scores in a nationally representative sample of young adults. The primary hypothesis was that the 30-year CVD risk score would *increase* as segregation *increases* among Blacks, but there would be *no change* or *a reduction* in 30-year CVD risk as segregation *increases* for Whites. I examined three measures of residential segregation (i.e., IoD, IoI, ICEtract) and one measure of school segregation (ICEschool). IoD was not associated with 30-year CVD risk among Blacks or Whites. Contrary to expectations, increased segregation, as measured by IoI, was associated with *increased* CVD risk among Whites and *decreased* CVD risk among Blacks, although the interaction between IoI and race

was only marginally significant. Additionally, the relationship between 30-year CVD risk and ICE_{tract} was dependent on race in a way that it was harmful to both Blacks and Whites, but more so in Blacks. Finally, a significant interaction between ICE_{school} and race suggests an *increased* CVD risk in Blacks and a *decreased* risk in Whites as school segregation increases.

In this study, the segregation measures of the Index of Concentration of Extremes or ICE (both by tract and school) operated as hypothesized (i.e., more harmful to Blacks), but unlike ICE_{school}, ICE_{tract} was also harmful to Whites. Notably, by the start of the Add Health Survey in the mid-1990s, segregation was well entrenched into American society despite being outlawed for nearly 20-years by the passage of the Fair Housing Act in 1968, the Equal Credit Opportunity Act in 1974, and the Community Reinvestment Act in 1977, which together banned discrimination in the sale and rental of housing, mortgage lending, and redlining in Black neighborhoods.[266] The results using the ICE measures are somewhat consistent with the broader literature on residential segregation and health outcomes in general where a higher level of residential segregation is more strongly associated with adverse birth outcomes,[90,91,580-594] cancer outcomes,[595-597] HIV mortality,[598] both fatal and non-fatal cardiovascular outcomes,[126,507] hypertension,[177] survival,[72] fatal police shootings,[89] and community violence among Blacks compared to Whites.[178,599,600] Additionally, some research suggests that increasing segregation is also harmful to Whites with respect to CVD risk factors and outcomes, which may depend on poverty status.[507,609,610]

4.4.a. Residential Segregation and CVD

Residential segregation, and the neighborhood instability that is born from it, is considered a social determinant of health.[94,485,500] The stress associated with living in segregated neighborhoods is disproportionately experienced by Black Americans due to the historic, and current, oppression through both *de facto* and *de jure* means.[10,11,485,500] Additionally, this stress mechanism likely contributes to earlier onset CVD or the risk factors associated with CVD.[485,500] The presence of earlier onset of CVD is a rationale for utilizing the Add Health study to examine CVD risk as it sampled people while they were in 7th through 12th grade, likely before risk factors or CVD developed, with the exception of smoking. Additionally, the outcomes were measured when the Add Health participants were aged in their late 20s to 30s, when sub-clinical CVD is likely to start developing. This study observed that segregation is harmful, and more harmful to Blacks compared to Whites, which is consistent with the literature.[126]

Housing is a very important resource for general health. Research shows that Americans gain generational wealth through homeownership.[36] Housing also provides stability, safety, and improves the neighborhood environment.[611] These factors in housing are directly associated with residential segregation.[94] Policies affecting access to housing resources for Blacks produce a disparate impact that can influence CVD risk even when education and income levels are comparable to Whites.[487] While our study did not demonstrate an association between the more traditional measures of segregation (i.e., indices of dissimilarity, isolation, and concentration of extremes) and CVD risk, this is not necessarily inconsistent with the literature.[177]

There are a few studies linking segregation and risk factors for CVD. For example, Feldman et. al. reported that ICE for race, or a higher concentration of Whites in a community, was associated with lower odds of hypertension after controlling for race, sex, and traditional risk factors for hypertension.[177] Other studies have demonstrated similar findings that suggest that less segregation produces lower blood pressure especially for Blacks.[612] This finding may be somewhat related to neighborhood disadvantage which has been shown to be a risk factor for CVD.[507,613] Segregation is associated with other CVD risk factors as well,[614] such as cardiometabolic risk,[615] obesity,[616] and behaviors such as inactivity.[617]

A unique observation in this study is the result on school segregation. In this context, Black students are exposed to more Black students while Whites are exposed to more White students (i.e., school segregation). I observed that the association between school segregation and 30-year CVD risk score was dependent on race and more harmful to Blacks than Whites for both the Full and Hard CVD outcomes. This finding is consistent with another study that measured school segregation as percent White students in the school and non-cardiovascular disease outcomes such as mental health, substance abuse, and self-rated health.[576] By adulthood, Black students exposed to a school with a higher proportion of White students had worse outcomes later in life, whereas Whites were healthier with respect to these outcomes. The direction of the association for Blacks and Whites was consistent with the school segregation finding.

4.4.b. Potential Mechanisms

Housing policies are but one example of how structural racism is reinforced and shaped over time. Given that polices can affect one's access to resources that are health promoting and that

policies are written in a way to limit certain populations' access to resources, it is plausible that one mechanism by which structural racism can affect health is through a material pathway, such as education, income, or housing.[6,10] Another plausible mechanism operates through a psychosocial pathway through perceived injustice, social status, and stress.[10] For example, Braveman et. al. reported in a review paper that because of racism that is experienced every day in some fashion, Blacks accumulate the effects of stress and without adequate resources, over time, the effect of accumulated stressors eventually takes its physical toll on a person.[618] The stressor of anticipating racial discrimination (i.e., vigilance) also is associated with higher levels of hypertension.[619]

Other research suggests that there may be stress buffering behaviors that may explain some of the weaker findings in this study. For example, Hsieh et. al. observed that support from friends played a protective role against hypertension later in life (but still in early adulthood).[620] Additionally, some researchers posit that certain personal characteristics can prevent or soften the effect of discrimination despite lower levels of resources.[555]

4.4.c. Limitations and Strengths

The limitations and strengths of this study can be organized into person, place, and time. A potential limitation is that the individual data are from Add Health which is an observational, cohort study, making the results subject to the potential for residual confounding, for example moving during the follow-up period. Additionally, the amount of exposure each individual may experience could be a problematic source of bias. For example, some Add Health respondents may have taken the survey in one Census tract and experienced a different level of structural racism in another Census tract because they may have moved before completing the survey. This source of bias cannot be accounted for through this study because I do not have information in subsequent waves about neighborhood changes.

Another limitation to this study is that there could be some selection bias due to non-participation. This non-participation may be related to the outcome as well as the exposure, where those who are more disadvantaged, due possibly to strained resources from neighborhood disinvestment, may be less likely to participate. To address this potential limitation, Add Health data is adjusted for non-response and post-stratification. Additionally, there were a substantial

number of respondents for which tract level or block group level data could not be linked. These cases were excluded. In the full dataset, there were 9,206 respondents who were Black or White. The final sample size was 6,835 respondents. The difference is made up of those missing tract level information because their addresses could not be geocoded, or missing race at the schools survey, and the remaining is related to missing data on covariates or the outcome. If these missing cases are related to the exposure or outcome this may present a source of bias.[621] I did attempt to mitigate this bias by coding an unknown category for income and education, which allowed us to maintain some of the sample size.

I hypothesized parental income and parental education to be confounding variables as they are thought to be associated with both segregation and CVD. However, they may be mediators as living in more segregated areas could have affected parental education and/or income. I did examine this possibility by running models with and without these variables (data not shown), and there was no change in associations between exposures and outcomes.

There may be issues related to measurement of residential segregation especially for the Indices of Dissimilarity and Isolation. Namely, Add Health is completely de-identified, including geographic indicators. As such, I did not have access to a metropolitan area/city (e.g., Census Place). The definitions of these indices include a larger area, which was Census tract in this study. I summed up data at the Block group level and aggregated up to the Census tract level. Policies are not generally different at the tract level, but they may be differentially experienced at the tract level. Having access to the metropolitan area would be more consistent with the definitions, which may explain why the mean values of these measures were so low in comparison to other studies. For example, this study has a mean Index of Dissimilarity of 14%, while others report this level at closer to 60% in the 1990s.[599,622] While our study reported an Index of Isolation of 9%, the value from other studies are about 26%.[623] The mean values for Indices of Concentration of Extremes at the tract level are consistent with other studies.[177]

Despite these limitations, there are several strengths of note. First, temporality is established in Add Health as residential segregation is measured at Wave I while the outcome is measured at Wave IV. While it is possible that CVD begins in high school ages, this is unlikely, as older adolescents have not accumulated the needed risk factors to develop chronic CVD, so it is

unlikely that respondents at Wave I had CVD at the time of the first interview. A major strength of this study is the use of a nationally representative sample.

Another major strength is the use of several measures of residential and school segregation as a measure of structural racism that captures some of the most longstanding and disadvantaging policy areas in US history. For example, segregation is related to wealth, a downstream effect of discriminatory housing and education policies. Another strength in the use of Add Health is that the outcome is based on objectively and systematically collected data. Finally, the structural racism data source is another strength. The source of data used for the tract-level structural racism measures are from the US Decennial Census, an extremely reputable source. Finally, a novel measure of school segregation was generated. The measure collected examines the level of concentration of privilege of disadvantage in a school using self-reported race in the in-school survey of ~90,000 students.

4.5 Conclusion

I observed that, consistent with other studies, the relationship between segregation and CVD risk is dependent on race and that segregation, as measured by the two ICE measures, is a greater risk factor for 30-year CVD risk for racial and ethnic minorities, who endure disparate impacts of historic policies. Contrary to expectations, the standard segregation measures (e.g., IoD and IoI) showed either no difference by race (IoD) or that segregation was harmful for Whites but not Blacks (IoI). Studies could incorporate other housing related indicators of structural racism such as wealth inequality, home ownership, and education. Another possible research avenue is to examine hyper-segregation (IoD $\geq$ 90%),[460] which may be more damaging to one's health, or potentially comparing people in the highest levels of segregation to the lowest using cut-points,[606] especially if there appears to be a bimodal distribution. Another area is to examine parental socioeconomic indicators in a formal mediation analysis to determine what role, if any, it has on the relationship between segregation and CVD risk. Additionally, measures capturing a wide range of policies could be examined either one by one or in some kind of an index reflecting several racist policies rather than just one. Finally, given Add Health's data structure, multi-level models can be incorporated into an analysis to examine the level of variation in CVD risk between and within schools (or neighborhoods) in this relationship.

This study examines several measures of residential segregation, which capture the evenness (IoD), exposure (IoI), and concentration (ICE) of racial groups. Together these measures assess how many people need to move out of an area to achieve an equitable distribution of residents by race; how likely a Black person is to be exposed to another Black person in that tract; and the extreme racial concentration in a tract or school. Additionally, this study names policies and explains how and why housing policy contributed to residential segregation and examines how old and obsolete policies affect present-day health. This study adds to the literature that examines area-level indicators of structural racism as predictors of individual health outcomes. This study also adds to this literature by incorporating a novel measure of structural racism (segregation in schools) as well as examining the additional aspects of segregation of evenness, exposure, and concentration and the relationship between these indicators and CVD risk later in life. The implications of this study suggest that understanding the role of residential segregation on several risk factors that place racial and ethnic minorities at a disadvantage for the health of cardiovascular system is critical for eliminating cardiovascular disease disparities.

4.6 Tables and Figures

Table 4. 1 Measures of Structural Racism through Residential and School Segregation.

Measure	Formula	Conceptual Definition
Index of Dissimilarity[118,177,178,623]	$D = \left[\frac{1}{2}\sum_{i=1}^{n}\left\|\left(\frac{b_i}{B}\right) - \left(\frac{w_i}{W}\right)\right\|\right]$ i=Block group in a tract; b_i=number of Black residents in the block group; B=number of Black residents in the tract; w_i=number of White residents in the block group; W_i=number of White residents in the tract.	Relative proportion of Blacks who would have to change Census tracts to achieve an even residential distribution in the Tract. Values range from 0-1, interpreted as a percent.
Index of Isolation[118]	$xPx* = \sum_{i=1}^{n}\left[\frac{x_i}{X}\right]\left[\frac{x_i}{t_i}\right]$ i=Block group x_i=number of Blacks in block group i, X=number of Black in the tract, t_i=number of people in block group i.	The probability of a Black person in the area being exposed to another Black person. Values range from 0-1, interpreted as a percent.
Index of Concentration of Extremes[177,178]	$ICE = \frac{(Number\ of\ Whites\ in\ tract) - (Number\ of\ Blacks\ in\ tract)}{Total\ population\ in\ tract\ with\ information}$ The number of White residents is defined as the number of non-Hispanic White residents in the tract. The number of Black residents is defined as the number of non-Hispanic Black in the tract. The total population includes only those where race is available in the tract.	Examining the extreme concentration of racial group in a Census tract. Values range from -1 to +1. More negative values indicate a higher concentration of those who are Blacks in the tract.
Index of Concentration of Extremes (novel)	$ICE = \frac{(Number\ of\ Whites\ in\ school) - (Number\ of\ Blacks\ in\ school)}{Total\ population\ in\ school\ with\ information}$ The number of White students in the schools is defined as the number of non-Hispanic White students. The number of Black students in the school is defined as the number of non-Hispanic Black students in the school. The total population includes all students in the school with where race is available.	Examining the extreme concentration of a racial group in schools. Values range from -1 to +1. More negative values indicate a higher concentration of those who are Black in the school.

Table 4. 2 Descriptive Statistics Overall and by Race, Add Health Wave I (n=6,835).

	Overall N=6,835			Non-Hispanic Blacks N=1,990			Non-Hispanic Whites N=4,845			*P-value*[a]
Measure	**Mean**	**SE**	**%**	**Mean**	**SE**	**%**	**Mean**	**SE**	**%**	
Race										
non-Hispanic Black	---	---	18.1	---	---	---	---	---	---	---
non-Hispanic White	---	---	81.2	---	---	---	---	---	---	---
Age at Wave IV (yrs)	28.1	0.131	---	28.4	0.215	---	28.1	0.144	---	0.2056
Sex										
Female	---	---	52.3	---	---	56.7	---	---	51.2	0.0099
Male	---	---	47.7	---	---	43.3	---	---	48.7	Ref
Parental Education										
<HS	---	---	8.1	---	---	11.7	---	---	7.3	0.1639
HS graduate	---	---	52.4	---	---	53.3	---	---	52.2	0.0057
College grad	---	---	35.1	---	---	27.0	---	---	36.9	Ref
Unknown	---	---	4.4	---	---	8.0	---	---	3.5	0.1943
Parental Income										
<$20,000	---	---	14.7	---	---	27.7	---	---	11.7	<0.0001
$20,000-$39,999	---	---	23.3	---	---	24.6	---	---	23	0.2032
$40,000-$74,999	---	---	31.4	---	---	16.7	---	---	34.8	<0.0001
$75,000 or more	---	---	11.4	---	---	5.4	---	---	12.8	Ref
Unknown	---	---	19.2			25.6			17.8	0.0013
Outcomes										
30-Year CVD Risk (Full)[b]	0.123	0.002	---	0.135	0.004	---	0.120	0.002	---	0.0026
30-Year CVD Risk (Hard)[b]	0.068	0.002	---	0.077	0.003	---	0.065	0.002	---	0.0018
Exposures										
Index of Dissimilarity	0.397	0.015	---	0.296	0.016	---	0.421	0.016	---	<0.0001
Index of Isolation	0.146	0.019	---	0.479	0.028	---	0.068	0.009	---	<0.0001
ICE (race in tract)	0.673	0.044	---	-0.133	0.069	---	0.859	0.017	---	<0.0001
ICE (race in school)	0.346	0.033	---	-0.076	0.046	---	0.444	0.024	---	<0.0001
Covariates										
Income inequality (ICE) - tract	-0.064	0.019	---	-0.240	0.029	---	-0.023	0.019	---	<0.0001
% in Tract with <HS	0.268	0.010	---	0.351	0.013	---	0.249	0.01	---	<0.0001
Urban Tract, % Yes	---	---	40.0	---	---	49.6	---	---	37.8	0.1477
% Latinx/Hispanic in tract	0.031	0.003	---	0.032	0.006	---	0.030	0.004	---	0.7144
% Black in Tract	0.147	0.021	---	0.549	0.036	---	0.054	0.008	---	<0.0001
% below Federal Poverty Level in tract	0.142	0.009	---	0.254	0.016	---	0.116	0.008	---	<0.0001

Ref = reference value; HS=High School; ICE=Index of Concentration of Extremes

[a] *P-values* are generated though t-test for continuous variables and the chi-square test for categorical/binary variables.

[b] 30-Year cardiovascular disease risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke (hard) + coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure (full). The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

Table 4. 3 Distribution of Area Level Variables at the Tract Level.

Measure	Level	N	Mean	SE	Range	%	Q1	Median	Q3	IQR
Exposures										
Index of Dissimilarity	Census tract	675	0.141	0.009	0.000-0.894	---	0.048	0.109	0.193	0.145
Index of Isolation	Census tract	675	0.093	0.018	0.000-1.000	---	0.000	0.014	0.119	0.119
ICE (race in tract)[a]	Census tract	675	0.525	0.084	-1.000-1.000	---	0.348	0.841	0.950	0.602
ICE (race in school) [a]	School	67	0.307	0.047	-0.436-0.870	---	0.065	0.346	0.537	0.472
Covariates										
Income inequality (ICE) [a]	Census tract	675	-0.026	0.031	-0.807-0.730	---	-0.249	-0.040	0.192	0.441
% in tract with <HS	Census tract	675	0.248	0.014	0.013-0.726	---	0.123	0.229	0.352	0.229
Urban, %	Census tract	675	0.750	0.011	0.000-1.000	75.0	0.000	0.333	0.667	0.667
% Latino in tract	Census tract	675	0.053	0.008	0.000-0.924	---	0.004	0.013	0.066	0.062
% Black in tract	Census tract	675	0.211	0.041	0.000-1.000	---	0.008	0.044	0.272	0.264
% below FPL in tract	Census tract	675	0.045	0.013	0.008-0.765	---	0.048	0.104	0.194	0.146

ICE=Index of Concentration of Extremes; HS=High School; FPL=Federal Poverty Level; Q refers to quartile; IQR=inter-quartile range.

a. Interpreting ICE: Negative values indicate that there is less privilege in tract, whereas positive values indicate more privilege in the tract

Table 4. 4 Regression Coefficients[a] for 30-Year Full Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on the Index of Dissimilarity (Residential Segregation) at Wave I (n=6,835).

	Log-Transformed Full 30-year CVD Risk Score[b]								
	Model 1[c]			Model 2[c]			Model 3[c]		
Measure	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-2.2752	0.0311	<0.0001	-5.4709	0.1265	<0.0001	-5.4699	0.1254	<0.0001
Index of Dissimilarity	-0.0944	0.0676	0.1626	-0.0448	0.0442	0.3107	-0.0466	0.0474	0.3256
Race									
Black	0.0814	0.0277	0.0033	0.0339	0.0263	0.1972	0.0300	0.0401	0.4547
White (ref)	---			---			---		
Age at Wave 4 (yrs)	---			0.0934	0.0042	<0.0001	0.0934	0.0042	<0.0001
Sex									
Male	---			0.6716	0.0171	<0.0001	0.6716	0.0171	<0.0001
Female (ref)	---			---			---		
Parental Education									
<HS	---			0.1991	0.0395	<0.0001	0.1992	0.0394	<0.0001
HS graduate	---			0.0965	0.0209	<0.0001	0.0965	0.0208	<0.0001
College grad (ref)	---			---			---		
Unknown	---			0.1143	0.0437	0.0088	0.1141	0.0437	0.0090
Parental Income									
<$20,000	---			0.1068	0.0343	0.0019	0.1069	0.0343	0.0019
$20,000-$39,999	---			0.1180	0.0296	<0.0001	0.1181	0.0296	<0.0001
$40,000-$74,999	---			0.0422	0.0253	0.0960	0.0422	0.0254	0.0959
$75,000 or more (ref)	---			---			---		
Unknown	---			0.0373	0.0267	0.1615	0.0373	0.0267	0.1615
Income inequality (ICE) in tract	---			-0.0352	0.0592	0.5516	-0.0356	0.0594	0.5496
Urban tract (Yes)	---			0.0446	0.0201	0.0262	0.0447	0.0200	0.0257
% Black in tract	---			-0.0907	0.0512	0.0761	-0.0915	0.0510	0.0732
% Latinx/Hispanic in tract	---			-0.2633	0.1065	0.0134	-0.2631	0.1065	0.0135
% <HS in tract	---			0.2351	0.0937	0.0121	0.2353	0.0937	0.0121
% below FPL in tract	---			0.2938	0.1573	0.0618	0.2927	0.1585	0.0648
Index of Dissimilarity*Race	---			---			0.0137	0.1000	0.8913
Exchangeable Correlation	0.0144			0.0016			0.0016		

[a] General Estimating Equations (GEE) with the identity link

[b] 30-Year full cardiovascular disease risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke, coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[c] Model 1: Includes race and exposure; Model 2: include Models 1 + age, sex, parental education, parental income, income inequality, urbanicity, % Black, % Latinx/Hispanic, % with <HS, % below Federal Poverty Level (FPL); Model 3: includes Models 1-2 & a race*exposure interaction

SE=standard error

Table 4. 5 Regression[a] Coefficients for 30-Year Full Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on the Index of Isolation (Residential Segregation) at Wave I (n=6,835).

	Log-Transformed Full 30-year CVD Risk Score[b]								
	Model 1[c]			Model 2[c]			Model 3[c]		
Measure	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-2.3226	0.0202	<0.0001	-5.4974	0.1258	<0.0001	-5.5071	0.1239	<0.0001
Index of Isolation	0.1100	0.0668	0.0999	0.0427	0.0856	0.6215	0.1190	0.0901	0.1864
Race									
Black	0.0526	0.0361	0.1454	0.0338	0.0271	0.2114	0.0749	0.0362	0.0388
White (ref)	---			---			---		
Age at Wave 4 (yrs)	---			0.0936	0.0042	<0.0001	0.0936	0.0042	<0.0001
Sex									
Male	---			0.6723	0.0171	<0.0001	0.6723	0.0170	<0.0001
Female (ref)	---			---			---		
Parental Education									
<HS	---			0.1985	0.0396	<0.0001	0.1988	0.0399	<0.0001
HS graduate	---			0.0957	0.0210	<0.0001	0.0958	0.0209	<0.0001
College grad (ref)	---			---			---		
Unknown	---			0.1131	0.0435	0.0094	0.1149	0.0436	0.0084
Parental Income									
<$20,000	---			0.1053	0.0345	0.0023	0.1055	0.0344	0.0022
$20,000-$39,999	---			0.1175	0.0297	<0.0001	0.1171	0.0296	<0.0001
$40,000-$74,999	---			0.0413	0.0256	0.1060	0.0414	0.0255	0.1053
$75,000 or more (ref)	---			---			---		
Unknown	---			0.0360	0.0267	0.1781	0.0362	0.0267	0.1758
Income inequality (ICE) in tract	---			-0.0320	0.0596	0.5917	-0.0179	0.0596	0.7647
Urban tract (Yes)	---			0.0473	0.0199	0.0174	0.0459	0.0197	0.0200
% Black in tract	---			-0.1128	0.0824	0.1713	-0.1032	0.0841	0.2200
% Latinx/Hispanic in tract	---			-0.2370	0.1042	0.0229	-0.2568	0.1037	0.0133
% <HS in tract	---			0.2243	0.0941	0.0171	0.2298	0.0930	0.0134
% below FPL in tract	---			0.2993	0.1579	0.0581	0.3259	0.1560	0.0368
Index of Isolation*Race	---			---			-0.1632	0.0911	0.0732
Exchangeable Correlation	0.0147			0.0017			0.0016		

[a] General Estimating Equations (GEE) with the identity link

[b] 30-Year full cardiovascular disease risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke, coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[c] Model 1: Includes race and exposure; Model 2: include Models 1 + age, sex, parental education, parental income, income inequality, urbanicity, % Black, % Latinx/Hispanic, % with <HS, % below Federal Poverty Level (FPL); Model 3: includes Models 1-2 & a race*exposure interaction

SE=standard error

Table 4. 6 Regression[a] Coefficients for 30-Year Full Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on the Index of Concentration of Extremes at the Tract (Residential Segregation) at Wave I (n=6,835).

	Log-Transformed Full 30-year CVD Risk Score[b]								
	Model 1[c]			Model 2[c]			Model 3[c]		
Measure	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-2.2841	0.0326	<0.0001	-5.5304	0.2174	<0.0001	-5.5500	0.2092	<0.0001
Index of Concentration of Extremes (tract)	-0.0320	0.0334	0.2811	0.0352	0.1705	0.8364	0.0418	0.1653	0.8002
Race									
Black	0.0602	0.0370	0.1042	0.0359	0.0265	0.1759	-0.0301	0.0400	0.4515
White (ref)	---			---			---		
Age at Wave 4 (yrs)	---			0.0936	0.0042	<0.0001	0.0936	0.0042	<0.0001
Sex									
Male	---			0.6724	0.0171	<0.0001	0.6719	0.0171	<0.0001
Female (ref)	---			---			---		
Parental Education									
<HS	---			0.1984	0.0394	<0.0001	0.1958	0.0394	<0.0001
HS graduate	---			0.0957	0.0210	<0.0001	0.0956	0.0209	<0.0001
College grad (ref)	---			---			---		
Unknown	---			0.1136	0.0436	0.0092	0.1133	0.0437	0.0096
Parental Income									
<$20,000	---			0.1056	0.0346	0.0023	0.1067	0.0344	0.0019
$20,000-$39,999	---			0.1173	0.0296	<0.0001	0.1168	0.0294	<0.0001
$40,000-$74,999	---			0.0411	0.0255	0.1065	0.0413	0.0254	0.1038
$75,000 or more (ref)	---			---			---		
Unknown	---			0.0364	0.0268	0.1755	0.0368	0.0268	0.1707
Income inequality (ICE) in Tract	---			-0.0319	0.0605	0.5978	-0.0154	0.0596	0.7956
Urban Tract (Yes)	---			0.0462	0.0199	0.0202	0.0471	0.0200	0.0187
% Black in tract	---			-0.0119	0.3406	0.9721	0.1713	0.3280	0.6014
% Latinx/Hispanic in tract	---			-0.2063	0.1718	0.2299	-0.2079	0.1677	0.2150
% <HS in tract	---			0.2273	0.0948	0.0165	0.2342	0.0916	0.0106
% below FPL in tract	---			0.2999	0.1578	0.0573	0.3153	0.1549	0.0418
ICE (tract)*Race	---			---			0.1289	0.0545	0.0181
Exchangeable Correlation		0.0148			0.0018			0.0016	

[a] General Estimating Equations (GEE) with the identity link

[b] 30-Year full cardiovascular disease risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke, coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[c] Model 1: Includes race and exposure; Model 2: include Models 1 + age, sex, parental education, parental income, income inequality, urbanicity, % Black, % Latinx/Hispanic, % with <HS, % below Federal Poverty Level (FPL); Model 3: includes Models 1-2 & a race*exposure interaction
SE=standard error

Table 4. 7 Regression[a] for 30-Year Full Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on the Index of Concentration of Extremes at the School (School Based Segregation) at Wave I (n=6,835).

	Log-Transformed Full 30-year CVD Risk Score[b]								
	Model 1[c]			Model 2[c]			Model 3[c]		
Measure	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-2.3256	0.0316	<0.0001	-5.4901	0.1251	<0.0001	-5.4780	0.1250	<0.0001
Index of Concentration of Extremes (School)	0.0279	0.0669	0.6764	-0.0172	0.0397	0.6645	-0.0559	0.0459	0.2229
Race									
Black	0.1008	0.0335	0.0026	0.0330	0.0278	0.2357	0.0129	0.0296	0.6630
White (ref)	---			---			---		
Age at Wave 4 (yrs)	---			0.0937	0.0042	<0.0001	0.0939	0.0042	<0.0001
Sex									
Male	---			0.6723	0.0171	<0.0001	0.6723	0.0172	<0.0001
Female (ref)	---			---			---		
Parental Education									
<HS	---			0.1974	0.0394	<0.0001	0.1966	0.0395	<0.0001
HS graduate	---			0.0956	0.0209	<0.0001	0.0945	0.0211	<0.0001
College grad (ref)	---			---			---		
Unknown	---			0.1127	0.0435	0.0096	0.1104	0.0436	0.0114
Parental Income									
<$20,000	---			0.1057	0.0346	0.0023	0.1080	0.0344	0.0017
$20,000-$39,999	---			0.1172	0.0296	<0.0001	0.1186	0.0294	<0.0001
$40,000-$74,999	---			0.0414	0.0256	0.1053	0.0430	0.0253	0.0894
$75,000 or more (ref)	---			---			---		
Unknown	---			0.0363	0.0268	0.1767	0.0374	0.0267	0.1607
Income inequality (ICE) in tract	---			-0.0344	0.0590	0.5593	-0.0292	0.0587	0.6192
Urban tract (Yes)	---			0.0445	0.0200	0.0259	0.0388	0.0202	0.0555
% Black in Tract	---			-0.0933	0.0555	0.0927	-0.0679	0.0581	0.2427
% Latinx/Hispanic in tract	---			-0.2538	0.1072	0.0179	-0.2960	0.1119	0.0081
% <HS in tract	---			0.2316	0.0979	0.0180	0.2391	0.0951	0.0120
% below FPL in tract	---			0.2944	0.1581	0.0625	0.2937	0.1569	0.0613
ICE (School)*Race	---			---			0.1575	0.0757	0.0375
Exchangeable Correlation	0.0180			0.0023			0.0022		

[a] General Estimating Equations (GEE) with the identity link

[b] 30-Year full cardiovascular disease risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke, coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[c] Model 1: Includes race and exposure; Model 2: include Models 1 + age, sex, parental education, parental income, income inequality, urbanicity, % Black, % Latinx/Hispanic, % with <HS, % below Federal Poverty Level (FPL); Model 3: includes Models 1-2 & a race*exposure interaction

SE=standard error

Table 4. 8 Regression[a] Coefficients for 30-Year Hard Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on the Index of Dissimilarity (Residential Segregation) at Wave I (n=6,835).

	Log-Transformed Hard 30-year CVD Risk Score[b]								
	Model 1[c]			Model 2[c]			Model 3[c]		
Measure	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-2.9643	0.0381	<0.0001	-6.6623	0.1480	<0.0001	-6.6620	0.1466	<0.0001
Index of Dissimilarity	-0.1228	0.0817	0.1329	-0.0561	0.0508	0.2698	-0.0565	0.0547	0.3012
Race									
Black	0.0963	0.0346	0.0054	0.0492	0.0318	0.1219	0.0482	0.0475	0.3108
White (ref)	---			---			---		
Age at Wave 4 (yrs)	---			0.1060	0.0049	<0.0001	0.1060	0.0049	<0.0001
Sex									
Male	---			0.8953	0.0201	<0.0001	0.8953	0.0201	<0.0001
Female (ref)	---			---			---		
Parental Education									
<HS	---			0.2363	0.0483	<0.0001	0.2363	0.0482	<0.0001
HS graduate	---			0.1130	0.0241	<0.0001	0.1131	0.0241	<0.0001
College grad (ref)	---			---			---		
Unknown	---			0.1365	0.0517	0.0082	0.1365	0.0517	0.0083
Parental Income									
<$20,000	---			0.1217	0.0401	0.0024	0.1217	0.0401	0.0024
$20,000-$39,999	---			0.1326	0.0347	0.0001	0.1326	0.0347	0.0001
$40,000-$74,999	---			0.0445	0.0297	0.1339	0.0445	0.0297	0.1339
$75,000 or more (ref)	---			---			---		
Unknown	---			0.0424	0.0311	0.1718	0.0424	0.0311	0.1718
Income inequality (ICE) in tract	---			-0.0418	0.0692	0.5460	-0.0419	0.0696	0.5471
Urban tract (Yes)	---			0.0501	0.0234	0.0322	0.0501	0.0234	0.0319
% Black in tract	---			-0.1055	0.0616	0.0869	-0.1057	0.0617	0.0866
% Latinx/Hispanic in tract	---			-0.3015	0.1290	0.0194	-0.3014	0.1289	0.0193
% <HS in tract	---			0.2564	0.1098	0.0195	0.2564	0.1098	0.0195
% below FPL in tract	---			0.3386	0.1821	0.0629	0.3383	0.1833	0.0650
Index of Dissimilarity*Race	---			---			0.0036	0.1199	0.9760
Exchangeable Correlation	0.0130			0.0016			0.0016		

[a] General Estimating Equations (GEE) with the identity link

[b] 30-Year hard cardiovascular disease risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[c] Model 1: Includes race and exposure; Model 2: include Models 1 + age, sex, parental education, parental income, income inequality, urbanicity, % Black, % Latinx/Hispanic, % with <HS, % below Federal Poverty Level (FPL); Model 3: includes Models 1-2 & a race*exposure interaction

SE=standard error

Table 4. 9 Regression[a] Coefficients for 30-Year Hard Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on the Index of Isolation (Residential Segregation) at Wave I (n=6,835).

	Log-Transformed Hard 30-year CVD Risk Score[b]								
	Model 1[c]			Model 2[c]			Model 3[c]		
Measure	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-3.0240	0.0238	<0.0001	-6.6945	0.1474	<0.0001	-6.7060	0.1450	<0.0001
Index of Isolation	0.1176	0.0795	0.1392	0.0321	0.1018	0.7526	0.1227	0.1057	0.2458
Race									
Black	0.0676	0.0443	0.1268	0.0501	0.0326	0.1243	0.0988	0.0436	0.0234
White (ref)	---			---			---		
Age at Wave 4 (yrs)	---			0.1063	0.0050	<0.0001	0.1064	0.0049	<0.0001
Sex									
Male	---			0.8962	0.0201	<0.0001	0.8962	0.0201	<0.0001
Female (ref)	---			---			---		
Parental Education									
<HS	---			0.2354	0.0484	<0.0001	0.2358	0.0487	<0.0001
HS graduate	---			0.1121	0.0242	<0.0001	0.1123	0.0242	<0.0001
College grad (ref)	---			---			---		
Unknown	---			0.1353	0.0516	0.0087	0.1374	0.0516	0.0077
Parental Income									
<$20,000	---			0.1200	0.0403	0.0029	0.1202	0.0401	0.0027
$20,000-$39,999	---			0.1319	0.0348	0.0002	0.1313	0.0347	0.0002
$40,000-$74,999	---			0.0433	0.0299	0.1480	0.0434	0.0299	0.1470
$75,000 or more (ref)	---			---			---		
Unknown	---			0.0410	0.0311	0.1882	0.0412	0.0311	0.1856
Income inequality (ICE) in tract	---			-0.0386	0.0699	0.5803	-0.0219	0.0698	0.7540
Urban tract (Yes)	---			0.0528	0.0232	0.0228	0.0512	0.0230	0.0264
% Black in tract	---			-0.1176	0.0994	0.2372	-0.1062	0.1014	0.2949
% Latinx/Hispanic in tract	---			-0.2687	0.1266	0.0337	-0.2921	0.1259	0.0204
% <HS in tract	---			0.2438	0.1106	0.0275	0.2502	0.1093	0.0221
% below FPL in tract	---			0.3444	0.1828	0.0596	0.3759	0.1806	0.0373
Index of Isolation*Race	---			---			-0.1937	0.1062	0.0682
Exchangeable Correlation	0.0132			0.0018			0.0016		

[a] General Estimating Equations (GEE) with the identity link

[b] 30-Year hard cardiovascular disease risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[c] Model 1: Includes race and exposure; Model 2: include Models 1 + age, sex, parental education, parental income, income inequality, urbanicity, % Black, % Latinx/Hispanic, % with <HS, % below Federal Poverty Level (FPL); Model 3: includes Models 1-2 & a race*exposure interaction

SE=standard error

Table 4. 10 Regression[a] Coefficients for 30-Year Hard Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on the Index of Concentration of Extremes at the Tract (Residential Segregation) at Wave I (n=6,835).

	Log-Transformed Hard 30-year CVD Risk Score[b]								
	Model 1[c]			Model 2[c]			Model 3[c]		
Measure	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-2.9820	0.0398	<0.0001	-6.7015	0.2515	<0.0001	-6.7248	0.2418	<0.0001
Index of Concentration of Extremes (ICE, race at tract)	-0.0395	0.0413	0.3395	0.0083	0.1946	0.9659	0.0163	0.1888	0.9313
Race									
Black	0.0749	0.0467	0.1089	0.0515	0.0321	0.1081	-0.0273	0.0480	0.5690
White (ref)	---			---			---		
Age at Wave 4 (yrs)	---			0.1063	0.0050	<0.0001	0.1064	0.0049	<0.0001
Sex									
Male	---			0.8963	0.0202	<0.0001	0.8956	0.0201	<0.0001
Female (ref)	---			---			---		
Parental Education									
<HS	---			0.2354	0.0482	<0.0001	0.2322	0.0482	<0.0001
HS graduate	---			0.1121	0.0242	<0.0001	0.1120	0.0242	<0.0001
College grad (ref)	---			---			---		
Unknown	---			0.1357	0.0517	0.0087	0.1352	0.0518	0.0090
Parental Income									
<$20,000	---			0.1202	0.0404	0.0029	0.1215	0.0401	0.0024
$20,000-$39,999	---			0.1317	0.0347	0.0001	0.1311	0.0344	0.0001
$40,000-$74,999	---			0.0432	0.0299	0.1480	0.0434	0.0297	0.1443
$75,000 or more (ref)	---			---			---		
Unknown	---			0.0412	0.0313	0.1874	0.0417	0.0312	0.1819
Income inequality (ICE) in tract	---			-0.0395	0.0708	0.5764	-0.0199	0.0696	0.7750
Urban tract (Yes)	---			0.0518	0.0232	0.0255	0.0530	0.0234	0.0239
% Black in tract	---			-0.0778	0.3890	0.8415	0.1412	0.3740	0.7058
% Latinx/Hispanic in tract	---			-0.2617	0.2018	0.1947	-0.2634	0.1974	0.1820
% <HS in tract	---			0.2455	0.1110	0.0269	0.2537	0.1072	0.0180
% below FPL in tract	---			0.3435	0.1824	0.0597	0.3618	0.1791	0.0434
ICE(Tract)*Race	---			---			0.1540	0.0652	0.0181
Exchangeable Correlation	0.0133			0.0018			0.0016		

[a] General Estimating Equations (GEE) with the identity link

[b] 30-Year hard cardiovascular disease risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[c] Model 1: Includes race and exposure; Model 2: include Models 1 + age, sex, parental education, parental income, income inequality, urbanicity, % Black, % Latinx/Hispanic, % with <HS, % below Federal Poverty Level (FPL); Model 3: includes Models 1-2 & a race*exposure interaction

SE=standard error

Table 4. 11 Regression[a] Coefficients for 30-Year Hard Cardiovascular Risk Score (Log Transformed) at Add Health Wave IV on the Index of Concentration of Extremes at the School (School Based Segregation) at Wave I (n=6,835).

	Log-Transformed Hard 30-year CVD Risk Score[b]								
	Model 1[c]			Model 2[c]			Model 3[c]		
Measure	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***	**Beta**	**SE**	***P-value***
Intercept	-3.0296	0.0376	<0.0001	-6.6873	0.1466	<0.0001	-6.6730	0.1468	<0.0001
Index of Concentration of Extremes (ICE, Race at School)	0.0361	0.0801	0.6518	-0.0180	0.0470	0.7018	-0.0631	0.0539	0.2418
Race									
Black	0.1224	0.0415	0.0032	0.0486	0.0335	0.1470	0.0252	0.0350	0.4707
White (ref)	---			---			---		
Age at Wave 4 (yrs)	---			0.1064	0.0050	<0.0001	0.1066	0.0049	<0.0001
Sex									
Male	---			0.8962	0.0201	<0.0001	0.8962	0.0202	<0.0001
Female (ref)	---			---			---		
Parental Education									
<HS	---			0.2342	0.0481	<0.0001	0.2333	0.0483	<0.0001
HS graduate	---			0.1119	0.0241	<0.0001	0.1106	0.0243	<0.0001
College grad (ref)	---			---			---		
Unknown	---			0.1347	0.0515	0.0090	0.1319	0.0516	0.0106
Parental Income									
<$20,000	---			0.1203	0.0404	0.0029	0.1230	0.0402	0.0022
$20,000-$39,999	---			0.1316	0.0347	0.0002	0.1333	0.0345	0.0001
$40,000-$74,999	---			0.0435	0.0300	0.1494	0.0454	0.0297	0.1261
$75,000 or more (ref)	---			---			---		
Unknown	---			0.0411	0.0313	0.1882	0.0425	0.0311	0.1712
Income inequality (ICE) in tract	---			-0.0407	0.0690	0.5548	-0.0347	0.0686	0.6132
Urban tract (Yes)	---			0.0502	0.0233	0.0309	0.0436	0.0235	0.0642
% Black in tract	---			-0.1062	0.0672	0.1143	-0.0765	0.0700	0.2748
% Latinx/Hispanic in tract	---			-0.2859	0.1303	0.0282	-0.3350	0.1359	0.0137
% <HS in tract	---			0.2509	0.1146	0.0286	0.2595	0.1113	0.0198
% below FPL in tract	---			0.3397	0.1827	0.0630	0.3387	0.1817	0.0623
ICE (school)*Race	---			---			0.1834	0.0932	0.0491
Exchangeable Correlation	0.0161			0.0023			0.0023		

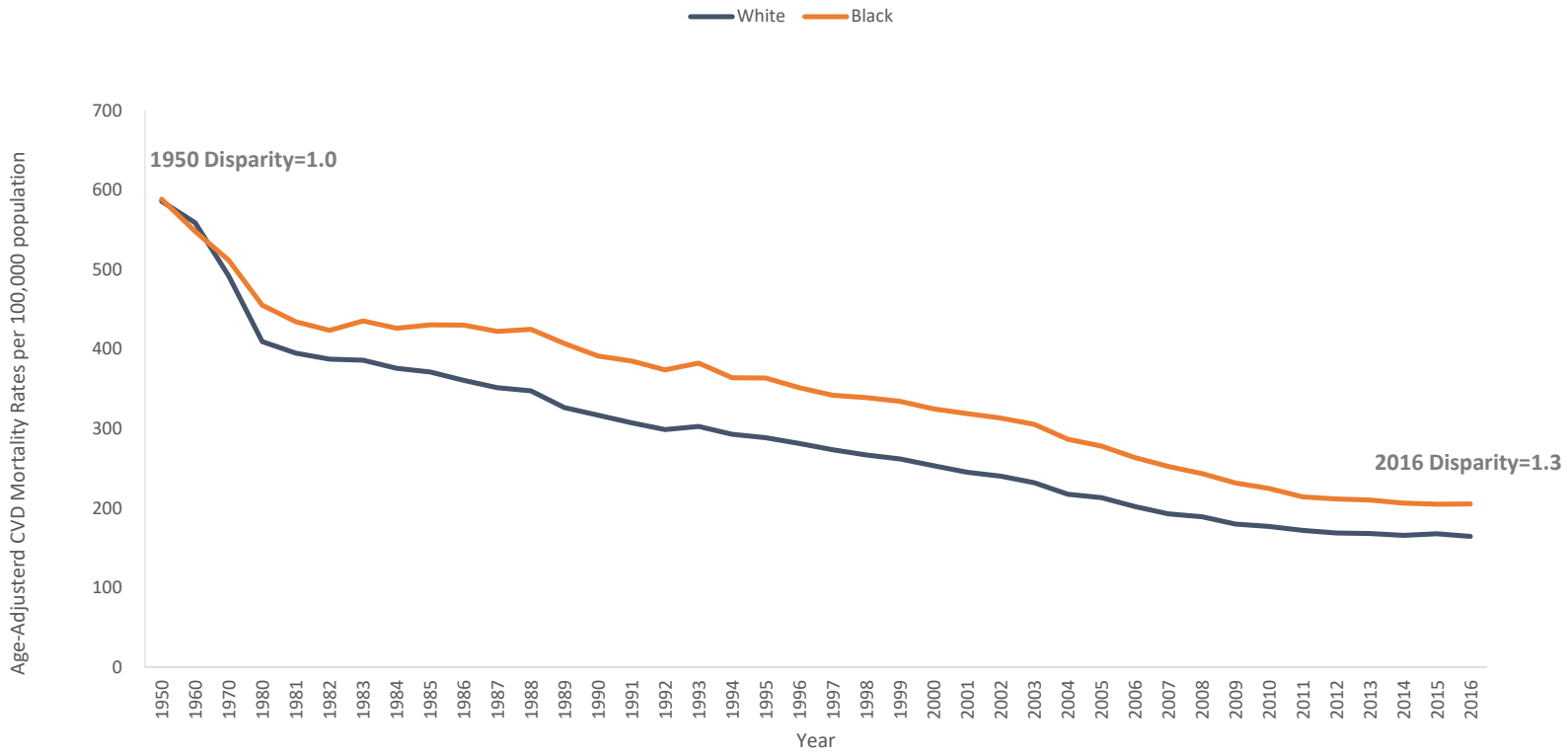
[a] General Estimating Equations (GEE) with the identity link

[b] 30-Year hard cardiovascular disease risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[c] Model 1: Includes race and exposure; Model 2: include Models 1 + age, sex, parental education, parental income, income inequality, urbanicity, % Black, % Latinx/Hispanic, % with <HS, % below Federal Poverty Level (FPL); Model 3: includes Models 1-2 & a race*exposure interaction

SE=standard error

Figure 4. 1 Age Adjusted Cardiovascular Mortality Rates by Race, United States, 1950 – 2016.



Source: National Center for Health Statistics, 2017

Figure 4. 2 Directed Acyclical Graph of the Relationship between Structural Racism and 30-year Cardiovascular Risk Score.

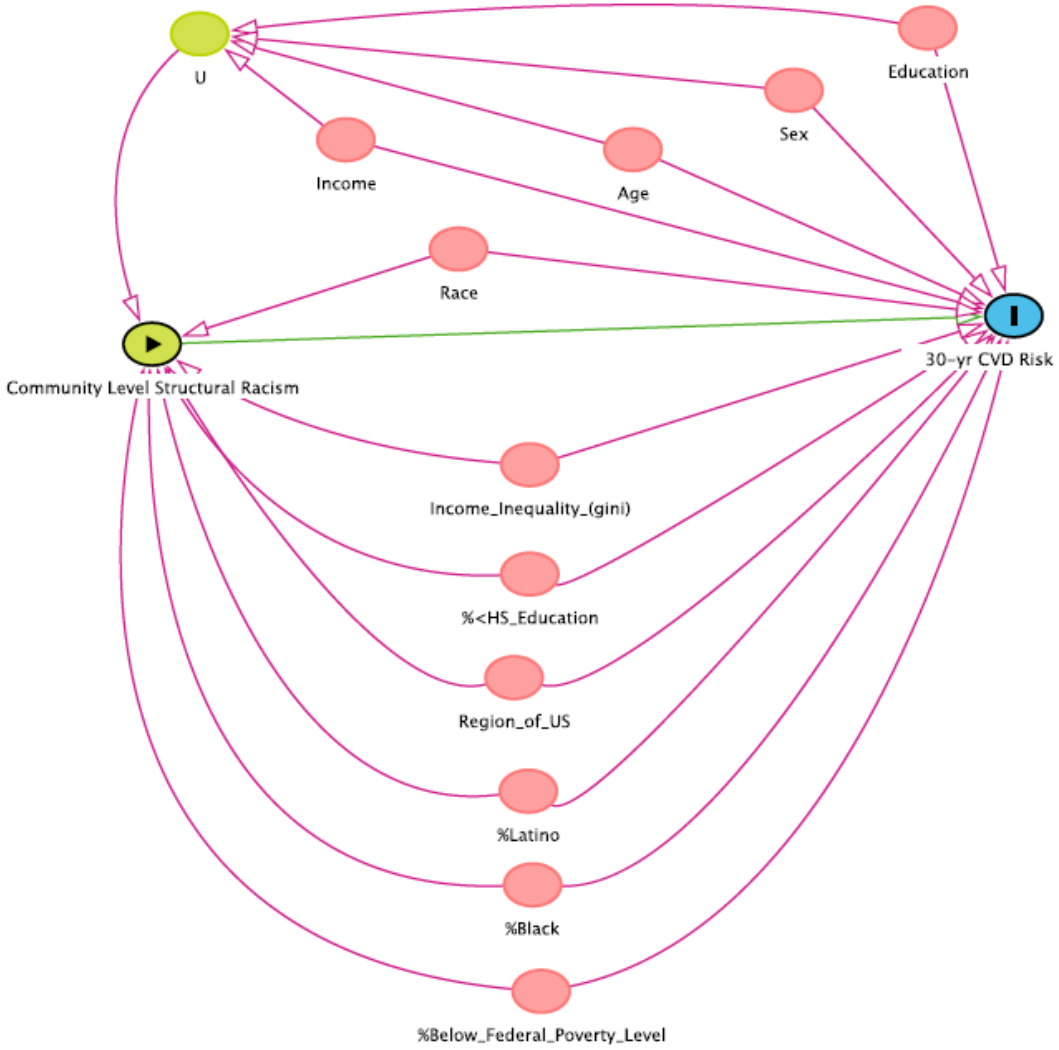
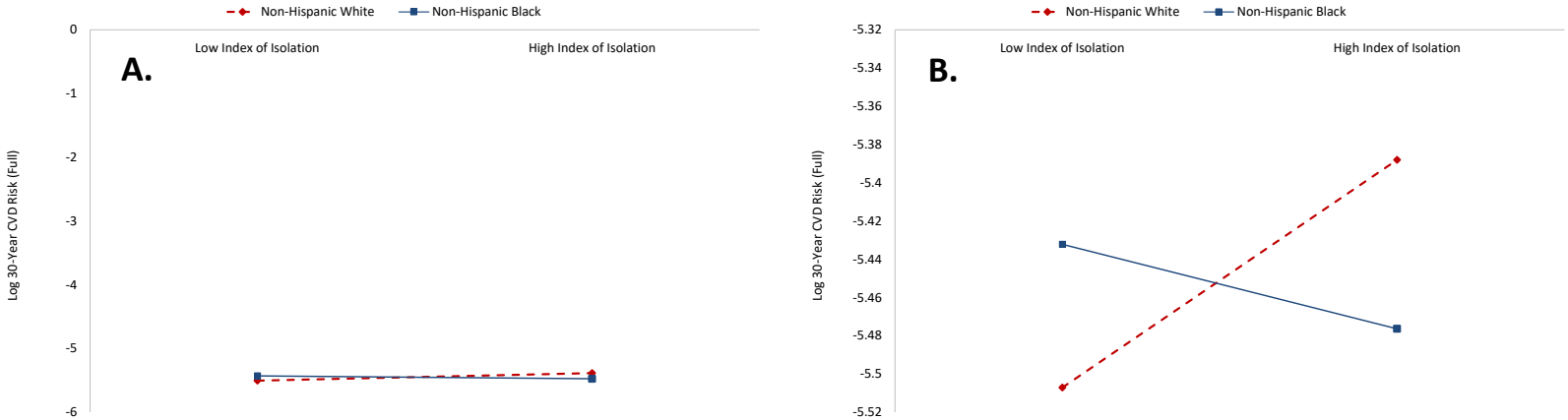


Figure 4. 3 Estimated Wave IV Log – 30-Year Full Cardiovascular Disease (CVD) for Low and High Residential Segregation (Index of Isolation) at Wave I by Race, Add Health.



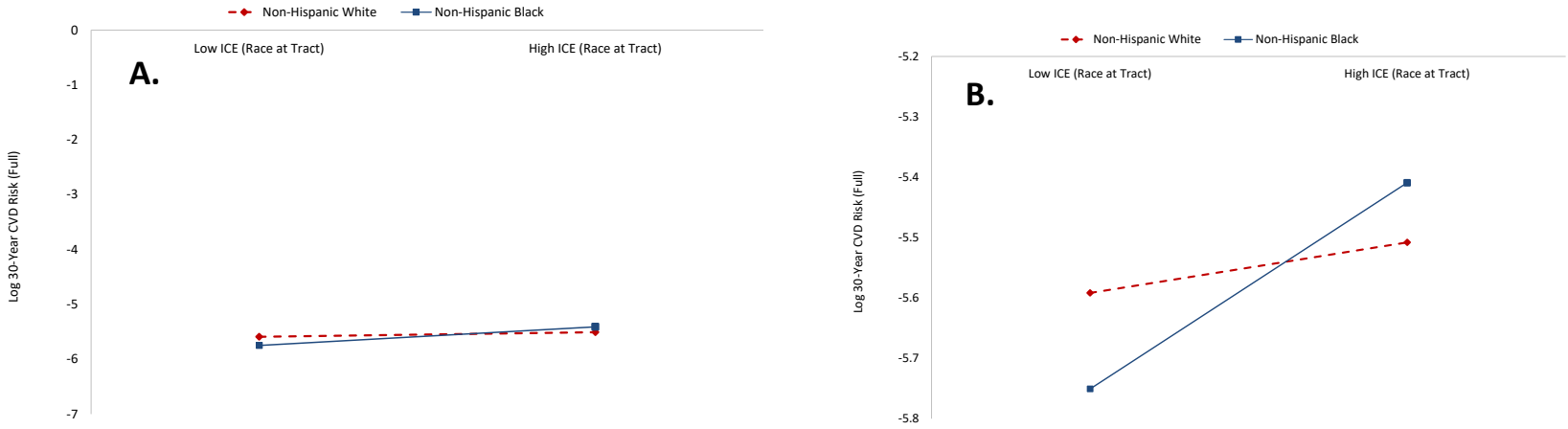
A. Original graphic with axis set with 0 as the high value. B. Zoomed in to see the relationship between the slopes

[a] General Estimating Equations (GEE) with the identity link, p-value for interaction=0.0732 (see Table 4.5). Interpretation: for each unit increase in Index of Isolation, there is a decrease in CVD risk of 4.3% among Blacks and an increase of 13% for Whites.

[b] 30-Year full cardiovascular disease risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke, coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[c] Model includes exposure, race, age, sex, parental education, parental income, income inequality, urbanicity, % Black, % Latinx/Hispanic, % with <HS, % below Federal Poverty Level (FPL)

Figure 4. 4 Estimated Wave IV Log – 30-Year Full Cardiovascular Disease (CVD) for Low and High Residential Segregation (Index of Concentration of Extremes at Tract) at Wave I by Race, Add Health.



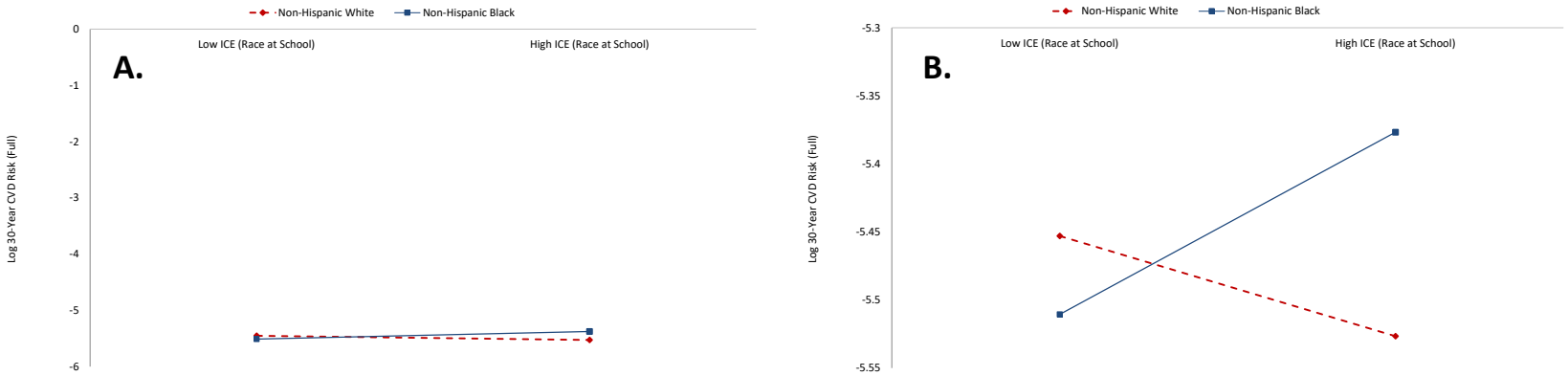
A. Original graphic with axis set with 0 as the high value. B. Zoomed in to see the relationship between the slopes

[a] General Estimating Equations (GEE) with the identity link, p-value for interaction=0.0181 (see Table 4.6). Interpretation: for each unit increase in Index of Concentration of Extremes (tract) there is an increase in CVD risk of 19% among Blacks and an increase of 4% for Whites.

[b] 30-Year full cardiovascular disease risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke, coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[c] Model includes exposure, race, age, sex, parental education, parental income, income inequality, urbanicity, % Black, % Latinx/Hispanic, % with <HS, % below Federal Poverty Level (FPL)

Figure 4. 5 Estimated Wave IV Log – 30-Year Full Cardiovascular Disease (CVD) for Low and High Residential Segregation (Index of Concentration of Extremes at School) at Wave I by Race, Add Health.



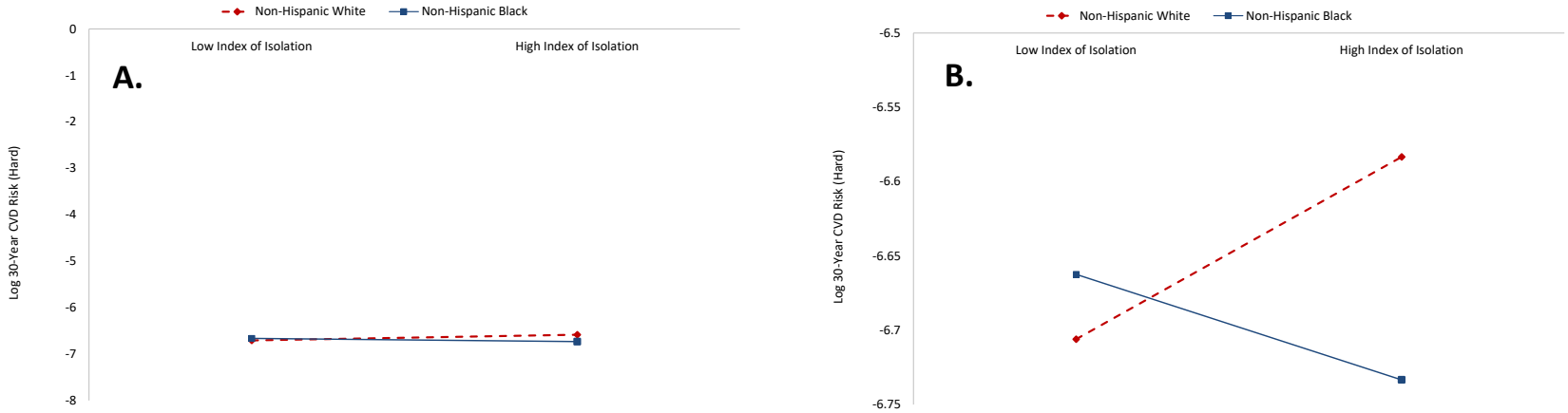
A. Original graphic with axis set with 0 as the high value. B. Zoomed in to see the relationship between the slopes

[a] General Estimating Equations (GEE) with the identity link, p-value for interaction=0.0375 (see Table 4.7). Interpretation: for each unit increase in Index of Concentration of Extremes (school) there is an increase in CVD risk of 11% among Blacks and a decrease of 5% for Whites.

[b] 30-Year full cardiovascular disease risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke, coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[c] Model includes exposure, race, age, sex, parental education, parental income, income inequality, urbanicity, % Black, % Latinx/Hispanic, % with <HS, % below Federal Poverty Level (FPL)

Figure 4. 6 Estimated Wave IV Log – 30-Year Hard Cardiovascular Disease (CVD) for Low and High Residential Segregation (Index of Isolation) at Wave I by Race, Add Health.



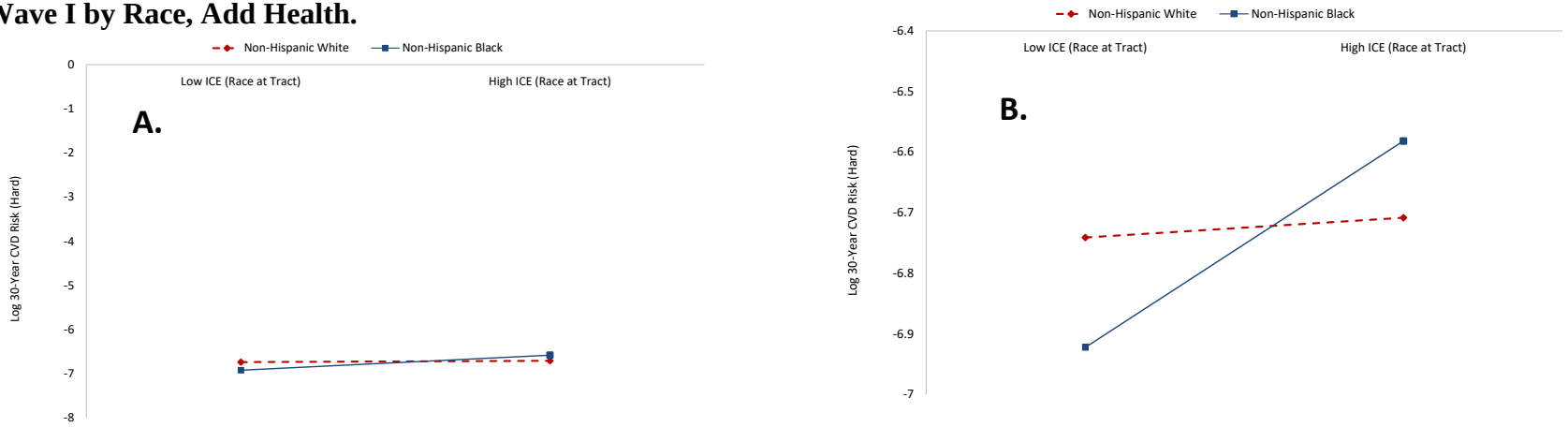
A. Original graphic with axis set with 0 as the high value. B. Zoomed in to see the relationship between the slopes

[a] General Estimating Equations (GEE) with the identity link, p-value for interaction=0.0682 (see Table 4.9). Interpretation: for each unit increase in Index of Isolation there is a decrease in CVD risk of 7% among Blacks and an increase of 13% for Whites.

[b] 30-Year hard cardiovascular disease risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[c] Model includes exposure, race, age, sex, parental education, parental income, income inequality, urbanicity, % Black, % Latinx/Hispanic, % with <HS, % below Federal Poverty Level (FPL)

Figure 4. 7 Estimated Wave IV Log – 30-Year Hard Cardiovascular Disease (CVD) for Low and High Residential Segregation (Index of Concentration of Extremes at Tract) at Wave I by Race, Add Health.



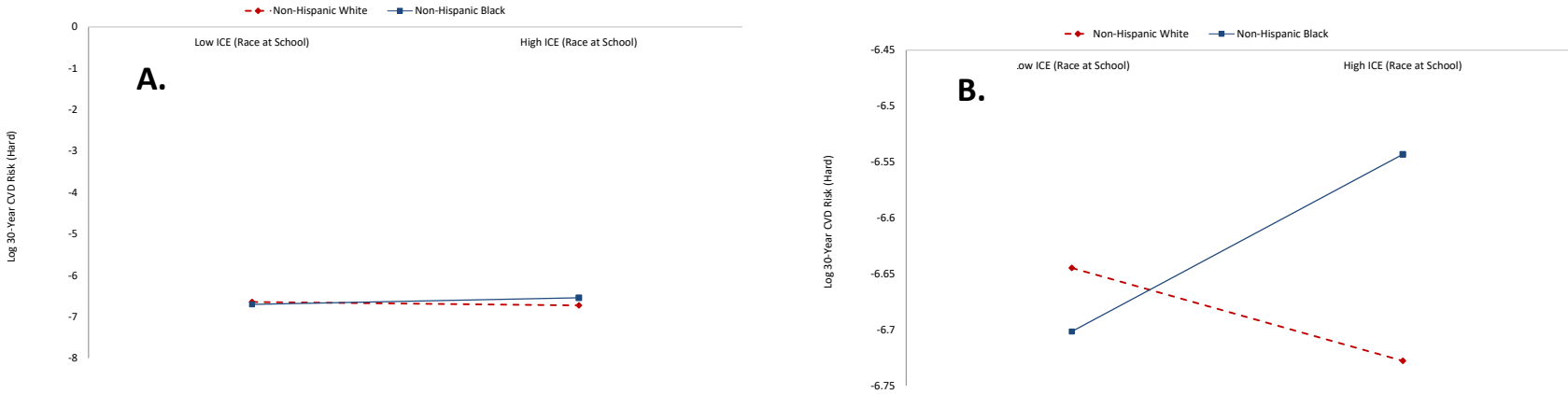
A. Original graphic with axis set with 0 as the high value. B. Zoomed in to see the relationship between the slopes

[a] General Estimating Equations (GEE) with the identity link, p-value for interaction=0.0181 (see Table 4.10). Interpretation: for each unit increase in Index of Concentration of Extremes (tract) there is an increase in CVD risk of 19% among Blacks and an increase of 2% for Whites.

[b] 30-Year hard cardiovascular disease risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[c] Model includes exposure, race, age, sex, parental education, parental income, income inequality, urbanicity, % Black, % Latinx/Hispanic, % with <HS, % below Federal Poverty Level (FPL)

Figure 4. 8 Estimated Wave IV Log – 30-Year Hard Cardiovascular Disease (CVD) for Low and High Residential Segregation (Index of Concentration of Extremes at School) at Wave I by Race, Add Health.



A. Original graphic with axis set with 0 as the high value. B. Zoomed in to see the relationship between the slopes

[a] General Estimating Equations (GEE) with the identity link, p-value for interaction=0.0491 (see Table 4.11). Interpretation: for each unit increase in Index of Concentration of Extremes (tract) there is an increase in CVD risk of 13% among Blacks and a decrease of 6% for Whites.

[b] 30-Year hard cardiovascular disease risk score predicts the risk of the following outcomes occurring within 30 years: coronary death, myocardial infarction, fatal & non-fatal stroke. The risk score is calculated using a Cox-Proportional Hazards model including the following variables in the prediction model: sex, blood pressure, hypertension medication use, diabetes status, smoking status, and body mass index (BMI); see Pencina et. al., 2009 for methods.

[c] Model includes exposure, race, age, sex, parental education, parental income, income inequality, urbanicity, % Black, % Latinx/Hispanic, % with <HS, % below Federal Poverty Level (FPL)

Chapter 5 Discussion: Low - Hanging Fruit: How Can We Change What We Can't See?

"We the People of the United States, in Order to form a *more perfect* Union, establish *Justice*, insure domestic *Tranquility*, provide for the common defense, promote the general Welfare, and secure the Blessings of *Liberty* to ourselves and our Posterity, do ordain and establish this Constitution for the United States of America."

~ Preamble of the US Constitution, 1787

The architects of American democracy understood that we, and our nation, were imperfect and as such provided us with a template to begin creating something that is "more perfect." This is where we are. Things get better with each passing year, but things can always get better and we can always do better. While we, as a nation and individuals, have not always ensured equality and social justice to all residents, that does not mean that we cannot *ever* provide full equality and justice to all. Our history of attempting to maintain a social hierarchy based on race has been extensively referenced in the previous chapters and summarized in Table 1.1 focusing only on the areas of civil rights, economics, education, housing and criminal justice. The three studies presented in this dissertation focus on two institutions formed by either racist policies, rulings, or customs: housing and criminal justice.

Study hypotheses and results are displayed in Table 5.1. Each study examines whether the health effects of exposure to structural racism depends on or differs by race. In this dissertation structural racism was measured or operationalized in three distinct ways. The first measure of structural racism was *in utero* exposure to the Flint Water Crisis emergency declaration, which was conceptualized as a racialized stressor experienced vicariously to Black mothers. The sample included Michigan women (outside of Flint) who were pregnant before and during the declaration of a State of Emergency. The second measurement was direct exposure to the police

through self-reported police encounters among a nationally representative sample of young adults. The final measure of structural racism was community- and school-level segregation, an inherently area-level measurement, among a nationally representative sample of adolescents entering adulthood. The main hypothesis of these studies was that the associations between these three distinct structural racism exposures and health outcomes will be more harmful to Blacks than Whites. This hypothesis is grounded in research and theory suggesting that, despite color-blind policies that on the surface appear to affect all people in the same way, the racially disparate impacts of the policies are simultaneously privileging Whites while disadvantaging Blacks.[6,7,11] Finally, these studies examined the association between three distinct exposures to structural racism and birth outcomes (Chapter 2) and 30-year CVD risk scores (Chapters 3 and 4).

In Chapter 2 I observed that the Flint Water Crisis (FWC) affected both Black and White women in a way that suggested poorer birth outcomes after the FWC for babies born to both groups of mothers. However, I observed a marginally significant relationship between exposure to the FWC and a lower gestational age among Black women with no change for White women. The association of the FWC declaration on gestational age appears to be more pronounced when the exposure occurred during the third trimester of pregnancy. There was no evidence that associations of the FWC on birthweight or size-for-gestational-age differed by race. In Flint, the environmental injustice of switching the water source to a contaminated local river without properly treating it exposed Flint residents to lead contaminated water directly for 1.5 years. The indirect exposure to the FWC for the Michigan's Black residents (and the US) began when the most egregious of the environmental injustice was long over and the governor declared a state of emergency in Flint in January 2016. After comparing birth outcomes in Michigan communities outside of Flint, I observed a significant difference between the gestational ages of babies born to Black and White mothers after the declaration compared to before the entire FWC occurred in 2013, where there was a significant decline in gestational age among babies born to Black mothers and no concurrent changes were observed among White mothers. Finally, I observed that there was a decline in gestational age each year under study for babies born to Black mothers, while gestational age remained virtually unchanged over the study period for White mothers. This is a trend that occurred nationally, suggesting that there may be effects of other

racially charged occurrences, such as police involved killings of Black and Brown people in the US, which were routinely occurring during the study period. While police involved killings of racial minorities was, and still is, frequently reported on by the news and social media, these were not the only racialized events occurring at this time. In addition to police involved violence, we also observed racialized mass shootings. Additionally, of particular concern to pregnant women was the Zika outbreaks in the Americas. Finally, 2015-2016 was a particularly contentious and racially charged presidential election cycle. Vicarious exposure to these stressors may have also contributed to poor birth outcomes.

In the second study (Chapter 3), I examined structural racism defined as individual experiences with the police. In this study I examined the association between a high number of police stops and the risk of a cardiovascular disease event occurring within the next 30 years. I observed that the association between a high number of police stops and 30-year CVD risk score differed by race, but not as hypothesized. Blacks with a high number of police encounters had a significantly *lower* 30-year CVD risk compared to Blacks with a low number of police encounters, while Whites with a high number of police encounters had a marginally significantly *higher* 30-year CVD risk compared to Whites with a lower number of police encounters. In ancillary analyses, men with a high number of police encounters had a significantly lower 30-year CVD risk compared to men with a low number of police encounters, while there was no difference in 30-year risk for women. Finally, Black men with a high number of police encounters had a significantly *lower* 30-year CVD risk compared to Black men with a low number of police encounters; White women with a high number of police encounters had a significantly *higher* 30-year CVD risk score than White women with a low number of encounters; and there was no difference for Black women or White men based on numbers of police encounters.

In the third and final study (Chapter 4), I examined area-level structural racism and 30-year CVD risk score. In this study, structural racism was measured as census tract-level residential segregation and school-level segregation using three standard measures (Index of Dissimilarity, Index of Isolation, and Index of Concentration of Extremes for race at the tract level) and one novel measure of school segregation (Index of Concentration of Extremes (ICE) for race at the school level). I observed no association between Index of Dissimilarity and the 30-year risk

score, and the risk did not differ by race. I observed that there was a marginally statistically significant difference in 30-year CVD risk score by race for each unit increase in the Index of Isolation such that among Blacks as residential segregation increased 30-year CVD risk *decreased*, while it *increased* for Whites. I observed a statistically significant difference in 30-year CVD risk score for ICE for race at the tract-level by race such that as residential segregation increased the 30-year CVD risk score increased for both Blacks and Whites, but more so for Blacks. Finally, I observed a statistically significant difference in 30-year CVD risk for each unit increase in ICE for race at the school level by race, such that as school segregation increased 30-year CVD risk increased for Blacks, but decreased for Whites.

Taken together, the findings reveal somewhat limited support for the hypothesis of this dissertation. Findings were in the expected direction, for at least some of the studies examining the effect of residential and school segregation (Chapters 2 and 4) and in an unexpected direction for the study examining criminal justice (Chapter 3). Each study measured structural racism in various ways (1) by comparing the time before and after a vicarious exposure to a racialized extended environmental injustice, (2) by evaluating individual experiences with police, and (3) by examining the effect of tract- and school-level segregation as disparate impacts of racist housing policies. These exposures were examined in relation to two outcomes: birth outcomes and 30-year CVD risk scores among young adults. Racialized stressors were found to be more harmful for babies born to Black mothers compared to White mothers and racial segregation in communities and schools was found to be more harmful to Black young adults than White young adults. In contrast, police encounters were found to be more harmful to White young adults than Black young adults, specifically White women.

5.1 The Policy Conundrum

The mixed results of this research suggest that measuring structural racism is extremely difficult. The difficulty lies in the fact that <u>everyone</u> is exposed to structural racism because federal policies, especially those that are color-blind, affect all citizens. Despite the changing definition of "citizen" over time, generally, the US Constitution is the foundation of all our laws, even locally. Thus, we are all exposed to some form of structural racism. Today this exposure is observed in the disparate impact of policies. For example, criminal justice policies are color-blind. They do not refer to any specific racial group in the written law and thus, on the surface, it

appears that all citizens will be held to the same standard. However, we know this to be untrue as we see the disproportionate incarceration rates between Black and White Americans.[35,624] As reported in Chapters 1 and 3, this is not an accident. Many scholars argue that the criminal justice system operates exactly as intended despite the color-blind laws.[35] This is because lawmakers were strategic in how the laws were written so that the language in the law differs by drug type rather than by targeting a specific racial group, which would easily be deemed unconstitutional under the 14th Amendment. With a closer look, we can observe who uses those drugs more frequently (at least when the laws were enacted). Then we begin to understand why the laws were written in this manner, and also why some racial groups suffer under color-blind policies more than others. This is not the only system that incorporated color-blind policies in this manner. In the 1930's several of the New Deal policies specifically refer to the exclusions of domestic and agriculture workers being exempt from Social Security benefits.[235] Again, on the surface, it appears that all domestic or agriculture workers will be unable to save for Social Security, however these sectors, at the time of the law, employed mainly Blacks, then later Latinx/Hispanics, limiting their ability to retire with some resources.[235] These examples can be referenced in many policies that have disparate impact or outcome by race (see Table 1.1). They also are one of the reasons why defining or measuring structural racism is so challenging.

Oftentimes, there are several policies or laws that seem as if they are not connected but contribute to similar disparate impacts. An example of this is the connection between residential segregation and schools. Housing disparities affect health in numerous ways, but housing also affects neighborhoods, environmental injustices, noise, access to care and other resources, school quality and segregation, and wealth, among others.[36] Thus when studying segregation, one is not merely examining housing but a range of potential health-related exposures.[36]

5.2 The Challenge of Measuring Structural Racism

Measuring the construct of structural racism is difficult. The majority race tends to benefit from federal and local policies while the minority race(s) are harmed by them.[7,43] Additionally, structural racism measures tend to focus on one structure or institution (e.g., education or housing) rather than several policies together (e.g., criminal justice, housing, education, economic opportunities, and voting). Most studies on structural or institutional racism focus on one institution,[73,118,177,178,266,460,591] while very few focus on several, examined

separately[63,77,87,88,91,625], or in an index.[89,626] Some researchers have examined racialized occurrences and/or vicarious racism and health.[75,78,79] These racialized occurrences (e.g., immigration raids, hurricanes, 9/11 attacks) are thought to be a form of structural racism because they either affect racial groups differently (i.e., immigration raids) or the response to such occurrences affect one race more than another (i.e., Hurricane Katrina). The themes of this collective literature is that the measures are area-based and are typically proxy measures, including measures of the disparate impact of policies. Rarely are there studies examining measures closer to the policies or a direct exposure to the policies.[627-632] Indeed, most studies fail to even mention the policies that caused the disparate impact under study (Needham et. al., forthcoming). Herein lies the challenge with studying structural racism and thus comparing findings across place and time.

A reasonable public health approach has generally been to take advantage of existing data resources to leverage what we already have available. Recently there have been calls to enhance the data we already collect with contextual variables over space and time so that at a minimum institutional racism can be evaluated in longitudinal studies.[633] This call to action is a reasonable approach to investigating the effects of structures and institutions, especially in long term studies that include diverse participants. However, this implies that the data is available and that it can be linked to such studies, which is not always the case.[166,168,173] For example, in the evaluation of criminal justice, we do not have a mechanism to measure police use of force, nor do we have a mechanism to quantify routine police encounters, especially those that do not result in a ticket or an arrest.[519,520] Nor do we have data available at a local level, in most cities, with the exception of Chicago and New York, which have released citizen complaint data[g] (Chicago) or track stop-and frisk data (New York)[h]. While citizen complaint data is an attractive source of data it is likely substantially underestimating police encounters.[634] With the exception of the New York data, the available data are difficult to include into health studies.

Another understudied area of racism is vicarious or indirectly experienced racism. Much of this research capitalizes on seemingly random racialized occurrences by conducting natural experiments to estimate the harmful effect of being exposed to a racist encounter[78] (e.g.,

[g] https://beta.cpdp.co/
[h] https://www.nyclu.org/en/stop-and-frisk-data

immigration raid), a natural disaster[402,635] (e.g., Hurricane Katrina), or a manmade disaster[79] (e.g., 9/11 attacks). Additionally, a recent study suggests that exposure to state-level number of police involved killings of unarmed Black men and women have a detrimental association with individual-level mental health.[75] Taken together, this literature suggests that higher exposure to vicarious racism is harmful to the health of racial and ethnic minorities.[377,636,637]

5.3 Racism as a Public Health Issue

This dissertation contributes to a larger discussion on racism that has been occurring in recent months. There has been an abundance of occurrences of police involved violence against unarmed and/or subdued men and women of color reported in both traditional and social media outlets. We have seen the effects of racism played out in the streets, literally daily. Additionally, the current Covid-19 pandemic has illustrated how the structures of this country have failed racial minorities. We have observed substantial racial disparities in Covid-19 infections often citing exposure level differences (i.e., essential worker professions or higher levels of chronic conditions), but it could also be related to structural factors as well such as differential quality in education that leads to essential worker status, or over crowded or substandard housing availability.[638] This is an important area of future research. Understanding the structural effects of racism on the pandemic may provide the evidence needed for improvement. With a vaccine in hand to fight new infections, researchers point to vaccine hesitancy as the reason why people will not take the vaccine. Rarely has structural racism entered the conversation to understand *why* people may not be able to get, or even want, the vaccine. So is it really hesitancy or lack of access to care or lack of access to resources that would enable one to schedule a vaccine, such as internet access/proficiency, ability to take time off of work, child care…etc.?[639] Additionally, it seems that if there is hesitancy, given how the medical field has historically treated Black and Brown Americans, it is certainly justified.[640] Yet, combating these disparities has been challenging with limited access to funding and avenues for publishing empirical findings on the effects of racism and health.[i] These three studies taken together with the broader literature on discrimination and health begs the question what can be done about it?

[i] https://www.healthaffairs.org/do/10.1377/hblog20210415.305480/full/

Prior to the Covid-19 pandemic, there has been an influx of local governments declaring racism as a major public health crisis. Indeed, the first local government to do so was Milwaukee County, Wisconsin (4/4/2019).[j] This declaration came after understanding that this county had some of the largest racial disparities in incarceration, wealth inequality, and other social factors. As an attempt to solve the disparate impacts of prior policies from a governance point of view, the declaration required that any new policy must include an evaluation of how various groups would or could be differentially affected. The American Public Health Association has catalogued the declarations across the US of which there are 194 declarations as of April 2021 at several levels of government.[k] The effect of these declarations is unknown and ripe for research. This is certainly an avenue to extend the findings of this dissertation.[39]

5.4 Translating Findings for Public Health Practice

Given that the effects observed in this study are relatively small and inconsistent, the results here are not ready for translation. First, we need more data. Specifically, we need accessible data on police interactions that can be linked to health data. The data we have available to us include private administrative records that are closely held by police agencies that include citizen complaints made against the police, assuming the police jurisdiction has a mechanism for citizens to complain.[434] These data are critical to our understanding of police interactions and use of force, yet few local police agencies have made this data available to the public.[l] Police agencies routinely aggregate their data and report several administrative factors through the Law Enforcement Management and Administrative Statistics survey.[m] Here we can get a broad look at excessive force complaints. But these data cannot be linked to health data. Another source is a national survey called the Police and Public Contact Survey which is funded by the US Department of Justice. While this survey is nationally representative, it does not over-sample people most likely to be victimized by police, nor does it allow researchers to link the survey to local contextual or health outcome data sources where interventions and policy changes can make a lasting impact.[n,o,p]

[j] https://www.wbur.org/onpoint/2019/05/30/milwaukee-racism-public-health-crisis
[k] https://www.apha.org/topics-and-issues/health-equity/racism-and-health/racism-declarations
[l] https://beta.cpdp.co/
[m] https://www.icpsr.umich.edu/icpsrweb/ICPSR/series/00092
[n] https://www.icpsr.umich.edu/icpsrweb/ICPSR/series/95
[o] https://www.icpsr.umich.edu/icpsrweb/ICPSR/series/95/studies/34276?archive=ICPSR&sortBy=7
[p] https://www.bjs.gov/index.cfm?ty=dcdetail&iid=245

With this limited data, studies examining police have relied on limited and relatively inaccurate data available on fatal police encounters (Table 5.2)[168,173,472,473] or injuries due to police intervention.[73] It has become apparent that the most accurate data on violent police encounters is crowd-sourced by journalists. While this data records the deaths due to legal intervention, they do not necessarily track what happens to the officer after the death of a citizen. Additionally, given that most police encounters are non-violent, we need a dataset tracking use of force in a meaningful way so we can understand its effects on health outcomes and identify ways to intervene to change the practices of harming the public. Notably, New York City began to require data collection on stop and frisk activities in 2002 reaching a height of nearly 700,000 police stops in 2012.[q] The New York Civil Liberties Union filed a lawsuit that illustrated the racially disparate practices of the New York Police Department, and the practice of stop-and-frisk began to dramatically decline to about 13,000 stops in 2019. This suggests that there is substantial power in gathering data as evidence against unfair practices stemming from structural racism. If this data did not exist, it is likely that the trend would have continued to rise.

Police data is not the only data that is lacking. A major disparate impact with respect to structural racism is wealth inequality and intergenerational wealth. Homeownership is one way in which people build wealth, but it isn't the only way.[36] Having reliable data on income and assets would assist researchers in understanding the effect of both race and income inequality on health. This is vitally important to advance the field's understanding of wealth, income, and intergenerational wealth effects on health outcomes.

While examining the epidemiology of structural racism is a burgeoning field within public health, some evidence is building that suggests a revolutionary change in policies can have a lasting effect on heath. Krieger and colleagues reported that once Jim Crow was abolished by the Civil Rights Act of 1964 there was a significant decline in premature mortality (or deaths prior to age 65 years old)[92], infant mortality[175], and estrogen-receptor negative breast cancer diagnoses in those states.[93] Taken together, these results suggest that major shifts in policy can improve health outcomes, although more work is needed to understand which policies produce the strongest health effects.[641]

[q] https://www.nyclu.org/en/stop-and-frisk-data

5.5 Future Directions

This dissertation aimed to understand the effect of criminal justice and residential segregation on health outcomes. The main limitation in these collective studies is that the measures of structural racism available were limited, particularly in the use of the Add Health dataset. This suggests that longitudinal studies need to incorporate more data that is already available so that structural racism can be measured at least at a proxy or indirect level. A public health approach is to enhance what we have available and not waste resources recreating it. This applies to research as well. We collect annual cross-sectional health survey data and federally fund major longitudinal studies with many participants followed over decades. These studies are strong contenders for incorporating contextual data and data on policies, especially over time.

Another area for future research is to develop a set of measures that consistently and reliably measure disparate impacts of policies, court rulings, and local customs. Currently, researching structural racism mainly revolves around examining the downstream effects of policies within one institution, such as residential segregation, which evolved over time, rather than effects of many policies (Table 1.1) and local customs and practices. Policies of segregation were enacted regionally (e.g., Plessey v Ferguson ruled by the Supreme Court, or Jim Crow in the South), nationally (e.g., the Home Owner's Loan Act, the federal law that created redlining), and locally through *de facto* practices (e.g., block busting or contract lending). These "policies," loosely defined as state enabled rules, can be indirectly measured by population estimates in the areas and how they compare to the surrounding areas. But this is an effect of the policy, not the policy itself. Thus, more work is needed to define the policies and link them to direct measurement (i.e., the actual policy) and indirect measurement (e.g., indices of residential segregation). Additionally, as noted throughout this research it is clear that structural racism occurs through many policies that intersect. Quantifying the intersecting policies is an area for future research. In any study examining structural racism, the explicit policy or policies must be named. Often, policies change or are eliminated over time, thus we need consistent measurement, but also measurement over time to evaluate the benefits and harms of these changes.

Additionally, there is little research on vicarious experiences of structural racism. Vicarious racism is defined as observing racism occurring to someone else (i.e., friends, family, neighbors, or even strangers).[349] While not the only method,[74] research on vicarious structural racism has been conducted using before and after types of study designs where a major racialized event

occurs in a location, such as the Flint Water Crisis (Chapter 2). Researchers can examine a period prior to the racialized occurrence and compare it to after with the assumption that these events are random in nature and thus are quasi-experimental or randomized controlled trial - like. Vicarious racism has been found to be associated with mental health[75,642,643] and birth outcomes.[78,79] More work is needed to understand effects of vicarious exposure to racism on various health outcomes. The Flint study in this dissertation follows suit with similar studies such as comparing before and after a discrete immigration raid, which included babies born to White mothers as the control group and babies born to Latinas as the exposed group. To enhance the causal inference of such studies econometric methods, such as a difference-in-difference analytic approach, may be useful to examining a vicarious exposure to a major shock like the FWC. [644] The method assumes that the "common shocks" assumption is met, which states that any other event occurring before, during, and after a racialized occurrence (i.e., the exposure) must equally affect the groups.[644] The latter assumption makes using this method challenging with some exposures, as structural racism affects all people, but in theoretically opposite directions, making this assumption potentially difficult to meet while examining racial disparities. This is an area for future research. Indeed, while racialized occurrences are random, there are similar, and likely traumatic, racialized occurrences happening routinely and at frequent intervals (Figure 2.7). A challenge future research must tackle is to disentangle these occurrences from one another to identify if it is one source of stress or a near constant barrage of stressors through routine racialized stressors that affects health.

Examining discrimination, structural racism and internalized racism together is another potential future direction. Logically, we need to understand the cycle of racism. Does discrimination cause structural racism? Or the other way around? Which form of racism causes internalized racism? Perhaps this relationship has changed over time. Or perhaps, these constructs are too correlated with one another. In the early formation of America's democracy it is possible that discrimination caused structural racism through policies and court rulings, whereas now, structural racism may be driving discrimination through stereotypes and stigma resulting from policies and court rulings.

Finally, there is a field within public health that is growing called "Legal Epidemiology." This multidisciplinary field examines laws and policies and their relationship to health outcomes. The

multidisciplinary approach allows researchers who typically work in silos to work together with a mixture of expertise in laws and research methods for empirical studies.[645-647] This approach combines legal expertise through public health law and practice (i.e., counsel, representation, and research) and epidemiology with a focus on laws or legal practices as potential causes of disease or health.[645] For example, throughout this dissertation laws are not the only "policies" affecting health. The US Supreme Court rulings also have implications for health (e.g., Jim Crow with the ruling of Plessey v Ferguson). Additionally, there are *de facto* customs that also may impact health (e.g., not informing a newly released prisoner that his voting rights have been restored).[r]

5.6 Conclusion

As I began this dissertation with a nod to the US Constitution I will end it as such. The preamble of the US Constitution implies that the Union is imperfect. It is upon us to find ways to make the Union better for all. As I reflect on the findings of this dissertation, I have realized that not all are treated equally and that we can (and will) do better.

[r] https://www.aclu.org/other/de-facto-disenfranchisement-introduction

5.7 Tables

Table 5. 1 Summary of Dissertation Hypotheses and Findings.

Policy	Exposure and Outcome	Hypothesis	Methods Summary	Evidence
Residential segregation led to disinvestment, lower tax base, and city manager appointment	Exposed = babies born in the 37 weeks after the declaration of a state of emergency in Flint Jan 2016; Unexposed = babies born the same time periods in 2013-2015. Outcome: birthweight (grams), gestational age (week), size-for-gestational age (z-score)	There will be a decrease in birthweight, gestational age, and size-for-gestational age for babies born to Black and White mothers in Michigan (but outside of Flint) in the 37 weeks following the Flint Water Crisis declaration of a State of Emergency in Flint compared to the same time in the 3 prior years. The effect of the FWC declaration of a state of emergency in Flint will be modified by race in that babies born to Black mothers will have a larger decrease in birthweight, gestational age, and size-for-gestational age, while babies born to White mothers will have a smaller decrease in birthweight, gestational age, and size-for-gestational age during the same time periods.	Quasi-experimental design, all singleton live births born to non-Hispanic Black and non-Hispanic White mothers using birth records from Michigan (excluding Flint), difference-in-difference, tested race, exposure, and their interaction while controlling for covariates.	In nearly all analyses, we found statistically significant associations between exposure to the FWC and birth outcomes suggesting that both Black and White mothers were affected by the FWC. (Tables 2.2-2.11, Figures 2.6-2.7) The interaction between race and exposure was marginally significant after controlling for covariates but only for gestational age, suggesting that gestational age declined for Black women, while staying the same for White women after exposure (Table 2.2) and when comparing births from 2013 and 2016 (Table 2.4). A declining trend for gestational age was observed in MI and the US (table 2.7) suggesting that the FWC in addition to other racialized stressors may be affecting birth outcomes.

War on Drugs; criminal justice	Exposure: high self-reported police street encounters (PE) (6+ for men, 2+ for women) measured at Wave III. Outcome: 30-year CVD risk score measured at Wave IV.	The association between a high number of reported PEs and 30-year CVD risk will depend on race, such that the effect of exposure to a high number of PEs on risk for a CVD event occurring in the next 30 years will be higher for Blacks compared to Whites.	Cross sectional study, non-Hispanic Black respondents and non-Hispanic White respondents in the National Longitudinal Study of Adolescent to Adult Health, complex survey design. Linear regression analysis accounting for non-response, unequal probability of selection, and post stratification, with sub-groups via domain analysis.	The relationship between a high level of police encounters is dependent on race, but not as hypothesized (Tables 3.2 and 3.3). Whites have a higher risk with a higher level of PEs, while Blacks have a lower risk with a higher level of PEs. In sensitivity analysis, men have lower CVD risk with higher number of encounters, while women have no change (Table 3.5). Black men have lower risk and White women have higher CVD risk with higher number of encounters, but Black Women and White man had no difference in risk between high and low numbers of encounters Table 3.6).
Residential and school segregation	Exposures: Tract level index of dissimilarity, index of isolation, and index of concentration of extremes at both the tract and school levels measured at Wave I. Outcome: 30-year CVD Risk Score measured at Wave IV.	The association between community-level and/or school-level segregation and 30-year CVD risk will differ between Blacks and Whites Among Blacks, being in communities and schools with higher segregation will be associated with an increased 30-year CVD risk score, whereas among Whites, being in communities and schools with higher levels of segregation will not be associated with an increased 30-year CVD risk score	Cross-sectional design; GEE analysis, accounting for non-response, unequal probability of selection and post stratification; subgroup analysis via domain analysis	No association between Index of Dissimilarity and 30-year CVD risk Score; marginal relationship between Index of isolation and 30-year CVD risk score (Tables 4.3, 4.4, 4.7, 4.8.) The ICE scores for tract and school were dependent on race such that the relationship for ICE at the tract level suggested a harmful risk for both Blacks and White, but more so for Blacks (Tables 4.5 and 4.9). The ICE at the school level suggests that as school segregation increase the 30-year CVD risk increases for Blacks but decreases for Whites (Tables 4.6 and 4.10).

Table 5. 2 Number of Deaths Due to Legal Intervention by Source and NYC Stop-Frisk and Search Encounters, 2015-2016.

Name	Type	Source	2015	Compared to CDC (2015)	2016	Compared to CDC (2016)
CDC/Vital Records	US surveillance	CDC - Wonder	500	---	527	---
The Counted	Journalist driven	The Guardian	1,146	2.29	1,093	2.07
The Counted (shot by police)	Journalist driven	The Guardian	1,017	2.03	1,011	1.92
Fatal Force (shot by police)	Journalist driven	Washington Post	995	1.99	958	1.82
Killed by Police	Private citizen	Crowd-sourced databases, media, obituaries, social media, criminal records, police reports, & others	1,222	2.44	1,171	2.22
Mapping Police Violence	Private organization		1,187	2.37	1,129	2.14
Fatal Encounters	Journalist driven	Media & police reports	1,595	3.19	1,587	3.01
NYC* Stop and Frisk Registry	Police database	NYPD**	22,565	na	12,404	na

*NYC=New York City, NYPD=New York Police Department

Appendix: Framingham 30-year Cardiovascular Disease Risk Index Modified for the Use of Add Health.

The Framingham 30-year Cardiovascular (CVD) Risk Prediction Index, introduced by Pencina et. al. (2009), was used as the dependent variable for this study.[491] This index was selected for two reasons. First the Add Health population at Wave IV range in age from 24-32 years, thus this group has yet to experience many CVD outcomes, however many have begun developing the habits that can lead to CVD. Additionally, there are several versions of CVD risk prediction indices, we selected the Framingham CVD Index because it is based on a younger population, somewhat consistent with the Add Health population, compared to other risk indices.[491]

Briefly, the Pencina et. al. paper describes four risk models based on 30-years of Framingham data. These models predict either (a) coronary death, myocardial infarction, fatal & non-fatal stroke [hard CVD outcomes] or (b) those in (a) plus coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, and congestive heart failure [full CVD risk].

All models include a combination of sex, age, systolic blood pressure, anti-hypertensive treatment, smoking, and diabetes. The use of total cholesterol, HDL, and BMI distinguishes two of the models from the others. Each outcome option (a) or (b) includes two variations that strongly predicts the 30-year CVD risk. Variation 1, called the 'Main Model' uses all variables except BMI, and variation 2, called the 'Simple Model' substitutes BMI for lipid levels. The 'simple model' contains variables that are easily gathered at an office visit.

To generate the Framingham 30-year CVD risk index (CVD Index)-Main Model using Add Health data, some modifications would have been necessary. Jeanne et. al. (2018) used the 'Main Model' predicting all cardiovascular outcomes because Add Health participants have a low incidence of cardiovascular events. Add Health includes measurements for all of the components of the 30-year risk score: sex, blood pressure, hypertension medication use, diabetes

status, smoking status, body mass index (BMI), and cholesterol levels. However, cholesterol levels are reported in deciles rather than values. Deciles were reported, as opposed to the measured values, due to potential bias in the assay technology, as such kept the individual values remain unavailable.[648] Additionally, the blood was taken in the field, while systematic methods were used to gather the blood, the participants were not expected to fast.

Studies have used Add Health data and the Framingham 30-year risk score in other studies. For example, Jeanne et. al. (2018) substituted average cholesterol levels from the National Health and Nutrition Examination Survey for the decile values in Add Health.[540] While, Wright et. al. (2018) incorporated the Framingham 30-year risk score using the BMI values measured at the time of the interview, as well as estimated a meaningful cut-point to use in logistic models.[534,535] In order to include all of the data provides by Add Health participants, this study uses the Framingham 30-year risk prediction model with measured height and weight to calculate BMI (Simple Model).

SAS macro code used to generate the individual risk score was provided by Dr. Pencina and Mr. Williams from Duke University and Kenanco Biostatistics, respectively. The code provided included four macros which predicted cardiovascular risk in 30 years using a Cox proportional hazards model that accounts for competing causes of death using data from the Framingham Heart Study. The Cox model included the following covariates: age, sex, use of antihypertensive medications, smoking status, diabetes status, systolic blood pressure, and body mass index.

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