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Epidemiology of Childhood Adversity: Centering Multiracial Americans in a Critical and Computational Analysis

By

Tracy Lam-Hine

A dissertation submitted in partial satisfaction of the

requirements for the degree of

Doctor of Public Health

in the

Graduate Division

of the

University of California, Berkeley

Committee in charge:

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Epidemiology of Childhood Adversity: Centering Multiracial Americans in a Critical and Computational Analysis

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Abstract

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Doctor of Public Health

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According to the 2020 United States (US) Census, Multiracial people - those who identify as two or more races - grew faster than any other racial group in the US. Despite this trend, research, scholarship, and knowledge on the health and well-being of this population has struggled to keep pace with this growth, and the health of Multiracial people is still not well-understood. Early research from psychology and sociology focused on risk and resilience factors associated with individuals' Multiracial identities. However, patterns emerging from both national health statistics as well as etiologic health research suggest that Multiracial people experience elevated rates of poor cardiometabolic, respiratory, mental, and behavioral health compared to monoracial (single-race) groups. In California, the state with the largest Multiracial population, they are more likely than monoracial White people to be living in poverty, disabled, and lacking health insurance. In addition to these health outcomes, descriptive studies of large nationally-representative datasets show that Multiracial people have the highest mean adverse childhood experiences (ACEs) score. ACEs, also sometimes referred to as childhood or early life adversity, are traumatic events occurring during childhood or adolescence, and are associated with numerous physical, mental, and behavioral health outcomes. Several of these outcomes disproportionately affect Multiracial people, and are leading causes of death in the United States. The goal of this dissertation is to marry knowledge and methods from disparate disciplines in describing the epidemiology of childhood adversity, while centering the health experience of Multiracial Americans.

In chapter 2 of this dissertation, I examine whether the high mean ACE score in the Multiracial population is driven primarily by Multiracial people with American Indian/Native American (AI/NA) ancestry, the largest subgroup of Multiracial people. AI/NA populations also have a high mean ACE score, possibly reflecting the legacy of occupation of Indigenous land and structural racism affecting generations of AI/NA peoples — what I call the "land occupation" hypothesis. This study found that estimates of mean ACE scores and risk of

most ACE components were not significantly higher for Multiracial people with AI/AN ancestry than those without. These results suggest that the high mean ACE score is likely due to factors independent of the land occupation hypothesis. Further research should investigate causes of the disparities in ACEs between Multiracial and monoracial groups.

Chapter 3 of this dissertation investigates racial differences in the association between ACEs and some of the health outcomes disproportionately affecting Multiracial people, including metabolic syndrome, hypertension, asthma, anxiety, depression, suicidal ideation, and problematic drug use. Like in other interaction analyses, a goal of this study is to identify populations most affected and thus most urgently in need of intervention. Associations between ACEs and mental and behavioral health outcomes were strongest across racial groups. While Multiracial people do experience stronger associations between ACEs and anxiety than other racial groups, overall, the strength and direction of association varies by outcome, group, and interaction scale, with no group uniformly experiencing stronger associations than others.

Chapter 4 discusses some limitations to interaction analyses such as the one in chapter 3, and offers stochastic substitution methods as an alternative approach with some important benefits for health disparities research. I simulate a stochastic health equity intervention that provides an answer to the question: "how many cases per 100 of depression would be averted in the population if the ACEs distribution of each racial group approximated that of the White group?" Results from this simulation show that the Multiracial group would benefit the most from such an intervention, a finding that would be masked if only interaction effects were considered.

Childhood adversity contributes significantly to poor health among the Multiracial population. Prevention of ACEs could be an important and effective health equity strategy to reduce disparities affecting this group. More research is needed to identify why Multiracial children are predisposed to experiencing adversity, as is greater attention to the patterns of socially-constructed risks that contribute to the unique health experience of Multiracial people in the US.

To Ow Wai Kuen, my poh-poh,
至：區慧娟，我婆婆，

with whom I shared a wall as child, and who unknowingly instilled in me a fire and desire to connect with the unspoken history of my family. 希望你安息駕鶴西歸，於天堂安享。

May she and the ancestors of all those advancing justice protect us, as we work piece by piece to dismantle systems of prejudice, discrimination, and oppression.
我們會一步一步地拆解結構性壓迫和種族歧視，愿婆婆與前世祖先保佑我們。

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Chapter 1

Introduction

Modern biomedical science has long been preoccupied with justifying a biological conception of race. The origin of such claims can be traced to seventeenth-century European epistemology, where early anthropologists adopted the emergent scientific practice of classifying natural objects, categorizing them into taxonomies reflecting perceived similarity and difference [1]. It is well-accepted in social science disciplines that race is not a biologically meaningful category, but rather an artificial and ever-evolving representation of social hierarchy [2]. Social epidemiologists, or those who espouse a social-constructivist worldview in studying patterns of health and disease, have upended biological conceptions of race in medicine, arguing that social structures — and not biology — are primarily responsible for racial disparities in health [3].

From this perspective, understanding the causes of racial disparities requires a precise definition of "race" and its constituent categories, and a nuanced perspective on the social processes that determine such disparities. Unfortunately, the majority of modern health sciences research handles both of these tasks clumsily [4]. A salient example is the paucity of research on the health of Multiracial people, or those who identify as two or more races. Health researchers have largely ignored Multiraciality, resulting in a large knowledge gap on the health status and risks of this population [5–7].

The aim of this dissertation is to center the health experience of Multiracial Americans, exploring childhood adversity — an exposure disproportionately affecting this population — and to offer new approaches and methods for epidemiologists interested in improving health research on Multiracial people.

Who are Multiracial Americans?

The 2020 US Census marks the third consecutive decennial Census that has allowed people to identify as more than one race. Although this practice has now become a regular feature of most demographic forms in the US, strong cultural, institutional, and methodological preferences exist for aggregating such demographic data into neat population-level

racial groups [8]. The choice of algorithm for categorizing Multiracial people into discrete categories (e.g. a distinct "two or more races" category, reassignment into monoracial groups, or into an "other" category) can significantly alter statistical findings and study conclusions [7]. This practice has a long history in the US, and can be thought of as an institutional form of *monoracism*, racism that privileges monoracial identification and devalues Multiracial experiences [9]. Multiracial individuals may also change their racial identity over time, at times identifying as monoracial and at other points in life as Multiracial, making precise enumeration of this group even more challenging [10, 11].

Despite these difficulties, a clearer picture of the Multiracial population in the US is slowly emerging. Multiracial people form the fastest-growing segment of the US population, increasing 276% from 9 million (2.9%) to 33.8 million (10.2%) of the population between the 2010 and 2020 Censuses [12]. The Multiracial population is now the fourth-largest racial/ethnic group in the US, behind White, Latine, and Black populations and larger than the Asian population in size, although the Census attributes much of this growth to improvements in the measurement of race [12]. Multiracial Americans form more than 10% of the population in twelve states, with the greatest proportion (25.3%) in Hawaii and largest numbers (5.7 and 5.1 million, respectively) in California and Texas. While the 2010 Census reported that the largest Multiracial subgroup was those identifying as White and Black, the 2020 Census reports that it is those identifying as White and "some other" racial group [12, 13]. Other sources suggest that almost 70% of Multiracial people claim some American Indian/Native American (AI/NA) ancestry, with 50% alone identifying as White and AI/NA [14].

A major feature of the US social structure is its stratified racial hierarchy, with Whites systematically and structurally advantaged compared to people of color [2]. Because Multiracial people do not fit neatly into singular racial categories, and because they may simultaneously claim membership in structurally advantaged and disadvantaged racial groups, they occupy a unique position in the US racial hierarchy [15, 16]. Much of the literature on the historical racial classification of Multiracial people describes the practice of "hypodescent", in which Multiracial people are classified as the lowest-status racial group with which they identify [17, 18]. More recently, some researchers have proposed an "upward iteration" pathway by which part-White Multiracial people more closely identify with and receive treatment similar to White people [19]. These theoretical pathways and hypothesized position in US society are important for contextualizing health disparities facing Multiracial people [5].

History of racial classification in the United States

In the United States (US), a history of European colonial occupation, invasion of Indigenous land, and the trans-Atlantic slave trade has created a uniquely racialized society built on the exploitation of people of color [2]. The American economic system, with its explicit valuation and commodification of human bodies, created a need for identifying, enumerating, and differentially controlling people based on racialized phenotypic categories [20].

Beginning around the time of the Civil War, the officially-defined "racial" categories used in the US Census expanded to include "mulatto" groups, further dividing this population into "quadroons" and "octoroons" [18]. Although these categories were used to describe Multiracial people of Mixed White, Black, and/or AI/NA descent, much of both scholarship on and popular discourse about Multiraciality centered around Mixed Black-White people, given the historical Black-White divide in the US [21].

In the 1930's, the US Census adopted the one-drop rule, which instructed enumerators that: "a person of mixed White and Negro blood was to be returned as Negro, no matter how small the percentage of Negro blood" [18]. The one-drop rule is the clearest manifestation of hypodescent in America, a social phenomenon produced by deep racial anxieties related to protection of White "purity," and resulting in the monoracist classification of all Multiracial people as non-White. This fear was also a primary motivating factor behind the passage of state-level anti-miscegenation laws in up to half of US states, the first of which was passed in Virginia in 1661, and the last of which was struck down by Loving v. Virginia in 1967 [20].

The years immediately following the Civil Rights movement saw concurrent growth of a visible Multiracial population, assertion of Multiracial identities distinct from other communities of color, and emergence of colorblind and post-racial ideologies. Some among the Black community viewed Multiracial Black people's assertions of this new Multiracial identity as political abandonment of Blackness and a realignment with Whiteness [22, 23]. Neoconservatives and neoliberals quickly capitalized on this growing rift between monoracial and multiracial communities of color [9]. Like the positioning of Asian-Americans as "model minorities" to stigmatize Black people, Multiracial people were heralded in American society as futuristic harbingers of a new post-racial order defined by race-neutral policies [2, 16, 22, 24–27]. These stereotypes firmly established the racialization of Multiracial people as abnormal, with part-White Multiracial people simultaneously not White enough to claim Whiteness because of hypodescent, and not minority enough to fully claim their ancestral heritage because of upward iteration.

Distrust of people asserting Multiracial identities became institutionalized in resistance to growing pressure in the 1980s on the federal government to acknowledge the Multiracial population. In the years leading up to the 2000 Census, disagreement about how to count the increasing numbers of multiracial people in the US pitted traditional Civil Rights groups against newly formed multiracial organizations over ownership of the bodies of the multiracial population, and by extension, funding appropriations from the Civil Rights Act [28]. The culmination of many hearings on the proposed Census changes resulted in an outcome that neither Multiracial or Civil Rights organizations wanted: the "check all races that apply" data structure that now governs racial data collection in the US [28]. Two decades have now passed since the introduction of federal recognition of Multiraciality, yet most quantitative researchers continue to search for ways to reassign Multiracial people into monoracial categories [29–31].

Health of Multiracial people

Multiracial adults experience significantly greater prevalence of heart diseases (17.7 vs. 11.1%), asthma (25.9 vs. 13.2%), obesity (37.2 vs. 30.9%), hopeless feelings (8.5 vs. 4.9%) and serious psychological distress (7.4 vs. 3.8%) compared to monoracial people [32]. In California, the state with the largest Multiracial population, Multiracial adults are significantly more likely to be living in poverty, lack health insurance, and in poor physical health than monoracial White people [33]. Early life stage health disparities between Multiracial and monoracial populations are well-documented, including increased risky health behavior, poor psychological adjustment, worse mental health, and more sleep problems [11, 34–38]. However, research to date has not investigated connections between these patterns, leaving major gaps in the understanding of life course progression and health trajectories of Multiracial people.

Studies exploring negative health outcomes among Multiracial people often discuss the complexities of identity formation for Multiracial people, and how challenges of integrating multiple racial identities in adolescence can lead to poor social and psychological adjustment, which are in turn associated with poor health later in life [34, 38–40]. This history of investigating identity development within individuals as a source of health disparities reflects the fact that much of the early research on Multiracial people came from the field of psychology, with fewer studies originating from a critical sociological perspective [5, 41, 42]. However, Multiracial identity development cannot be separated from the social forces that shape the development of such identities [6]. Poor social and psychological adjustment leading to adverse health outcomes should be contextualized as a result of monoracist social norms, rather than as purported individual-level psychosocial "deficits" unique to Multiracial people.

One little-known statistic that may offer clues to understanding Multiracial-monoracial health disparities is the striking level of childhood adversity reported by Multiracial people in large, national health surveys such as the Behavioral Risk Factor Surveillance System (BRFSS). Several analyses of BRFSS data have shown that Multiracial people have the highest mean adverse childhood experience (ACE) scores among all racial groups [43, 44]. While other factors likely also contribute to Multiracial-monoracial health disparities, ACEs are strongly associated with increased risk of a variety of physical, mental, and behavioral outcomes, and are thus an important exposure and potential source of health risk for Multiracial people.

ACEs are just one form of exposures that result in what some have called "toxic stress" [45]. In general, stressful events activate the hypothalamic-pituitary-adrenal (HPA) axis, sympathetic-adrenal-medullary (SAM) axis, and consequently, the sympathetic and parasympathetic autonomic nervous system [46, 47]. While the "fight or flight" stress response has an evolutionary self-defense function, chronic or prolonged exposure to its effects can lead to physiologic dysregulation across multiple bodily systems [47]. Evidence from genomic and neuropsychobiological research further suggests that childhood adversity is associated with dysregulated epigenetic processes such as DNA methylation, which in turn can damage physical, mental, and immune function [48–50]. Simultaneously, toxic stress may cause adoption

of unhealthy behaviors, in particular those linked to elevated behavioral impulsivity and maladaptive coping such as substance use, disordered eating, and sexual risk-taking [51].

Although disparities in the prevalence of ACEs are well-documented, hypotheses about the causes of disparities are rare. Structural racism is occasionally offered as an explanation for disparities in ACEs between monoracial groups, but it is unclear how structural racism affects Multiracial populations distinctly from other groups of color. One potential hypothesis is stress from racism and prejudice burdening interracial couples and their Multiracial children [52]. This reasoning seems unlikely, however, to fully explain the disparities in exposure to ACEs and associated poor health outcomes. Further research is needed to explicate the pathways that predispose Multiracial people to excess risks across the life course.

Epidemiologic methods in health disparities

Increasing attention to the conceptual and methodological issues with investigating race as a cause has led to a more nuanced approach to the understanding and operationalization of race in etiologic research [53–55]. In epidemiology, race has often been treated as either a determinant of disparities or as a nuisance variable whose "effects" should be controlled away [53–57]. This practice is consistent with epidemiology's roots in biomedical traditions, which emphasize interventions at the individual level, and has led some in epidemiology to conflate risk factors with causes [58]. Treating race as a cause of disease is problematic because racial categories are socially constructed, fluid, context-dependent, erroneously operationalized discrete, and have shifted over time [2, 4, 53, 59, 60]. These intense preferences for conceptualizing race as discrete and unchanging also reflect a culture in the US of monoracism [9, 61].

Some have argued that instead of studying race as a cause, race-stratified and race interaction analyses are more theoretically valid etiologic approaches in investigating health disparities [53]. Statistical interactions are frequently used in regression analyses to determine if there are differences effect of primary exposures on an outcome vary across strata of a third independent variable. One common use of interactions is to model differences in effect of exposure across population subgroups, or "subgroup effects". Differences in effect are described using multiplicative or additive scale measures, which can be tested to determine if the joint associations of exposure and membership in the subgroup are significantly different than the products or sums of their individual associations. Results from such analyses can help answer questions such as: "do households receiving cash transfers to women see a greater reduction in household violence than when transfers are made to men?" Or, "does academic performance improve more for children of color than for White children when police are removed from schools?" When paired with contextual information and ethical analyses, these measures can help identify subgroups that should be prioritized for interventions, either because they experience comparatively greater relative or absolute gains in health (a utilitarian ethical approach) or because there are moral imperatives that warrant additional resources (one example of a deontologic approach). Conversely, such

analyses can also identify sub-populations for which interventions have less than expected impact or even harmful effects, and may not be appropriate targets for intervention.

Epidemiologists have struggled to agree on when statistical interactions do and do not represent biological interaction or causal interaction; however, because race is a social construct and not biological or causal, this dissertation only discusses statistical interactions [62, 63]. The validity of inference from statistical interactions is also compromised when measurement and classification of the modifying variable is imprecise, such as in the case of race. For these reasons, greater critical attention to racial classification when studying subgroup effects and racial disparities is needed, such as — at a minimum — not erasing Multiracial health experiences in research.

Epidemiologists are trained to understand that absolute (difference-based) measures of association are often of greater public health relevance than relative (ratio-based) measures because they incorporate information about population sizes, and accordingly, potential for overall population impact. While interaction analysis results can be presented on absolute scales, they still only compare subgroup effects in relation to each other, and thus have limitations in helping identify subgroup targets for interventions.

This dissertation

This dissertation consists of three empirical papers, all using the same dataset and all focused on describing and understanding the role of ACEs in contributing to racial health disparities between Multiracial and monoracial populations. The three papers are undergirded by the assertions that Multiracial experiences are valid and normal, and that inequities in health between Multiracial and monoracial people are the result of socially-constructed phenomena which can and should be rectified.

Chapter 2 investigates the elevated rates of exposure to ACEs among the Multiracial population, and tests a hypothesis that mean ACE scores are significantly greater among Multiracial people with AI/NA ancestry than those who do not. Evidence supporting this hypothesis would suggest that the high mean ACE score among the Multiracial population is driven by the large proportion of Multiracial people with AI/NA ancestry — another racial group with high exposure to ACEs. We explore differences in the prevalence of ACE score components across racial groups and conduct formal hypothesis tests to assess group differences in mean scores and risk of individual ACE components. This paper is one of the first we are aware of that offers any attempt to understand why Multiracial people have such high ACE scores compared to monoracial groups.

Chapter 3 incorporates health outcomes into our understanding of ACEs and Multiracial people. This paper explores differences by race in associations between ACEs and a variety of physical, mental, and behavioral health outcomes — outcomes for which Multiracial people have excess risk. We specify adjusted models regressing the outcomes of interest on an interaction term between ACEs and race, and investigate interactions on both the multiplicative and additive scale. Findings from this paper can help us understand which populations are

more vulnerable when exposed to ACEs, and thereby identify subgroups that may be best suited for preventive interventions.

Finally, Chapter 4 draws attention to the shortcomings of using effect modification or interaction analyses in identifying target subpopulations for interventions. Such approaches are common in public health and racial disparities research, but they can sometimes identify relatively strong effects in otherwise healthy populations. An uncritical use of interaction analyses could mislead researchers and practitioners into developing interventions that might in fact be better targeted to different populations. We highlight the benefits of simulated stochastic interventions as an alternative method to understanding "who benefits most?" in an application to a hypothetical intervention. Our simulation models the population-level risk difference in depression (or cases per 100 averted) associated with shifting each racial group's ACEs distribution to approximate that of the White population. Results from this analysis provide a practical consequentialist approach to health disparities research.

It is my hope that this dissertation contributes to a growing literature in the field of health psychology, epidemiology, and sociology that highlights and critiques outdated approaches to studying Multiracial people. We are a large and rapidly growing population, but research, scholarship, and clinical practice have moved slowly to embrace this demographic change. We reject radio buttons for racial categories in survey research, question clinicians who are supposedly trained in "cultural competencies" but continue to reify biological race in medicine, and ask those involved in the development of health policy how these policies address poor health in our community. We demand better, for inequities will remain unaddressed until the field recognizes that these health issues are not theoretical or abstract, but that they are real, unjust, and can be reversed.

Chapter 2

Adverse childhood experiences across Multiracial and monoracial groups with and without Indigenous ancestry

Abstract

Adverse childhood experiences (ACEs) are linked to increased risk of a host of health outcomes in adulthood. Descriptive ACEs prevalence studies have previously identified that Multiracial people have the highest mean ACE score of any racial group, but do not offer explanations for these disparities. However, Multiracial people form one of the fastest-growing populations in the US, and the largest subgroup of Multiracial people is those that claim American Indian/Native American (AI/NA) ancestry. Mean ACE counts (scores) are also high among the AI/NA population, which may reflect impacts of land occupation and structural racism. This descriptive study uses data from the National Longitudinal Study of Adolescent to Adult Health (Add Health) to test the hypotheses that (1) the mean ACE score of Multiracial participants is significantly higher than Whites, and (2) mean scores are higher among Multiracial participants with (M AI/NA) compared to without AI/NA (M not AI/NA) ancestry. Mean scores were highest among AI/NA (2.83, 95% CI: 2.28, 3.39), M AI/NA (2.73, 95% CI: 2.51, 2.94), and M not AI/NA (2.64, 95% CI: 2.39, 2.89) groups; none of these means were statistically different from one another (M AI/NA-AI/NA: $p = 0.920$, M not AI/NA-AI/NA: $p = 0.453$, M not AI/NA-M AI/NA: $p = 0.164$). Pairwise comparisons showed that both Multiracial groups had significantly higher scores than Whites (M AI/NA-White: $p < 0.001$, M not AI/NA-White: $p = 0.003$). The only ACE component where M not AI/NA participants had lower risk than M AI/NA participants was sexual abuse (risk ratio = 0.31; 95% CI: 0.10, 0.94; $p = 0.039$). Results from this study support hypothesis 1, but not 2, suggesting that the Multiracial population's high ACE scores are not driven primarily by those with AI/NA ancestry. Future studies should repeat this analysis in larger datasets, and explore other determinants of high mean ACE scores among the Multiracial

population.

2.1 Background

Adverse Childhood Experiences (ACEs)

Adverse childhood experiences (ACEs) are traumatic events occurring during childhood known to be associated with the development of physical, psychological, and behavioral health problems later in life [64, 65]. Strong dose-response effects of ACEs across multiple domains of health outcomes have been documented. One systematic review and meta-analysis found those reporting four or more ACEs were at higher risk for every studied health condition compared to those reporting no ACEs, with associations strongest for psychological and behavioral health outcomes and weaker for cancer and cardiometabolic outcomes [64]. Clinical guidance for ACEs risk assessments suggest that the risk of poor health tends to increase sharply at a score of three or four ACEs, depending on the outcome [66]. However, others have argued that ACE scores are more appropriately used to assess population-level differences in health risks, rather than prediction of a specific individual's future health [67].

The initial ACEs study conducted by Kaiser Permanente between 1995-97 found widespread prevalence of ACEs in a relatively high socioeconomic status (SES), commercially-insured population in San Diego, CA [68]. The original ten ACE domains include: emotional, physical, and sexual abuse, emotional and physical neglect, parental separation or divorce, mother treated violently, household substance abuse, mental illness in household, and incarceration of a parent. The study substantiated the need for wider screening for traumatic childhood events beyond low SES populations and communities of color. However, disparities in the prevalence of ACEs by race, SES, sexual orientation, and other characteristics persist, with populations in socially-privileged positions consistently reporting fewer ACEs than marginalized groups [44].

Discussions on the causes of racial disparities in ACEs are limited, which may reflect the reality that ACEs are most frequently used as an individual screening tool in medicine rather than as a measure of population health or health equity [69]. Additionally, ACEs are not a discrete condition, but a disparate set of traumatic events, which may also explain the paucity of literature on causes. The few studies that discuss racial disparities in ACEs suggest institutional and/or structural or systemic racism as potential causes [70–72]. Racism becomes institutionalized when social institutions — such as schools, employers, courts, etc. — adopt and enforce policies or practices that are either overtly racist or lead to racial disparities in outcomes. Some examples include: state-sponsored occupation of Indigenous land, racially discriminatory lending practices, overpolicing and mass incarceration of people of color, and racialized treatment standards in healthcare delivery. Structural or systemic racism reflects the cumulative effects of racism across interacting institutions, enabling preservation of White privilege across space and time, and is considered a fundamental cause of health inequities [73–76].

Some forms of structural and institutional racism cause ACEs directly; for example, over-policing and mass incarceration in Black and Brown communities results in large numbers of children of color with parents in prison [77]. Other forms may cause ACEs indirectly via material deprivation and the stress pathway across time. For example, the colonization and occupation of Indigenous lands and the subsequent forced resettlement of American Indian/Native American (AI/NA) peoples into reservations with substandard educational and employment opportunities has impacted generations of AI/NA families. The stress on AI/NA families resulting from these structurally racist events predisposes their children to adverse developmental environments and experiences [78, 79].

Multiracial people and ACEs

Multiple studies have found that Multiracial — those identifying with more than one racial group — and AI/NA people consistently report the highest number of ACEs of all race/ethnicities [43, 44, 71, 80, 81]. However, small population sizes and inconsistency of racial data collection instruments mean that such population health analyses infrequently focus on Multiracial and AI/NA groups. As described above, there is a clear connection between colonialist land occupation, structural racism, and ACEs for the AI/NA population. However, most literature on structural racism discusses its impact on monoracial populations of color, making it unclear if structural racism is also responsible for elevated ACEs among the Multiracial populations [6].

None of the studies identifying disparities in ACEs between monoracial and Multiracial populations discuss possible reasons for this difference. Given high rates of ACEs also reported in the AI/NA population and the disproportionate effects of structural racism and land occupation burdening this community, the Multiracial-monoracial disparity could be a result of the high numbers of Multiracial people claiming AI/NA ancestry. Disaggregation of Multiracial ancestry to investigate this theory is not possible in publicly-available versions of large, nationally representative public health datasets, such as the National Health Interview Survey (NHIS), the Behavioral Risk Factor Surveillance System (BRFSS), and the National Survey of Children's Health (NSCH), as the race variables provided collapse detailed multiple race responses into a single "Multiracial" category. Investigating disparities in ACEs in datasets that allow for detailed disaggregation of the Multiracial category could be a first step in better understanding the unique and urgent health risks facing this population.

In order to examine factors potentially associated with the disparity in exposure to ACEs between Multiracial and monoracial groups, this study aimed to test two hypotheses: 1) Multiracial participants will report significantly higher levels of ACEs than monoracial White participants, and 2) Multiracial participants with AI/NA ancestry will report significantly higher levels of ACEs than Multiracial participants without AI/NA ancestry. Support of the second hypothesis — here forward referred to as the "land occupation hypothesis" — would suggest that the Multiracial population's high mean ACE score is driven by the large proportion of those with AI/NA ancestry, possibly reflecting the multigenerational consequences of colonialist land occupation affecting AI/NA communities.

2.2 Methods

Data and analytic sample

The National Longitudinal Study of Adolescent to Adult Health (Add Health) is a longitudinal, nationally representative study following over 20,000 individuals from grades 7-12 in 1994-95 through four waves of follow-up (1996, 2001-02, 2008-09, 2016-18) [82]. Add Health is the largest nationally representative longitudinal study that allows study participants to select more than one racial category, making it an important source of research on Multiracial people [5, 83]. Participants were recruited through a school-based cluster sample, sampled with unequal probability and without stratification. From a database of all 26,666 high schools in the US, eighty public and private high schools and associated feeder middle schools were selected for size, type, grade range, setting, demographics, and geographic location. Within schools, specific racial/ethnic groups and students with disabilities were oversampled for population weighting. Students in grades 7-12 were sampled from school enrollment rosters and invited to complete an at-home interview during Wave 1, with a total sample of 20,745 participants. Wave 1 included detailed questions about the adolescent's demographics and family background, social networks, home and school environments, and health behaviors. Wave 4 was conducted 14 years after Wave 1 in 2008-09, when the participants were in their late 20's, and included measurements of the participants' metabolic and cardiovascular function. More details about the study design can be found in the Add Health study documentation [84].

Classification of race

Add Health participants were asked to self-identify their race and ethnicity in Waves 1 and 3. In Wave 1, participants were given five choices of racial categories: White, Black, American Indian/Native American (AI/NA), Asian, and Other, and were given the option to select multiple categories. In a separate question, participants were asked to indicate if they identified as Hispanic or Latino. At Wave 3, the "Other" option was removed, leaving respondents to select from four race options: White, Black, AI/NA, and Asian, still with the option to select multiple categories. Given the conceptual and methodological complexities of enumerating Multiracial people in quantitative data, this study aimed to maximize the number of participants who classified as Multiracial. Racial classification in this study thus aimed to balance 1) Multiracial identification, 2) recency of race data collection, and 3) detailed race information. If a participant identified as Multiracial in both waves, Wave 3 race was used. If a participant only identified as Multiracial in Wave 1 (but not in Wave 3), Wave 1 race was used. If a Multiracial participant selected "Other" in Wave 1, Wave 1 racial combination was used. Otherwise, Wave 3 race was used. Multiracial people were categorized as either having or not having AI/NA ancestry to test the land occupation hypothesis. The final racial categories were White, Black, Asian, AI/NA, Multiracial AI/NA, and Multiracial not AI/NA. Post-hoc analysis led to exclusion of the "Other alone" category due to the

much smaller sample size relative to other groups (n = 24) and large confidence intervals, preventing meaningful interpretation of results. However, the "Other" category was retained in classification of Multiracial status.

Despite official attempts to separate race and Hispanic ethnicity, a majority of Hispanic/Latino people consider Latinidad to be a core part of their racial identity [14]. When Hispanic ethnicity is assessed separately from race, it becomes impossible to differentiate between monoracial or Multiracial Hispanic/Latino people. For example, a European-descended Latino person who appears and self-identifies as monoracial White (such as many Argentinians or Uruguayans) could potentially be grouped together with a self-identified Multiracial person with one non-Latino White parent and one non-White Latino parent. To identify Multiracial people, some analyses of Add Health data have aligned with Census guidance and only used race data (ignoring Hispanic/Latino ethnicity), while others have acknowledged the difficulties in separating ethnically Hispanic/Latino participants into monoracial and Multiracial groups and simply excluded all Hispanic/Latino participants [34]. This study also excluded participants that identified as Hispanic/Latino in Wave 1 or Wave 3.

Measurement of ACEs

The original ACEs questionnaire included ten questions each covering a single domain of adverse experiences [85]: (1) emotional abuse, (2) physical abuse, (3) sexual abuse, (4) emotional neglect, (5) physical neglect, (6) parental separation or divorce, (7) mother treated violently, (8) household substance abuse, (9) mental illness in household, and (10) incarceration of household member. The original ACEs questionnaire was not administered in any Add Health surveys. Instead, researchers have used Add Health questions that approximate those asked in the original ACEs questionnaire [86–89]. This analysis used a modified version of the widest set of questions available in Add Health data to construct ten variables approximating the components of the original ACE score, following the methods of Lee et al. and Otero [89, 90]. Variables were coded as binary and summed, with a minimum of zero and maximum of ten. Further details on the questions and variables used to construct the ACEs score in Add Health are available in Appendix A.

Statistical analyses

Mean ACE scores (e.g.: the mean number of ACEs experienced) and unadjusted risk ratios of reporting each ACE component were compared across monoracial groups and Multiracial subgroups (those with and without AI/NA ancestry). Because the Add Health design specifically oversampled certain groups based on demographic characteristics, a design-based analysis was conducted in order to address selection bias built into the sampling plan. Complex survey weights were used in order to produce nationally-representative estimates and standard errors. Group differences in mean scores and risk of individual ACE components were assessed using Tukey's honest significance test and by comparing 95% confidence inter-

vals around mean score estimates [91, 92]. All data and statistical analyses were performed in R using the "tidyverse" and "multcomp" packages [93–96]. Analysis of design effects with complex survey weights was implemented using the "survey" package [97]. There were high levels of missingness in ACE components (frequencies presented in Table 2.1), and thus Markov chain multiple imputation with 30 iterations and 4 chains was performed using the "mi" and "mitools" packages [98, 99]. Imputation models specified all ACE components along with participant demographic information and hypothesized missingness predictor variables; results were pooled across 20 imputed datasets.

2.3 Results

Unweighted counts and weighted joint distributions of participant sex, age, component ACE scores, and summary ACE scores by racial group are presented in Table 2.1. Total sample size was 12,372 after removing Hispanic/Latino participants, "Other alone" participants, and those missing complex survey weights for design-based analysis. Of these, 9,538 (94.2%) were monoracial and 834 (5.82%) were Multiracial. Monoracial White (n = 7,742, 73.5%) and Black (n = 2,915, 16.9%) participants form the largest groups in the study sample. Among those identifying as Multiracial, 508 (69.9%) indicated that they had some AI/NA ancestry, while 326 (30.1%) did not. Figure 2.1 visualizes the intersection and overlap of racial categories endorsed by Multiracial Add Health participants using an UpSet plot [100, 101]. Among Multiracial participants, there are a total of eleven subgroups, with the most common being White and AI/NA (n = 347), White and Black (n = 123), White and Asian (n = 119), and Black and AI/NA (n = 96). Of Multiracial participants, a majority (n = 685) were Biracial and 149 selected three or more races.

Figure 2.2 visualizes the distribution of ACE scores by racial group using violin plots and jittered individual ACE scores, overlaid with group means and 95% confidence intervals. Among all racial groups, AI/NA participants and Multiracial participants (with and without AI/NA ancestry) reported the highest mean ACE scores. Formal pairwise tests of difference in mean ACE score between racial groups are presented in Table 2.2. Multiracial AI/NA participants reported significantly higher ACE scores than all monoracial groups except for the AI/NA group. There was no significant difference in mean score between the two Multiracial groups. The Multiracial groups were the only to have significantly higher mean ACE scores than both the White and Asian groups. Black and AI/NA participants had significantly higher mean scores than Whites, and AI/NA participants' mean scores were also higher than that of Asians.

Figure 2.3 visualizes proportions and 95% confidence intervals of the ACE component scores for each racial group. AI/NA participants were the most likely to report household substance abuse (43%), household mental illness (16%), and parental incarceration (30%), second most likely for emotional neglect (36%)), and least likely for sexual abuse (2.4%)) and physical neglect (35%). The Multiracial AI/NA group had the highest proportion of participants reporting sexual abuse (8.4%), emotional neglect (39%), and the second highest

proportion reporting emotional abuse (52%), household substance abuse (24%), and household mental illness (12%). Multiracial participants without AI/NA ancestry were the most likely to report emotional abuse (54%), parental separation or divorce (36%), and physical abuse (39%), second most likely for mother treated violently (6.1%), and least likely for household mental illness (6.2%). Asian participants were the second most likely to report physical abuse (38%) and physical neglect (44%), but least likely for parental separation or divorce (19%), household substance abuse (6.0%), and parental incarceration (6.1%). The Black group was the most likely to report mother treated violently (7.2%), and second most likely for sexual abuse (7.7%), physical neglect (44%), and parental incarceration (27%)). White participants were the least likely to report physical abuse (27%) and emotional neglect (28%).

Table 2.3 summarizes the statistically significant pairwise tests of ACE component score risk ratios. For each of the ACE components, significant risk ratios of pairwise racial group comparisons were detected. Differences across racial groups were most pronounced for household substance abuse and parental incarceration. Multiracial AI/NA participants had significantly greater risk than at least one other group of ever component except physical neglect and mother treated violently, as did Black participants other than for emotional and physical abuse. Non-AI/NA Multiracial participants had significantly greater risk of emotional abuse, physical abuse, parental separation or divorce, mother treated violently, household substance abuse, and parental incarceration than at least one other group. Asian and White participants had generally lower risk of reporting ACE components. Exceptions to this trend included physical abuse, emotional neglect, and physical neglect, where Asian participants had significantly greater risk than White participants. In the components where White participants had significantly greater risk, it was always in comparison to Asian participants. Notably, the two Multiracial groups were significantly different in only sexual abuse, where those without AI/NA ancestry had lower risk (RR = 0.31; 95% CI: 0.10, 0.94; p = 0.039), and mother treated violently, where those without AI/NA ancestry had higher risk (RR = 4.93; 95% CI: 1.35, 18.0; p = 0.016).

Table 2.1: Counts, imputed proportions[1], and missing proportions of ACE components stratified by racial group[2], Add Health Study Wave 4 (2008-09)[3]

Characteristic	White 7,742 (73.5%)	Black 2,915 (16.9%)	Asian 805 (3.18%)	AI/NA 76 (0.55%)	M AI/NA[4] 508 (4.07%)	M not AI/NA[4] 326 (1.75%)	Overall 12,372 (100%)
Male sex	3,672 (51%)	1,267 (50%)	418 (53%)	41 (64%)	230 (50%)	150 (50%)	5,778 (51%)
Age	28.90 (1.82)	29.20 (1.88)	29.15 (1.71)	28.83 (1.70)	28.75 (1.80)	28.78 (1.81)	28.95 (1.83)
Emotional abuse	3,571 (47%)	1,329 (46%)	406 (51%)	33 (46%)	268 (52%)	172 (54%)	5,779 (47%)
Missing	92 (1.4%)	63 (2.6%)	8 (1.3%)	3 (9.0%)	10 (1.5%)	5 (1.2%)	181 (1.7%)
Physical abuse	1,674 (27%)	603 (29%)	264 (38%)	22 (37%)	164 (36%)	98 (39%)	2,825 (28%)
Missing	1,502 (20%)	703 (27%)	141 (16%)	13 (21%)	68 (16%)	68 (23%)	2,495 (21%)
Sexual abuse	246 (5.0%)	133 (7.7%)	37 (6.4%)	4 (2.4%)	34 (8.4%)	7 (4.0%)	461 (5.6%)
Missing	1,453 (19%)	665 (26%)	125 (13%)	11 (19%)	57 (13%)	64 (21%)	2,375 (20%)
Emotional neglect	2,209 (28%)	903 (32%)	281 (35%)	27 (36%)	186 (39%)	109 (33%)	3,715 (30%)
Missing	37 (0.6%)	17 (0.8%)	4 (1.6%)	2 (1.0%)	4 (0.5%)	0 (0%)	64 (0.7%)
Physical neglect	2,453 (40%)	954 (44%)	330 (49%)	29 (35%)	212 (43%)	119 (42%)	4,097 (41%)
Missing	1,417 (19%)	646 (25%)	120 (13%)	12 (20%)	54 (12%)	59 (20%)	2,308 (19%)
Parental div. or sep.	1,922 (29%)	774 (33%)	64 (19%)	17 (33%)	149 (32%)	93 (36%)	3,019 (30%)
Missing	971 (12%)	605 (23%)	269 (30%)	19 (22%)	72 (12%)	67 (21%)	2,003 (15%)
Mother treated viol.	148 (3.7%)	61 (7.2%)	6 (4.3%)	0 (3.6%)	6 (2.5%)	10 (6.1%)	231 (4.3%)
Missing	2,102 (27%)	1,602 (58%)	355 (42%)	42 (68%)	169 (29%)	153 (50%)	4,423 (33%)
Hh. substance abuse	1,302 (17%)	408 (15%)	49 (6.0%)	23 (43%)	118 (24%)	49 (19%)	1,949 (17%)
Missing	4 (<0.1%)	6 (0.2%)	1 (< 0.1%)	0 (0%)	0 (0%)	0 (0%)	11 (0.1%)
Hh. mental illness	706 (9.3%)	313 (12%)	70 (6.9%)	14 (16%)	54 (12%)	30 (6.2%)	1,187 (9.8%)
Missing	8 (0.2%)	6 (0.2%)	3 (0.2%)	0 (0%)	0 (0%)	0 (0%)	17 (0.2%)
Parental incarceration	1,212 (15%)	726 (27%)	48 (6.1%)	24 (30%)	124 (23%)	72 (24%)	2,206 (17%)
Missing	4 (<0.1%)	4 (0.3%)	0 (0%)	0 (0%)	0 (0%)	1 (0.7%)	9 (0.1%)
ACE Score							
Complete case	1.99 (1.63)	2.17 (1.61)	1.96 (1.37)	2.51 (1.53)	2.53 (1.79)	2.32 (1.59)	2.05 (1.63)
Imputed	2.22 (1.82)	2.53 (1.77)	2.22 (1.58)	2.83 (1.76)	2.73 (1.83)	2.64 (1.78)	2.30 (1.85)

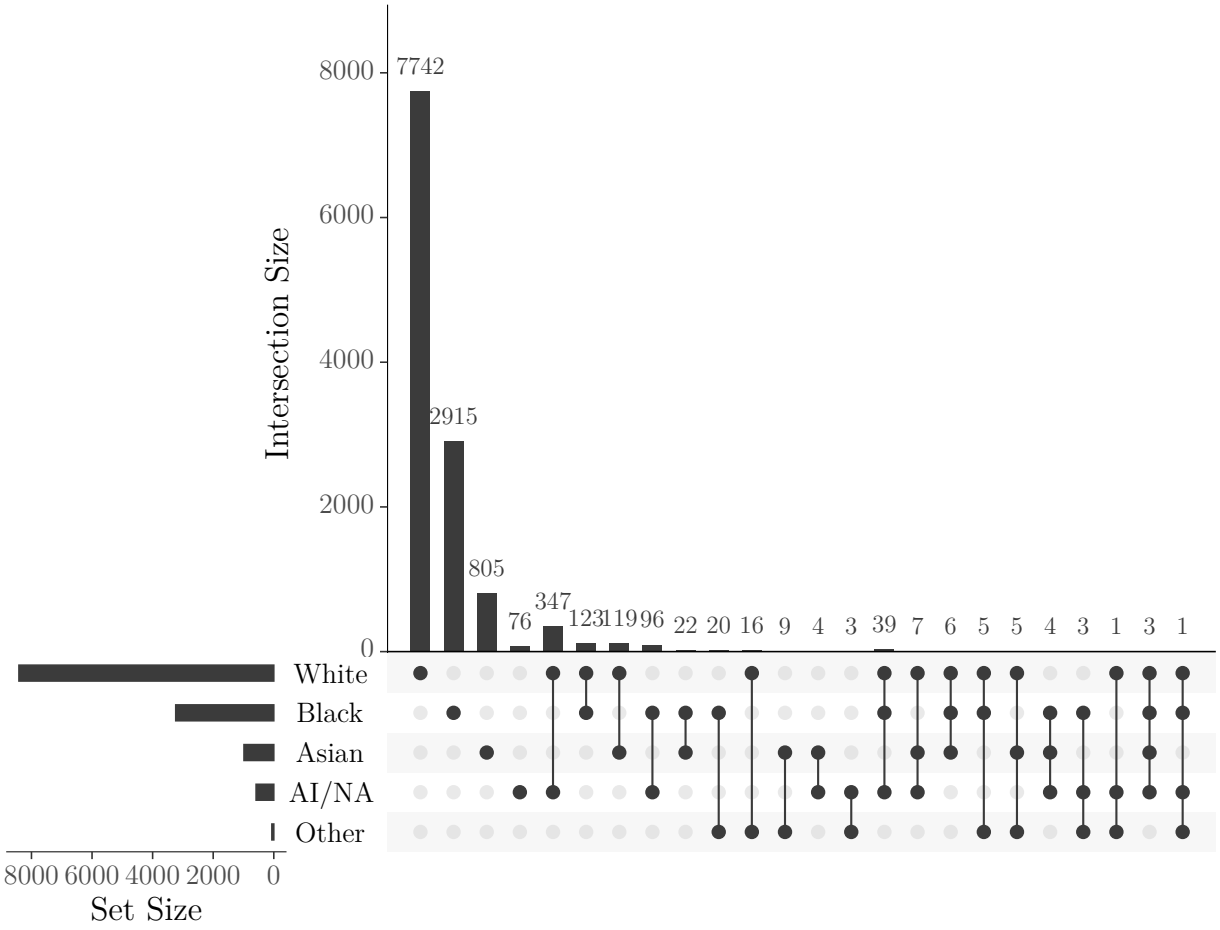
[1] Unweighted n (weighted, imputed proportions) reported for categorical variables; mean (SD) for continuous, unweighted count and proportion for missing

[2] Multiracial participants grouped according to presence or absence of AI/NA ancestry

[3] ACE components measured variously across Waves 1, 3, and 4, see Appendix A for details

[4] M = Multiracial

Figure 2.1: UpSet plot showing unweighted race intersection sizes of the sample of participants, Add Health Study Wave 3 (2001-02)



Column chart represents unweighted counts of unique racial combinations denoted by the connected dots below. Rows represent distinct racial categories, and the bar chart represents the unweighted count of all participants who selected that race alone or in combination with others.

Figure 2.2: Violin plots showing density of jittered individual ACE scores by racial group, overlaid with imputed group means and 95% confidence intervals

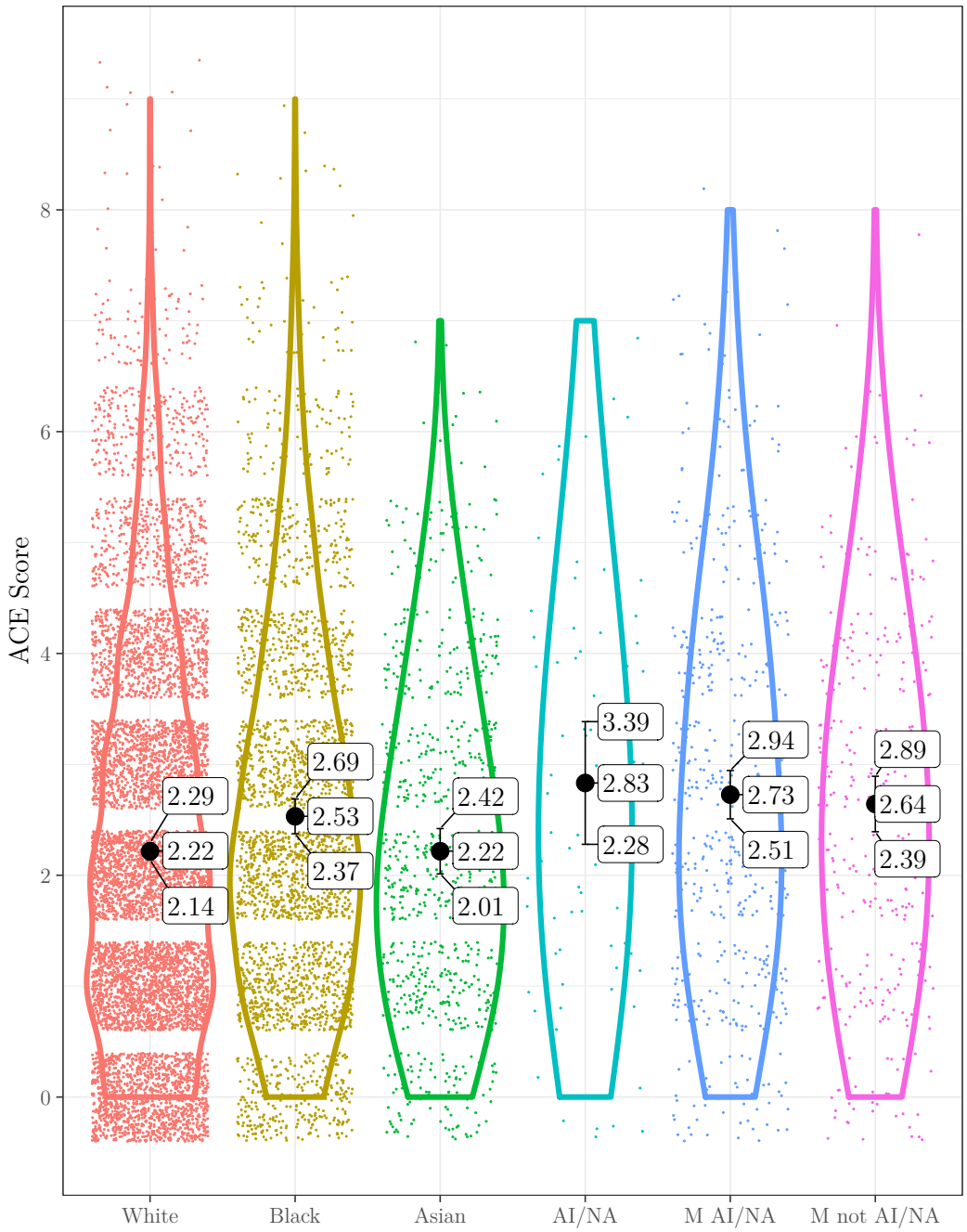


Table 2.2: Simultaneous pairwise comparisons[1] testing hypotheses of difference in mean ACE score between comparison and referent racial groups

Reference	**Comparison**	**Mean diff.**	**95% CI**	**p-value**	
White	Black	0.089	(0.016, 0.162)	0.017	*
	Asian	-0.013	(-0.112, 0.085)	0.793	
	AI/NA	0.230	(0.042, 0.419)	0.017	*
	M AI/NA	0.241	(0.152, 0.330)	< 0.001	***
	M not AI/NA	0.153	(0.052, 0.254)	0.003	**
Black	Asian	-0.102	(-0.212, 0.008)	0.069	
	AI/NA	0.142	(-0.059, 0.342)	0.166	
	M AI/NA	0.152	(0.044, 0.260)	0.006	**
	M not AI/NA	0.064	(-0.051, 0.179)	0.274	
Asian	AI/NA	0.243	(0.020, 0.467)	0.033	*
	M AI/NA	0.254	(0.128, 0.380)	< 0.001	***
	M not AI/NA	0.166	(0.044, 0.288)	0.007	**
AI/NA	M AI/NA	0.011	(-0.200, 0.221)	0.920	
	M not AI/NA	-0.077	(-0.280, 0.125)	0.453	
M AI/NA	M not AI/NA	-0.088	(-0.212, 0.036)	0.164	

[1] Multiple Comparisons of Means: Tukey Contrasts. Results from unadjusted Poisson regression model. Adjusted p values reported, single-step method.

Figure 2.3: ACE component prevalence and 95% confidence intervals by racial group, separating Multiracial participants into those with and without AI/NA ancestry

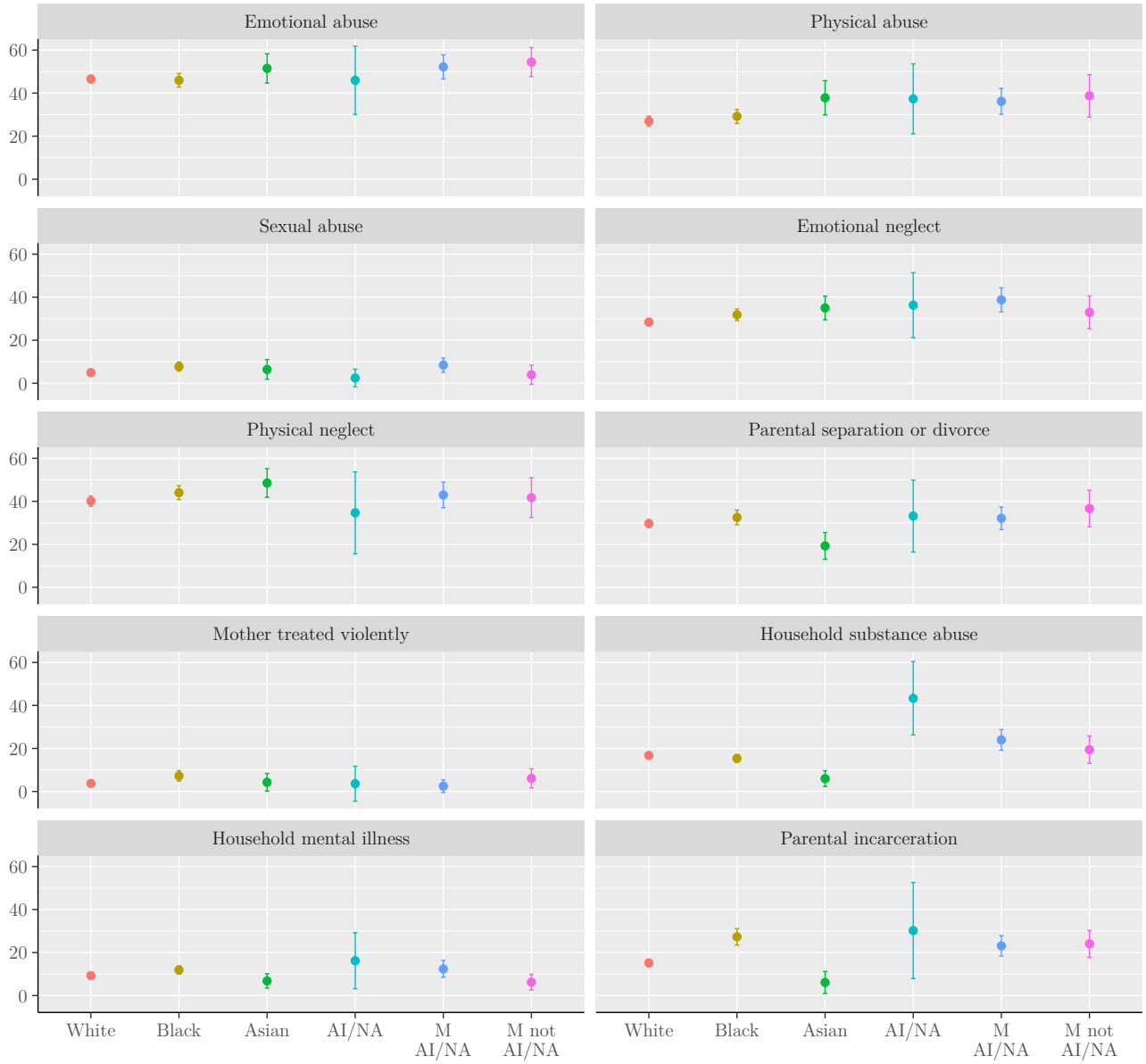


Table 2.3: Summary of significant[1] pairwise risk ratios (RR) of endorsing ACE components, comparing[2] contrast and referent racial groups

Component	**Ref.**	**Contrast**	**RR (95%CI)**	**p-val.**	
Emotional abuse	White	M AI/NA	1.12 (1.00, 1.26)	0.047	*
		M not AI/NA	1.16 (1.02, 1.33)	0.028	*
	Black	M AI/NA	1.14 (1.00, 1.30)	0.042	*
		M not AI/NA	1.18 (1.03, 1.36)	0.020	*
Physical abuse	White	Asian	1.45 (1.18, 1.77)	< 0.001	***
		M AI/NA	1.42 (1.19, 1.69)	< 0.001	***
		M not AI/NA	1.43 (1.10, 1.84)	0.007	**
	Black	Asian	1.33 (1.06, 1.66)	0.014	*
		M AI/NA	1.30 (1.07, 1.60)	0.010	**
Sexual abuse	White	Black	1.64 (1.27, 2.13)	< 0.001	***
		M AI/NA	1.98 (1.30, 3.01)	0.002	**
	M AI/NA	M not AI/NA	0.31 (0.10, 0.94)	0.039	*
Emotional neglect	White	Black	1.12 (1.01, 1.24)	0.029	*
		Asian	1.24 (1.04, 1.48)	0.016	*
		M AI/NA	1.37 (1.18, 1.59)	< 0.001	***
	Black	M AI/NA	1.22 (1.03, 1.45)	0.022	*
Physical neglect	White	Black	1.11 (1.01, 1.23)	0.003	*
		Asian	1.31 (1.12, 1.54)	< 0.001	***
Parental div. or sep.	White	Black	1.13 (1.02, 1.26)	0.025	*
		Asian	0.50 (0.30, 0.83)	0.007	**
	Black	Asian	0.44 (0.27, 0.73)	< 0.001	***
	Asian	AI/NA	2.44 (1.07, 5.57)	0.035	*
		M AI/NA	2.05 (1.22, 3.43)	0.006	**
		M not AI/NA	2.39 (1.43, 3.98)	< 0.001	***
Mother treated violently[3]	White	Black	2.33 (1.47, 3.69)	< 0.001	***
		Asian	0.19 (0.04, 0.83)	0.028	*
	Black	Asian	0.08 (0.02, 0.38)	< 0.001	***
		M AI/NA	0.18 (0.06, 0.55)	0.003	**
	M AI/NA	M not AI/NA	4.93 (1.35, 18.0)	0.016	*
Hh. substance abuse	White	Asian	0.36 (0.19, 0.67)	< 0.001	***
		AI/NA	2.59 (1.75, 3.85)	< 0.001	***
		M AI/NA	1.43 (1.16, 1.77)	< 0.001	***
	Black	Asian	0.39 (0.21, 0.74)	0.004	**
		AI/NA	2.82 (1.89, 4.22)	< 0.001	***
		M AI/NA	1.56 (1.26, 1.94)	< 0.001	***
	Asian	AI/NA	7.22 (3.57, 14.6)	< 0.001	***
		M AI/NA	3.99 (2.10, 7.58)	< 0.001	***

Table 2.3: Summary of significant[1] pairwise risk ratios (RR) of endorsing ACE components, comparing[2] contrast and referent racial groups

Component	Ref.	Contrast	RR (95%CI)	p-val.	
		M not AI/NA	3.20 (1.55, 6.59)	0.002	**
	AI/NA	M AI/NA	0.55 (0.35, 0.86)	0.009	**
	AI/NA	M not AI/NA	0.44 (0.25, 0.78)	0.005	**
Hh. mental	White	Black	1.28 (1.06, 1.53)	0.009	**
illness	Black	Asian	0.58 (0.35, 0.95)	0.031	*
		M not AI/NA	0.53 (0.30, 0.92)	0.025	*
		M AI/NA	3.99 (1.62, 9.85)	< 0.001	***
	Asian	M AI/NA	1.81 (1.02, 3.21)	0.043	*
		M not AI/NA	0.50 (0.26, 0.97)	0.040	*
Parental	White	Black	1.81 (1.54, 2.12)	< 0.001	***
incarceration		Asian	0.41 (0.17, 0.95)	0.037	*
		M AI/NA	1.53 (1.23, 1.90)	< 0.001	***
		M not AI/NA	1.58 (1.21, 2.07)	< 0.001	***
	Black	Asian	0.22 (0.10, 0.52)	< 0.001	***
	Asian	AI/NA	4.92 (1.62, 15.0)	0.005	**
		M AI/NA	3.77 (1.59, 8.96)	0.003	**
		M not AI/NA	3.91 (1.63, 9.34)	0.002	**

[1] Only significant pairwise comparisons are presented; all other comparisons were insignificant at the $\alpha = 0.05$ level

[2] Multiple comparisons of means (Tukey contrasts); adjusted p values reported, single-step method

[3] Risk ratio contrasts comparing groups with zero counts are suppressed

2.4 Discussion

This study aimed to test two hypotheses: first, that the mean ACE score among Multiracial participants is significantly higher than that of monoracial White participants, and second, the land occupation hypothesis: that the mean ACE score among Multiracial participants with AI/NA ancestry is significantly higher than those without AI/NA ancestry. The first hypothesis was supported by this analysis of Add Health data, suggesting that Multiracial children face an elevated risk of ACEs compared to their White peers. The land occupation hypothesis was definitively not supported by the summary ACE score data, and among ACE components was only supported in sexual abuse.

The findings supporting hypothesis 1 are consistent with existing literature on racial disparities on ACEs, where others have also found Multiracial participants to have the highest ACEs scores using national data from BRFSS and state-level data from Maryland [44, 81]. This study adds additional evidence using a nationally-representative sample. The lack of support for the land occupation hypothesis could suggest that the elevated mean ACE score

among Multiracial people is due to another cause. In addition to traditional forms of racism, Multiracial people may also experience monoracism, a system of racial subordination that privileges single-race identification [9, 102]. Monoracism can be perpetuated by strangers, but evidence suggests that monoracism is also a feature of many interracial and Multiracial family structures [103–105]. It is plausible that certain forms of ACEs could be a result of monoracism, particularly forms of abuse or neglect. Another potential explanation is implicit and explicit racism that interracial couples cite as a source of relationship stress, which some have hypothesized to lead to elevated rates of intimate partner violence, divorce, and household dysfunction among interracial families [52, 106–109]. Monoracism has received limited attention in public health literature, but could help explain disparities in health between monoracial and Multiracial people.

This study was subject to a number of limitations. First, ACEs are defined as events occurring before the age of 18, but several ACEs domains can only be constructed using Add Health data with questions only asked during Wave 1 when participants were ages 12-19. Therefore, observations for those ACE domains were censored for any participants that turned 18 after Wave 1. This is particularly problematic for the parental separation or divorce ACE, given how common divorce is in the US population. ACE scores may thus be biased downwards, especially for younger participants that had less under-18 observation time in Wave 1. Second, small sample sizes, especially for the AI/NA population, resulted in wide confidence intervals, making interpretation of study results challenging. Repeating this analysis in a larger dataset could reduce some of the random error due to Add Health's relatively small study size. Third, if there is indeed an association between mean ACE score and race, the removal of the "Other" race option during Wave 3 of Add Health may have unintentionally introduced differential misclassification that could have affected results in unpredictable ways, particularly for Multiracial people who had previously endorsed the "Other" racial group. Finally, given that ACE scores of 4 or greater are associated with increased risk across the greatest number of outcomes, an alternative approach to this study could be to dichotomize the ACE score into "low" (0-3) and "high" (4+) categories.

This study is the first to demonstrate that Multiracial people with and without AI/NA ancestry do not have significantly different ACE scores, suggesting the high mean ACE score among Multiracial people warrants deeper exploration. Future studies should replicate this analysis and test the land occupation hypothesis in larger datasets where participants are directly asked the ACEs questionnaire, where individual exposure to aggregate measures of structural racism are more readily available, and where race is assessed more stably over time such as BRFSS and NCHS. Detailed race variables should be more frequently made available in public-use versions of datasets, which could help provide insights to make significant progress in understanding Multiracial-monoracial health disparities.

Chapter 3

Are the health consequences of childhood adversity greater for Multiracial individuals? Investigating ACE-race interactions in a longitudinal cohort

Key Points

- **Question**: Do associations between childhood adversity (ACEs) and physical, mental, and behavioral outcomes vary significantly by race?
- **Findings**: Across all races, risk ratios between ACEs and outcomes were greatest for mental and behavioral outcomes; relative scale interaction was only present for problematic drug use comparing Black to White participants, though Black participants are least likely to report problematic drug use. Additive scale interactions compared to White participants were significantly positive among Multiracial participants for anxiety and among American Indian/Native American participants for problematic drug use. Other significant additive scale interactions were negative, representing weaker associations than White participants.
- **Meaning**: Associations were strongest among mental and behavioral health outcomes, but no single racial group consistently experienced stronger interactions.

Abstract

Importance: Multiracial people have the highest mean adverse childhood experiences (ACEs) score and elevated risk in physical, mental, and behavioral outcomes.

Objective: To determine whether associations between ACEs score and physical, mental, and behavioral health outcomes are modified by race.

Design, Setting, and Participants: The National Longitudinal Study of Adolescent to Adult Health (Add Health) is a prospective, nationally-representative, longitudinal cohort of 20,745 adolescents selected through a multistage, school-based design in 1994-95 (Wave 1). This study analyzed data from Wave 1 demographic data and follow-up outcomes and biomarker data from Waves 3 (2001-02) and 4 (2008-09).

Exposure: ACEs

Main Outcome(s) and Measure(s): Metabolic syndrome (MetS), hypertension, asthma, anxiety, depression, suicidal ideation, and problematic drug use

Results: Of 12,372 eligible participants, 73.5% identified as White, 16.9% Black, 3.18% Asian, 0.55% American Indian/Native American (AI/NA), and 5.82% Multiracial. Mean Wave 4 age was 29. Mean ACE scores were highest among AI/NA (2.83, SD = 1.76) and Multiracial (2.70, SD = 1.82) participants, and lowest among Whites (2.22, SD = 1.82) and Asians (2.22, SD = 1.58). All global tests for relative scale statistical interactions were insignificant except for problematic drug use (p = 0.01), where risk ratios for Black participants (1.23, 95% CI: 1.15, 1.31) were significantly greater than for Whites (1.11, 95% CI: 1.09, 1.13). Interaction on the additive scale was estimated as the relative risk due to interaction (RERI) compared to White participants. Additive scale interactions were all negative (less strong than for Whites) except for anxiety among Multiracial participants (RERI = 0.052, 95% CI: 0.007, 0.221) and for problematic drug use among AI/NA participants (0.068, 95% CI: 0.068, 0.102). Associations were strongest for mental and behavioral health outcomes, while Asian and AI/NA participants had interactions with greatest (but negative) magnitude.

Conclusions and Relevance: Strength and directions of ACEs-race interactions varied across groups, outcomes, and modification scale; no single racial group consistently experienced stronger associations. Interaction analyses should be paired with exposure and disease prevalence data to appropriately identify intervention target populations.

3.1 Background

Adverse childhood experiences (ACEs), also referred to as childhood or early life adversity, are a set of traumatic events happening during childhood and adolescence that create harmful or toxic stress, and are linked to poor health in adulthood, including several of the leading causes of death in the United States [110, 111]. ACEs are associated with increased risk of numerous adverse health conditions, particularly respiratory, behavioral, and mental health outcomes [65]. While children exposed to ACEs may show immediate signs of distress, the health impacts of ACEs may manifest over time, as exposure to toxic stressors early in life predisposes individuals to stressful situations later in life through the stress proliferation chain [112–115].

In addition to ACEs, social processes such as economic deprivation and racial discrimination are associated with toxic stress, and linked to elevated rates of disease in those groups [73, 116–120]. Theories about the biological embedding of toxic stress and links to health outcomes can be grouped into at least three distinct pathways: triggering of the biological stress response and resulting impacts on the neuroimmune-endocrine axis [46, 47] epigenetic expression [48–50], and changes in health behaviors due to adaptation or coping with severe or long-term stress [51, 121–123].

Over 60% of the US population reports experiencing at least one ACE; up to a third reports experiencing household emotional abuse, parental divorce, or substance abuse [43, 68]. Racial disparities in ACE scores are well-documented [43, 44, 124]; studies suggest disparities may be a consequence of systemic racism, which structures unequal access to life opportunities including healthy childhood development [45]. Multiracial and American Indian/Native American (AI/NA) populations report the highest mean ACE score of any racial group [43, 44, 79, 125, 126]. However, investigators frequently reclassify Multiracial participants into a catch-all "other" racial category, existing monoracial categories, or exclude them completely, effectively masking disparities affecting this group [5, 7].

In addition to higher ACE scores, Multiracial adolescents experience significantly greater rates of behavioral health issues, while Multiracial adults have elevated risk of cardiometabolic, respiratory, and mental health conditions compared to their monoracial peers [11, 32–40]. Nationally, Multiracial people experience the highest prevalence of heart diseases, asthma, obesity, hopeless feelings, and serious psychological distress of any racial group, and are significantly more likely to be living in poverty, uninsured, and in poor physical health than monoracial White people [32, 33]. Additionally, Multiracial people may experience stress from both racism and *monoracism*, a system of discrimination that privileges single-race identities and devalues Multiraciality, resulting erasure at institutional and individual levels [9, 61, 127]. Taken together, Multiracial people have the highest ACEs of any racial and/or ethnic group, experience multiple forms of racism-related stress, and heightened health risk across a number of outcomes, especially some of those traditionally linked with ACEs.

Despite the strong links between ACEs and adult psychobehavioral health outcomes and the prevalence of similar outcomes among Multiracial people across the life course, to our knowledge no subgroup studies estimate associations for Multiracial populations. Further, studies investigating interactions between ACEs and monoracial groups have found mixed results. One analysis of BRFSS data found that ACEs are more strongly associated with prevalent asthma for Asian women than for Black and AI/NA women [71]. Another BRFSS analysis found that race moderated the relationship between ACEs and mental (but not physical) health, with populations of color experiencing a relatively stronger association than Whites [128]. A study from a smaller longitudinal cohort of White and Black adolescents in the US South found additive interaction in models for depression, with Black participants experiencing stronger associations [129]. These findings suggest that interactions with race may vary across health outcomes. Further, the lack of studies to date explicitly including a category for Multiracial people leaves out a fast-growing and potentially vulnerable population in research on ACEs.

This study leverages a nationally-representative longitudinal sample of young adults to determine if the association between ACEs and cardiometabolic, respiratory, behavioral, and mental health outcomes — conditions for which Multiracial people have poor profiles — varies by racial group, to determine whether Multiracial people should be prioritized for interventions.

3.2 Methods

Data and analytic sample

The National Longitudinal Study of Adolescent to Adult Health (Add Health) is a longitudinal, nationally representative study following over 20,000 individuals enrolled in grades 7-12 in 1994-95 through four waves (1996, 2001-02, 2008-09, 2016-18) of follow-up [84]. Participants were recruited from 80 middle and high schools selected non-randomly for size, type, grade range, setting, demographics, and geographic location. Within schools, specific racial/ethnic groups and students with disabilities were oversampled for population weighting. Students were sampled without stratification from enrollment rosters and invited to complete an at-home interview during Wave 1 (n=20,745) which asked questions about the adolescent's demographics, family background, social networks, home and school environments, health behaviors. Wave 4 was conducted in 2008-09, when participants were in their late twenties, and included measurements of the participants' metabolic and cardiovascular function.

Participants were categorized into one of five racial groups: White alone, Black alone, Asian alone, American Indian/Native American (AI/NA) alone, (hereafter White, Black, Asian, or AI/NA) or Multiracial. Race was measured at Waves 1 and 3, and Wave 3 race was used unless an individual identified as Multiracial at Wave 1 but not Wave 3, in which case they were classified as Multiracial. Participants self-identifying as Hispanic/Latino were excluded because it is impossible to identify self-identifying Multiracial individuals when race and Hispanic ethnicity are assessed independently [34, 130].

Outcomes

Because differential exposure to ACEs may be linked to racial disparities in health through the stress pathway, this study investigated physical, mental, and behavioral outcomes chosen to reflect those (1) for which Multiracial Americans experience relatively poor health outcomes, and (2) that are associated with ACEs and overactivation of the stress pathway [117, 131–134]. Outcomes were measured once for each individual in either Wave 3 (ages 18-26) or 4 (24-32). Details on construction of outcomes and covariates are available in Appendix B.

Physical health: metabolic syndrome (MetS), hypertension, and asthma

MetS was assessed using clinically-relevant cutoffs for eight biomarkers across five categories: (1) hypertension (systolic blood pressure $\geqslant$ 130 mmHg, diastolic blood pressure $\geqslant$ 80 mmHg) [135], ever diagnosed with hypertension, and current hypertension medication use), (2) waist circumference ($>$ 102 cm for males, $>$ 80 cm for females), (3) triglycerides (top 3 deciles for males, top 2 for females), (4) high-density lipoprotein (bottom 2 deciles for males, bottom 3 for females), and (5) glycated hemoglobin (HbA1c) $>$ 5.7% [136]. Individuals with biomarker levels exceeding the cutoffs in three or more of the five categories were categorized as having MetS.[136–140]. Hypertension coding mirrored that used for the MetS measure. Participants were classified as having asthma if they indicated a previous diagnosis [141].

Mental health: depression and anxiety

Participants were classified as having depression if they indicated using prescription depression medications in the last year or having a previous depression diagnosis, and as having anxiety if they indicated a previous anxiety diagnosis.

Behavioral health: suicidal ideation and problematic drug use

Participants were classified as having suicidal ideation if they indicated seriously thinking about committing suicide in the last year, and using drugs problematically if they indicated ever using cocaine, crystal meth, heroin other illegal drugs other than marijuana, or ever misusing prescription drugs.

Exposure: ACEs

The original ACEs questionnaire included ten questions each covering a domain of adverse experiences [85]. Domains included emotional, physical, and sexual abuse, emotional and physical neglect, parental separation or divorce, mother treated violently, substance abuse or mental illness in household, and incarceration of household member. Researchers constructing an ACEs score using Add Health data have used varying sets of questions about events occurring before age 18 in order to approximate the original ACEs questionnaire [86–90]. The present study used a modified version of the widest set of questions available, some of which were answered by participants' parents in Wave 1, and others by participants during Waves 3 or 4 retrospectively. These questions were dichotomized and summed to create a score from zero to ten. Questions and variables used are available in Appendix A.

Control variables

Covariates in statistical analyses were pre-specified using a directed acyclic graph (DAG) and decided upon a priori. Age, sex, parental education, household size-adjusted income,

parental support, and neighborhood disadvantage score were included as confounding variables, while experiences of racism, and interviewer racial misclassification were included as additional control variables.

Age and sex were drawn from Wave 4 to match collected biomeasures. Parental education was calculated as the higher of either parent's Wave 1 parental education after categorization into a five-level ordinal variable (less than high school, completed vocational school, or GED; high school diploma; some college; college graduate or greater) [142]. Household size-adjusted income was calculated as total reported pre-tax household income divided by the square of the number of reported household members at Wave 1 [143].

Parental support was assessed at Wave 1 and coded as the mean of Likert-scale responses (very much, quite a bit, some, a little, none at all) to five questions about participants' relationship and communication with their parents.

Neighborhood disadvantage score was calculated as the mean of five proportions from the census tract of the participant's Wave 1 residence: (1) percent households with incomes below the federal poverty line, (2) percent households receiving public assistance, (3) civilian unemployment rate, (4) percent persons 25 years or older with no high school diploma or equivalency, and (5) percent female-headed households [139, 144].

In addition to these confounders, this study adjusted for experiences of racism and monoracism to control for additional sources of health-harming stress unrelated to ACEs. ACEs are most prevalent among populations of color, and for these groups experiences of racism are often an additional stressor. This study adjusted for experiences of racial discrimination using a modified version of the first and second items from the Everyday Discrimination Scale [116, 145, 146] in Wave 4. Responses endorsing "race," "ancestry or national origin," or "skin color" as reasons for disrespectful treatment were coded as ever experiencing racial discrimination; all others were coded as never experiencing racial discrimination.

A common way monoracism can result in stress among Multiracial people is through deliberate racial misclassification [9, 61, 127, 147]. In Wave 1, Add Health interviewers were instructed to categorize participants into monoracial categories based on appearance alone, even if this visual assessment did not match participants' self-identified race; others have also used this variable as a rough proxy for monoracism-related stress [148]. Participants were coded as racially misclassified if their self-identified race did not match the category that the Add Health interviewer chose. Multiracial participants were classified as not racially misclassified if interviewers chose "other" or did not provide a response.

Statistical analyses

Coding checks and univariate analyses were conducted by visual assessment of plotted variable distributions. Assumptions of linearity between exposure and log-transformed risk of outcomes were assessed visually for each racial group by comparing observed data with linear model-generated smooth plots. Three sets of models regressed each outcome on ACE scores: a crude bivariate analysis, a fully-adjusted analysis, and an interaction analysis that interacted racial group with ACEs to estimate subgroup associations. Models were

specified cross-sectionally, however Add Health's longitudinal design and the retrospective questions used to assess ACEs scores allows for temporal ordering of exposure preceding outcomes. For all outcomes, modified Poisson models were specified to model risk ratios. Because White participants form the largest racial group in Add Health and have relatively low ACEs scores, all regression and interaction analyses specified Whites as the reference group. Complex survey weights corresponding to a cross-sectional multi-wave analysis were incorporated to produce nationally representative estimates using the "survey" package in R [97, 149]. Because up to 30% of observations were missing data on an ACE component, 21% on a covariate, and 15% on an outcome (frequencies presented in Tables 3.1 and 3.2), Markov chain multiple imputation was performed with 30 iterations and 4 chains, including all outcomes in imputations models, with results pooled across 20 imputed datasets. Imputation was handled using the "mi" and "mitools" packages in R [98, 99]. Interactions were assessed on the relative scale by estimating sub-group specific risk ratios (RRs), and on the absolute scale by estimating the relative excess risk due to interaction (RERI). Significance of subgroup RRs were assessed by comparing 95% CIs with null values. Statistical differences between group RRs were assessed using p-values with pre-specified $\alpha = 0.05$ from global Wald tests for significance of interaction terms. Differences in RERI were assessed by comparing 95% CIs across groups.

3.3 Results

Post-hoc analysis resulted in exclusion of individuals who identified as "Other" race due to small sample size (n=23) and overly wide CIs. Table 3.1 shows unweighted counts and weighted proportions key characteristics and ACEs scores of the overall study sample. Sample size in models with self-reported outcome data was 12,372. Biomarkers can be skewed during pregnancy, thus 445 pregnant participants were excluded from the MetS model. In the overall study sample, there were 7,742 (73.5%) White, 2,915 (16.9%) Black, 805 (3.18%) Asian, 76 (0.55%) AI/NA, and 834 (5.82%) Multiracial participants. Mean age, sex ratios, and parental support scores were approximately even across racial groups. White and Asian participants' households had parents with higher educational attainment, higher equivalence-scaled income, and lower neighborhood disadvantage scores compared to Black and AI/NA participants. On these measures, Multiracial participants were less advantaged than White and Asian but more than Black and AI/NA participants. Experiences of racism were highest among Black participants, and racial misclassification was highest among Multiracial participants. Mean ACE score was highest among AI/NA (2.83, SD = 1.76) and Multiracial participants (2.70, SD = 1.82) and lowest among White alone (2.22, SD = 1.82) and Asian (2.22, SD = 1.58) participants.

Table 3.2 shows the weighted proportions and 95% confidence intervals of the investigated outcomes by racial group. AI/NA and Multiracial participants had the highest prevalence of most conditions. MetS (54%), hypertension (68%), and suicidal ideation (15%) were highest among AI/NA participants, whereas asthma (24%), depression (24%), and anxiety (17%)

were highest among Multiracial participants. Problematic drug use was highest among White participants (41%). Asian participants reported the lowest prevalence of all outcomes except for problematic drug use, for which Black participants reported the lowest prevalence (12%).

Table 3.3 summarizes subgroup-specific RRs and 95% confidence intervals for each of the outcomes studied, stratified by racial group. In general, RRs were greatest for mental and behavioral outcomes, however there were no outcomes where every racial group had a significant association with ACEs. ACEs scores were not significantly associated with MetS or hypertension for any of the racial groups. Associations for White and Black participants were significant for all other outcomes (asthma, depression, anxiety, and suicidal ideation), and for Multiracial participants other than asthma. Global Wald tests for interaction were only significant for problematic drug use (p = 0.01), where subgroup associations appear significantly greater for Black participants (RR = 1.23, 95% CI: 1.15, 1.31) compared to White participants (RR = 1.11, 95%CI: 1.09, 1.13).

Table 3.4 summarizes interactions on the additive scale (RERI), with Whites as the reference. In contrast to assessment of interaction on the relative scale, there were significant additive scale in at least one group for all outcomes except MetS and suicidal ideation. Among the seven outcomes, Asians had significantly negative associations for four, Blacks for three, AI/NA participants had negative associations with two and positive associations with one, and Multiracial participants had positive associations for one. The majority of significant interactions were negative, namely, these associations were weaker compared to Whites; the positive additive interactions were for anxiety among Multiracial and problematic drug use for AI/NA participants.

For physical health outcomes, associations were weaker with hypertension among AI/NA participants (RERI = -0.129, 95% CI: -0.296, -0.021) and with asthma among Asians (-0.138, 95% CI: -0.290, -0.057) compared to Whites. For mental health outcomes, associations were weaker with depression among Blacks (-0.067, 95% CI: -0.086, -0.056) and Asians (-0.086, 95% CI: -0.219, -0.076), and weaker with anxiety among Black (-0.062, 95% CI: -0.092, -0.033), Asian (-0.148, 95% CI: -0.231, -0.110), and AI/NA (-0.122, 95% CI: -0.771, -0.077) participants. Conversely, associations were stronger for anxiety among Multiracial participants (0.052, 95% CI: 0.007, 0.221). For behavioral outcomes, associations were weaker with problematic drug use for Blacks (-0.059, 95% CI: -0.071, -0.045) and Asians (-0.030, 95% CI: -0.100, -0.001) but stronger for AI/NA participants (0.068, 95% CI: 0.029, 0.102).

Table 3.1: Unweighted counts and weighted proportions[1] of ACE components stratified by racial group, Add Health Study Wave 4 (2008-09)[2]

Characteristic	White 7,742 (73.5%)	Black 2,915 (16.9%)	Asian 805 (3.18%)	AI/NA 76 (0.55%)	Multiracial 834 (5.82%)	Overall 12,372 (100%)
Male sex	3,672 (51%)	1,267 (50%)	418 (53%)	41 (64%)	380 (50%)	5,778 (51%)
Age	28.9 (1.82)	29.2 (1.88)	29.2 (1.71)	28.8 (1.70)	28.8 (1.80)	29.0 (1.83)
Highest parental education						
Less than high school[3]	685 (9.6%)	356 (17%)	69 (13%)	13 (29%)	64 (11%)	1,187 (11%)
High school diploma	2,520 (34%)	959 (42%)	164 (24%)	19 (34%)	221 (33%)	3,883 (35%)
Some college	1,405 (19%)	500 (16%)	106 (10%)	19 (24%)	191 (22%)	2,221 (18%)
College graduate or greater	3,039 (38%)	1,043 (25%)	445 (53%)	24 (14%)	346 (34%)	4,897 (36%)
Missing	93 (1.4%)	57 (2.5%)	21 (2.0%)	1 (<0.1%)	12 (1.5%)	184 (1.6%)
Household income[4]	3.46 (5.15)	2.03 (2.73)	3.89 (8.32)	1.56 (1.65)	2.78 (3.15)	3.21 (4.89)
Missing	1,481 (19%)	850 (30%)	338 (40%)	24 (27%)	186 (20%)	2,879 (21%)
Parental support index (0-5)	3.35 (0.58)	3.26 (0.65)	3.30 (0.54)	3.39 (0.53)	3.30 (0.60)	3.33 (0.60)
Missing	9 (0.2%)	9 (0.3%)	2 (0.3%)	1 (1.0%)	0 (0%)	21 (0.2%)
Experienced racism	57 (0.8%)	236 (7.9%)	30 (3.4%)	0 (0%)	31 (2.8%)	354 (2.2%)
Missing	17 (0.4%)	10 (0.5%)	2 (<0.1%)	0 (0%)	1 (0.1%)	30 (0.4%)
Racially misclassified	36 (0.4%)	32 (1.0%)	41 (7.8%)	3 (6.7%)	516 (61%)	628 (4.2%)
Missing	63 (0.8%)	12 (0.4%)	16 (3.0%)	4 (4.8%)	23 (3.0%)	124 (0.9%)
Neighborhood disadvantage (0-1)	0.12 (0.06)	0.23 (0.11)	0.13 (0.06)	0.23 (0.09)	0.15 (0.08)	0.14 (0.09)
Missing	80 (1.2%)	28 (1.1%)	4 (0.6%)	0 (0%)	9 (1.1%)	121 (1.1%)
ACE Score[5]	2.22 (1.82)	2.53 (1.77)	2.22 (1.58)	2.83 (1.76)	2.70 (1.82)	2.30 (1.85)

[1] Unweighted n (weighted proportions) reported for categorical variables; Mean (SD) for continuous
[2] ACE components measured variously across Waves 1, 3, and 4, see Appendix A for details
[3] Includes completed vocational school or GED
[4] Equivalence-scaled to adjust for household size
[5] Mean sums and SDs of survey-weighted, imputed ACE component scores reported

Table 3.2: Weighted proportion of health outcomes, stratified by racial group

Outcome	White 7,742 (73.5%)	Black 2,915 (16.9%)	Asian 805 (3.18%)	AI/NA 76 (0.55%)	Multiracial 834 (5.82%)	Overall 12,372 (100%)
Metabolic syndrome	1,799 (27%)	852 (38%)	221 (27%)	35 (54%)	224 (31%)	3,131 (29%)
Missing	1,009 (14%)	550 (19%)	110 (13%)	12 (17%)	110 (15%)	1,791 (15%)
Hypertension	3,775 (51%)	1,464 (55%)	390 (46%)	46 (68%)	407 (50%)	6,082 (51%)
Missing	192 (2.5%)	123 (4.2%)	21 (1.7%)	5 (12%)	18 (3.2%)	359 (2.9%)
Depression	1,652 (21%)	296 (10%)	53 (7.6%)	11 (14%)	183 (24%)	2,195 (19%)
Asthma	1,134 (15%)	453 (15%)	90 (10%)	14 (16%)	176 (24%)	1,867 (15%)
Missing	2 (<0.1%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	2 (<0.1%)
Anxiety	1,179 (15%)	182 (6.0%)	26 (2.9%)	4 (3.3%)	127 (17%)	1,518 (13%)
Missing	2 (<0.1%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	2 (<0.1%)
Suicidal ideation	527 (7.1%)	188 (6.9%)	48 (6.0%)	8 (15%)	74 (10%)	845 (7.2%)
Missing	29 (0.6%)	49 (3.2%)	0 (0%)	1 (5.7%)	4 (0.4%)	83 (1.0%)
Problematic drug use	3,049 (41%)	356 (12%)	197 (25%)	30 (41%)	333 (41%)	3,965 (35%)

Unweighted n (weighted proportions) reported

Table 3.3: Summary of subgroup risk ratios, 95% confidence intervals, and global tests for interaction term significance

Outcome	White	Black	Asian	AI/NA	Multiracial	p-value
Metabolic syndrome	1.01 (0.98, 1.03)	1.00 (0.96, 1.05)	0.96 (0.81, 1.13)	0.99 (0.82, 1.20)	1.00 (0.91, 1.09)	0.98
Hypertension	1.00 (0.98, 1.01)	1.01 (0.98, 1.04)	1.01 (0.91, 1.11)	0.91 (0.79, 1.04)	0.97 (0.92, 1.03)	0.55
Asthma	1.10 (1.05, 1.14)	1.09 (1.02, 1.17)	0.96 (0.78, 1.18)	1.38 (0.99, 1.92)	1.09 (0.99, 1.21)	0.47
Depression	1.16 (1.13, 1.20)	1.29 (1.20, 1.39)	1.26 (0.95, 1.68)	1.08 (0.77, 1.51)	1.20 (1.12, 1.28)	0.14
Anxiety	1.12 (1.08, 1.17)	1.18 (1.07, 1.30)	0.91 (0.70, 1.20)	1.00 (0.58, 1.71)	1.23 (1.11, 1.37)	0.28
Suicidal ideation	1.23 (1.15, 1.32)	1.22 (1.08, 1.38)	1.43 (1.04, 1.98)	1.26 (0.89, 1.80)	1.20 (1.05, 1.37)	0.90
Problematic drug use	1.11 (1.09, 1.13)	1.23 (1.15, 1.31)	1.14 (0.98, 1.33)	1.33 (1.08, 1.62)	1.16 (1.11, 1.22)	0.01

Table 3.4: Summary of RERI estimates and 95% confidence intervals from resampled parameters

Outcome	White	Black	Asian	AI/NA	Multiracial
Metabolic syndrome	(ref.)	0.000 (-0.057, 0.045)	-0.051 (-0.217, -0.018)	-0.019 (-0.380, 0.121)	-0.009 (-0.058, 0.062)
Hypertension	(ref.)	0.013 (-0.021, 0.041)	0.010 (-0.040, 0.070)	-0.129 (-0.296, -0.021)	-0.024 (-0.059, 0.010)
Asthma	(ref.)	0.009 (-0.056, 0.060)	-0.138 (-0.290, -0.057)	0.114 (-0.027, 0.132)	0.037 (-0.103, 0.114)
Depression	(ref.)	-0.067 (-0.086, -0.056)	-0.086 (-0.219, -0.076)	-0.095 (-0.689, 0.008)	0.015 (-0.051, 0.055)
Anxiety	(ref.)	-0.062 (-0.092, -0.033)	-0.148 (-0.231, -0.110)	-0.122 (-0.771, -0.077)	0.052 (0.007, 0.221)
Suicidal ideation	(ref.)	-0.017 (-0.208, 0.039)	0.001 (-0.258, 0.102)	0.194 (-0.102, 0.419)	0.042 (-0.060, 0.115)
Problematic drug use	(ref.)	-0.059 (-0.071, -0.045)	-0.030 (-0.100, -0.001)	0.068 (0.029, 0.102)	0.021 (-0.010, 0.054)

Null value is zero. 95% confidence intervals calculated from 10,000 resamples of RERI

3.4 Discussion

This study aimed to explore whether the association of ACEs on physical, mental, and behavioral health outcomes differs by race, and in particular, if Multiracial individuals should be prioritized for interventions given their unique health risk profile. This study did find evidence suggesting that Multiracial people face excess risk of anxiety compared to Whites on the additive scale given exposure to ACEs. AI/NA participants also face excess risk of problematic drug use on the additive scale, as do Blacks on the multiplicative scale, but all other significant interactions were negative, meaning that associations were weaker than for Whites. These results suggest that significant interactions between ACEs and race may exist across a variety of health domains, especially in mental and behavioral health outcomes, where links to ACEs are also generally the strongest. This study aligns with previous research which has found that the strength of association with ACEs depends on the outcome, racial group, and interaction measurement scale [71, 128, 129], but adds additional evidence about associations for Multiracial people, which has not previously been reported.

Researchers commonly assess subgroup differences in associations to identify which groups should be prioritized for interventions. While subgroup analyses can provide one answer to this question, identifying groups for which effects are strongest in the absence of other epidemiologic data may lead to flawed inference. For example, although this study found significant multiplicative interaction between ACEs and problematic drug use comparing Black to White participants, Blacks also have the lowest prevalence of problematic drug use of all races. In other words, interaction analyses can identify strong associations in otherwise healthy populations, which are not public health issues of note. Researchers should thus cautiously interpret the results of such interaction studies and pair them with contextual knowledge, such as the high levels of exposure to ACEs among Multiracial and AI/NA populations.

This study had several limitations. First, the racial categories used in this analysis and health research generally collapse large amounts of within-group variability in patterns of health and disadvantage; subgroup analyses thus produce only a rough proxy for markers of elevated risk and should be contextualized with other knowledge. Second, the ACEs measure used in this study is unique to Add Health and could result in different scores than if participants had been asked a different set of ACE questions. Future interaction studies should include Multiracial people and leverage datasets designed to collect ACEs data. Lastly, measures for asthma, anxiety, and depression relied on self-reported diagnoses, which could be biased lower for racial groups with less healthcare access [150]. Similarly, our measure of parental education relied on highest level of completed education between either parent rather than the primary caregiver, which some research suggests may be a better predictor of child health [151]. Future analyses should consider specifying alternative measures for these variables .

To our knowledge, this is the first study identifying excess additive risks associated with ACEs for Multiracial people, a finding that is obscured by the common practice of categorizing Multiracial people with monoracial people, preventing meaningful analysis. The elevated

risk profile of Multiracial people across ACEs scores, poor adolescent and adult mental and behavioral health, and this study's evidence suggest that greater attention is warranted to the processes that predispose Multiracial children to toxic stress exposure, pathways of stress proliferation, and adult morbidity and disease. The current paradigm of routine screening for ACEs within health care settings is often discussed as a preventive strategy, however screening for ACEs does not prevent their occurrence [152, 153]. Evidence is clear that the widespread prevalence and harmful effects of ACEs are urgent health issues; further research should focus on how much impact true, upstream prevention of the occurrence of ACEs could have on ensuring health equity for all children.

Chapter 4

Population-level metrics in racial disparities research: a Multiracial health equity simulation study

Abstract

Background: Multiracial people report the highest mean Adverse Childhood Experiences (ACEs) score of any racial group and have high risk of several physical and psychobehavioral outcomes. However, interaction studies, often used to identify priority subgroups for intervention, do not suggest that the association between ACEs and health outcomes are uniformly stronger for Multiracial people. Unlike simple interaction analyses, methods for stochastic intervention analyses respect differences in exposure distributions and produce population-level measures, which are important for evaluating interventions.

Methods: We analyzed data from Waves 1 (1995-97) through 4 (2008-09) of the National Longitudinal Study of Adolescent to Adult Health (Add Health), a nationally-representative longitudinal study of the life course and outcomes of 20,745 adolescents followed into adulthood. We simulated a stochastic intervention over 1,000 resampled datasets to quantify the race-specific risk differences in depression if all racial groups had the same exposure distribution of ACEs as Whites.

Results: Simulated risk differences were largest for the Multiracial population, (median = 1.470 cases per 100 averted, 95% UI: -2.330, -0.803). The model predicted smaller risk reductions per 100 for Black (-0.568, 95% UI: -0.896, -0.318) and AI/NA (-0.258, 95% UI: -3.450, 1.660) participants, and a risk increase for Asians (0.002, 95% UI: -0.361, 0.481). Uncertainty intervals around AI/NA and Asian group estimates included the null.

Conclusion: Multiracial participants stand to benefit the most from a health equity intervention on ACEs. Stochastic intervention simulations offer substantial benefits compared to traditional racial disparities research methods, and should be used more frequently in health disparities research and practice.

Key Messages

- Interaction analyses are commonly used in racial disparities analyses to identify which subgroups to target for interventions
- However, stochastic interventions offer several advantages over interaction analyses, including the ability to respect heterogeneity exposure distributions across subpopulations.
- This study simulates a stochastic intervention, showing that the Multiracial population would see the greatest reduction in cases of depression associated with a health equity-focused intervention to prevent ACEs.
- Stochastic intervention methods should be more frequently used in health disparities epidemiology.

4.1 Background

In his influential paper *Sick individuals and sick populations*, Geoffrey Rose argued that epidemiology's roots in biomedicine often leads to a focus on identifying individual risk factors, and failure to recognize the importance of differences in exposure distributions (i.e.: the causes of causes) across populations in determining disease incidence rates [154]. This paper initiated a burst of critical public health thinking, some of it focused on how Rose's "population" strategies — blanket interventions aiming to shift risk distributions of the whole population — might lessen or exacerbate unjust health disparities (inequities) between groups [155, 156]. Frohlich and Potvin described an alternative "vulnerable population" approach, which refocused attention on segments of the population facing excess risk, while reimagining risk as socially (rather than individually) determined [155]. Recently developed causal inference methods such as parametric G-computation are useful for epidemiologists interested in moving beyond individual risk factors and instead quantifying the differences in population-level risk associated with interventions [157]. Stratified causal estimands can answer the question of "who benefits, and by how much?" Such analyses can also identify large effects in "vulnerable", small, or oft-ignored populations that may benefit disproportionately from intervention, which is particularly relevant for redressing historical health injustices. However, causal methods are still infrequently applied in racial health disparities research, where race-stratified analyses or studying race as an effect modifier are more common approaches [53, 158]. While simple interaction analyses identify whether exposures operate differently in strata of a possible modifier (e.g., race), those working in health disparities are concerned with not just identifying but eliminating disparities. Substitution methods have much to offer in applied health disparities research because they take an explicitly consequentialist approach to answering the question of "who benefits from an intervention, and by how much?" [159–162]. We motivate application of stochastic substitution estimators to studying racial health inequities with the following hypothetical example focused on the high distribution of ACEs among the Multiracial population, a population that is frequently

overlooked in population health research but bears a disproportionate burden of poor health outcomes.

Hypothetical scenario

Suppose you are a program planner focused on violence prevention in the Family Health Services Division (FHSD) of the ABC County Department of Health (DoH). ABC County is a very populous, urban, and diverse county in California with a large and growing population of Multiracial people. One day while reviewing literature, you come across a few papers and reports that document stark disparities in both childhood adversity and depression between monoracial and Multiracial people [32, 43, 44, 71, 80, 81]. You know from your work in violence prevention that adverse childhood experiences (ACEs) are strongly associated with depression, but were unaware of the striking Multiracial-monoracial disparities. You find this data particularly troubling, as you realize that health data on Multiracial people is frequently unavailable, and that Multiracial people are often overlooked in the design of public health interventions [5]. Perhaps you are one of the now one in ten Americans that self-identifies as Multiracial and thus have a personal interest in the issue [12].

You reach out to a colleague in the DoH epidemiology program and ask for help in pulling some data on the prevalence of ACEs and depression, and to investigate patterns in their relationship across racial groups. Your colleague asks what your goal is, and you respond, "I want to know (1) if health inequities in depression are associated with differential exposure to ACEs, and if so, (2) what racial groups would benefit most if we could eliminate inequities in exposure?" Fortunately, CA SB-428 mandates California health insurance plans to reimburse providers that conduct screening for ACEs, and thus ABC County has a wealth of data on the pediatric prevalence of ACEs [152]. Your epidemiologist colleague decides that because you are interested in understanding what populations should be prioritized for intervention, a sensible approach would be to specify a model regressing depression on an interaction between ACEs and race [163]. Using this model, you can estimate subgroup-specific associations and perform statistical tests to identify which groups experience the strongest effects and should thus be prioritized.

The epidemiologist assembles an appropriate analytic dataset, runs these analyses, and tells you that while ACEs and depression are indeed prevalent, interaction analyses did not identify any groups that should clearly be prioritized based on subgroup effects (results in Table 4.1. While these results don't tell the story you might have expected, you present them anyways to the ABC County health officer and FHSD leadership, who are explicitly interested in health equity and reducing disparities. You suggest that investing in ACEs prevention could help reduce depression inequities, and that doing so might especially benefit Multiracial people given this group's high exposure to ACEs, despite the results from the interaction analyses. The health officer asks you pointedly, "show me exactly what kind of reduction in disparities we can expect — I want numbers". The objective of this chapter is to demonstrate one approach to answering this question.

Our alternative approach

We present the scenario described here in order to draw attention to the limitations of statistical interaction analyses in applied health disparities epidemiology. Statistical interactions have useful properties for causal inference in epidemiologic research that have been described exhaustively elsewhere [163]. However, they are only one piece of information among many that can help identify appropriate populations for interventions. If we are interested in understanding which populations will benefit most from an intervention, or which populations to intervene on in order to have the greatest overall impact on reducing racial health disparities, we also need to know the relative sizes of the population subgroups of interest, and the distribution of exposures and associated confounding characteristics across those subgroups.

With these data, we can implement a substitution estimator as an alternative approach to identifying subgroups that will benefit most from a health equity intervention [164]. For example, we could estimate and compare the number of cases of disease per 100 that would be averted in each subpopulation if their exposure values were set to approximate that of a relatively more advantaged subgroup, such as those with the lowest exposure levels. We can improve the plausibility of our estimates by introducing some uncertainty in the form of random variation around the substituted exposure value. These "stochastic interventions" [157] specify a distribution of possible exposure values rather than a set deterministic value, which may involve unrealistic assumptions such as universal uptake or perfect adherence [165]. Together, these methods provide a principled approach to health disparities analysis that aligns with calls in the field of epidemiology for a stronger consequentialist focus, or a focus on informing policy and programs to improve health [159, 166].

As a demonstration of this set of methods, we regress depression against an interaction between ACE score and race in an adjusted model to estimate the expected reduction in racial disparities of depression under a hypothetical scenario in which all racial groups have ACE exposure distributions that match those of Whites. Our results provide one answer to the question of which subgroups would benefit most from an intervention program to bring health equity in ACEs exposure and depression incidence.

4.2 Methods

Data and analytic sample

Data for this analysis come from The National Longitudinal Study of Adolescent to Adult Health (Add Health), a nationally-representative prospective cohort of participants followed from adolescence in the 1990's to early adulthood [82]. Eighty public and private high schools and associated feeder schools for each were selected based on specific school characteristics. From these schools' rosters, 20,745 participants were selected from gender and grade-stratified samples, with oversampling of specific population subgroups to create a representative cohort of all US 7th-12th graders in 1994-95. Five waves of interviews taking

place from 1994-95 to 2016-18 (response rates ranging from 79-80.3%) collected detailed demographic, life and family history, socioeconomic, educational, relationship, health behavior, genetic, biomarker, and health and socioeconomic outcomes data. Study design and details are described exhaustively elsewhere [84, 149, 167].

Statistical analyses

The primary outcome in this analysis is a binary indicator of diagnosed depression or depression medication use at Wave 4. The exposure is ACEs score (see Appendix A for details), interacted with Wave 3 race (categorized as monoracial White, Black, Asian, or American Indian/Native American (AI/NA), or Multiracial). We controlled for the following variables from the indicated waves; Wave 4: age, self-reported gender, experiences of racism (dichotomous indication of ever being treated with less respect or courtesy than others because of race, ancestry, national origin, or skin color); Wave 1: highest parental education (categorized ordinally as less than high school, completed vocational school, or equivalency (GED); high school diploma (HSD); some college; college graduate or greater), household size-adjusted income (continuous equivalence scale of pretax household income divided by the square of household size), parental support (mean of five Likert-scale questions about warmth, communication, caring, and closeness in relationship with parents, see Appendix B for details), neighborhood disadvantage score (mean of census-tract proportions of households earning less than federal poverty level, households receiving public assistance, Civilian unemployment, persons 25 years or older lacking HSD or GED, and female-headed households), and interviewer racial misclassification (if Add Health interviewer perceived the participant's race differently from participant's self-identified race), a variable others have used as a proxy for stressful, racist microaggressions often experienced by Multiracial individuals [9, 148].

We excluded participants self-identifying as "Other" race alone because of numbers too small for meaningful interpretation, and participants self-identifying as Hispanic/Latino ethnicity because it is impossible to accurately identify Multiracial Hispanic/Latino people when race and Hispanic/Latino ethnicity are assessed separately [34, 130].

Stochastic intervention

Our target causal quantity is the race-specific difference in average risk (Ψ_R) of depression (Y) if each racial group's (R) distribution of ACEs (A_R) reflected the exposure distribution of the White population (A_{White}), given an individual's characteristics W. We choose the ACEs distribution of Whites because this group on average has a low mean ACE score and the largest sample size to provide support for precision in our estimates. Let $g^{\mathrm{R}}_{A|w}$ denote the ACEs distribution for race R. We can then represent this target quantity as a risk difference using the following notation:

$$\Psi_R = E_{W,g^{\mathrm{White}}_{A|w}}\left(E[Y|A,W,R]\right) - E_{W,g^{\mathrm{R}}_{A|w}}[E[Y|A,W,R]]$$

To estimate this value, we used Monte Carlo simulation methods to represent interventions with some stochastic variation [157, 168]. Specifying a substitution estimator that accounts for the stochastic nature of the intervention across the population, rather than treating exposure as a static value, is beneficial as it is often unrealistic to assume that every person receiving the intervention is affected in a uniform manner [165]. In order to introduce random variability and calculate uncertainty intervals, we use parameter resampling instead of bootstrapping [169–171] as it is more computationally efficient for complex estimands such as population-level parameters that incorporate survey design effects.

To estimate our target parameter, we average over I simulated risk differences in depression outcomes for each racial group, which are each averaged across the intervention distribution A, given covariate distribution W. Following the epidemiologist's analytic approach described in the above fictional scenario, we specify an adjusted quasi-Poisson model to estimate relative risk [172], regressing depression on an interaction between ACEs and race, adjusting for all covariates listed above. To model the intervention exposure value, we estimate the mean ACE score for the White group using unadjusted Poisson regression.

We used complex survey weights to incorporate design effects using the "survey" package in R [97]. Because of high levels of missingness (up to 30%) in ACE components, covariates, and outcomes, we performed multiple imputation to reduce biased estimation due to missing data using the "mi" and "mitools" packages in R [98, 99]. We performed Markov chain imputation with 30 iterations and 4 chains, including the depression outcome in our imputation model, and pooled results from 20 imputed datasets.

1. First, we use the estimated beta coefficients and variance-covariance matrix from our interaction model to generate a single resampled value of the beta coefficients from a multivariate normal distribution [169–171].

2. We then resample with replacement m times from one imputed data frame, where m equals the number of observations in the dataset. We use these resampled data to get the distribution of covariates under no intervention.

3. We use the coefficients estimated in step 1 to obtain the predicted probability of depression for each observation, and then sum across all observations to estimate the race-specific prevalence of depression under no intervention.

4. Next, we create a copy of the resampled data from step 2, replacing the ACEs score for all non-White participants with the sampled rate parameter among White participants estimated from an unadjusted Poisson model.

5. We repeat step 3 to calculate the prevalence of depression in this single draw from the intervention distribution.

6. We calculate the causal risk differences by race as the averaged risk differences across each observation in the various racial groups from the resampled population.

7. Finally, we repeat this analysis, averaging over the race-specific exposure distribution $g^{\mathrm{R}}_{A|w}$) across each of $i = 1000$ resampled versions of the beta coefficients and dataset. From these $i = 1000$ resampled estimates, we report median differences as race-specific point estimates and quantile-based 95% uncertainty intervals around the risk differences.

Example code in R for this simulation is provided at https://github.com/lamhine.

4.3 Results

Table 4.1 summarizes the results of the baseline interaction analysis. The AI/NA and Multiracial groups have the highest mean ACE score, and depression prevalence is highest among the Multiracial group. ACEs were significantly associated with depression among White, Black, and Multiracial groups, but confidence intervals of subgroup risk ratios overlap and a global test for interaction was insignificant, suggesting the absence of interaction between ACEs and race on the multiplicative scale. However, the Black and Asian groups had small but significantly negative interaction (RERI) on the additive scale compared to the White group, while the White, AI/NA, and Multiracial groups were not significantly different.

Table 4.2 presents the baseline and intervened ACE scores for each racial group from 1,000 resamples of the single imputed dataset. Resampling the datasets 1,000 times resulted in much larger sample sizes for each racial group: the AI/NA sample size increased from 77 in the original dataset to 76,865, while the number of observations of Whites was over 7.76 million. The mean of the resampled baseline ACE scores are slightly lower for all groups than in the original dataset; differences are most pronounced for the AI/NA group (original mean baseline = 2.83, mean of resampled mean baselines = 2.75). Figure 4.1 shows that our stochastic intervention shifted the ACEs distribution substantially lower for the AI/NA, Black, and Multiracial groups and skewed the distribution slightly higher for Asians. We did not intervene on the White distribution, and so there was no change in shape for that group.

Table 4.3 summarizes the population-averaged results of our simulated intervention. The median risk difference was largest for the Multiracial population, with 1.470 cases per 100 population averted under repeated draws of the simulation. Our model estimated that the intervention would prevent 0.258 cases per 100 among the AI/NA population. The model estimate was not very precise for this group and despite the large estimate of the number of cases averted, the uncertainty interval included the null. The simulation predicted that the Black population would see a reduction of 0.568 cases per 100. The model predicted an increase of 0.002 in cases per 100 for Asians — consistent with the slight visual skew upwards in distribution — though the uncertainty interval for this estimate also crossed the null. Figure 4.2 plots the population-averaged risk differences produced in each of our 1,000 intervention draws across 1,000 resample datasets. Estimate precision was greatest for Black,

Table 4.1: Summary of results from adjusted[1] interaction model[2] for depression

Group	n	Mean ACEs	Depression prevalence	RR (95% UI)	RERI (95% UI)[3]
White	7760	2.22	20.6%	1.16 (1.13, 1.20)	(ref.)
Black	2917	2.53	10.3%	1.29 (1.20, 1.39)	-0.067 (-0.086, -0.056)
Asian	808	2.22	7.6%	1.26 (0.95, 1.68)	-0.086 (-0.219, -0.076)
AI/NA	77	2.83	14.1%	1.08 (0.77, 1.51)	-0.095 (-0.689, 0.008)
Multiracial	838	2.73	23.6%	1.20 (1.12, 1.28)	0.015 (-0.051, 0.055)

[1] Estimated from imputed, design-based, quasi-Poisson model regressing depression on interaction term between ACEs and race, adjusting for age, gender, parental education, equivalence-scaled household income, parental support, neighborhood disadvantage, experiences of racism, and racial misclassification
[2] Model global test of significance p-value = 0.14
[3] Null value is zero. 95% uncertainty intervals calculated from 10,000 resamples of RERI [169]

Table 4.2: Summary of counts and means of baseline and intervention ACE scores from resampled and simulated data

Group	n	Baseline	Intervened
White	7,760,066	2.21	2.21
Black	2,917,831	2.43	2.22
Asian	807,540	2.21	2.22
AI/NA	76,865	2.75	2.21
Multiracial	837,698	2.69	2.21

Table 4.3: Estimated number of cases of depression averted per 100 population, under hypothetical intervention

Group	Median Risk Difference	95% UI	Std. error
White	0	null	null
Black	-0.568	(-0.896, -0.318)	0.0047
Asian	0.002	(-0.361, 0.481)	0.0062
AI/NA	-0.258	(-3.450, 1.66)	0.0392
Multiracial	-1.470	(-2.330, -0.803)	0.0121

Asian, and Multiracial groups; there was much higher variability in the risk differences for AI/NA groups. These are reflected in the quantile-based 95% UIs in Table 4.3.

Figure 4.1: Comparison of intervention and baseline ACE score distributions resulting from 1,000 resampled datasets

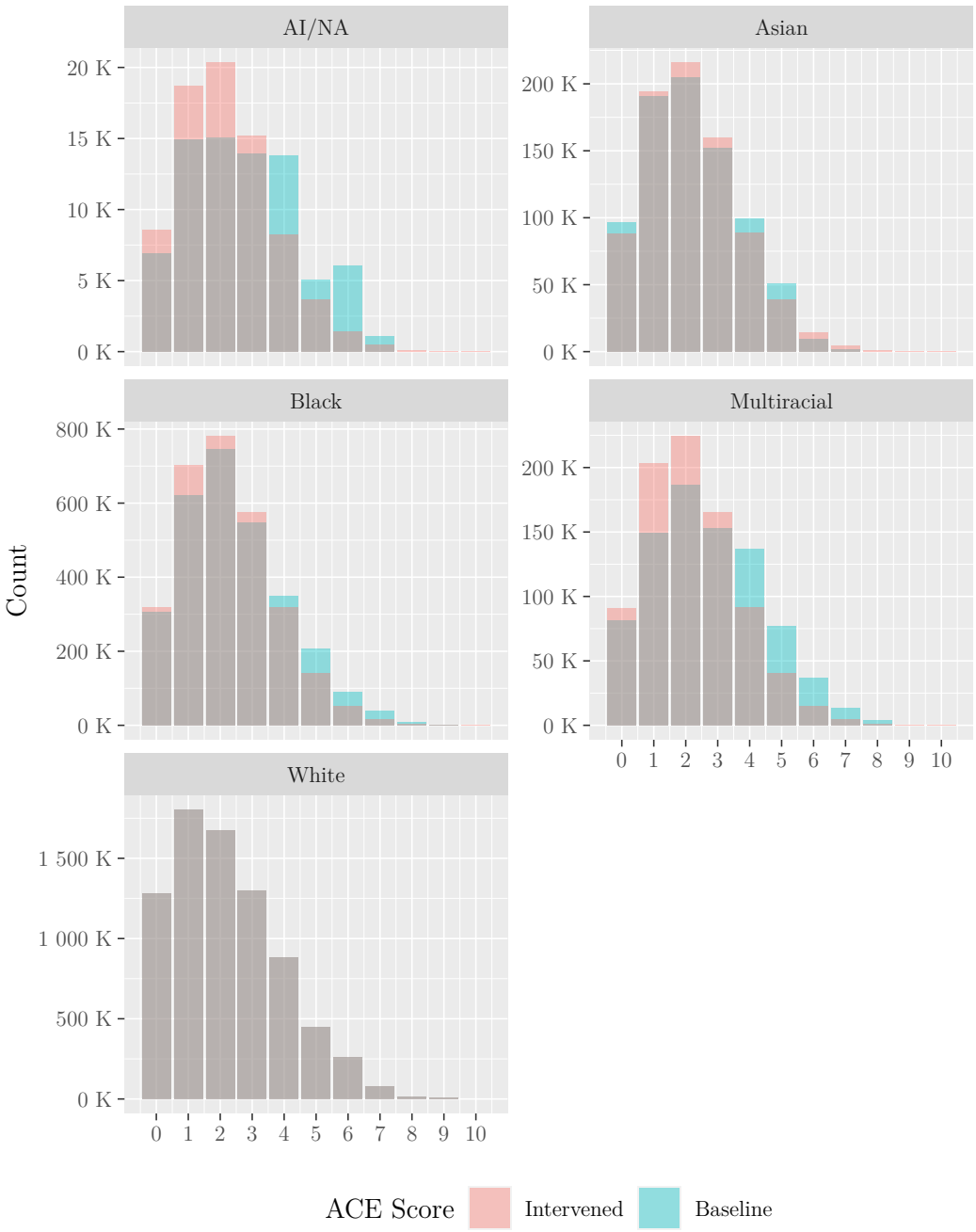
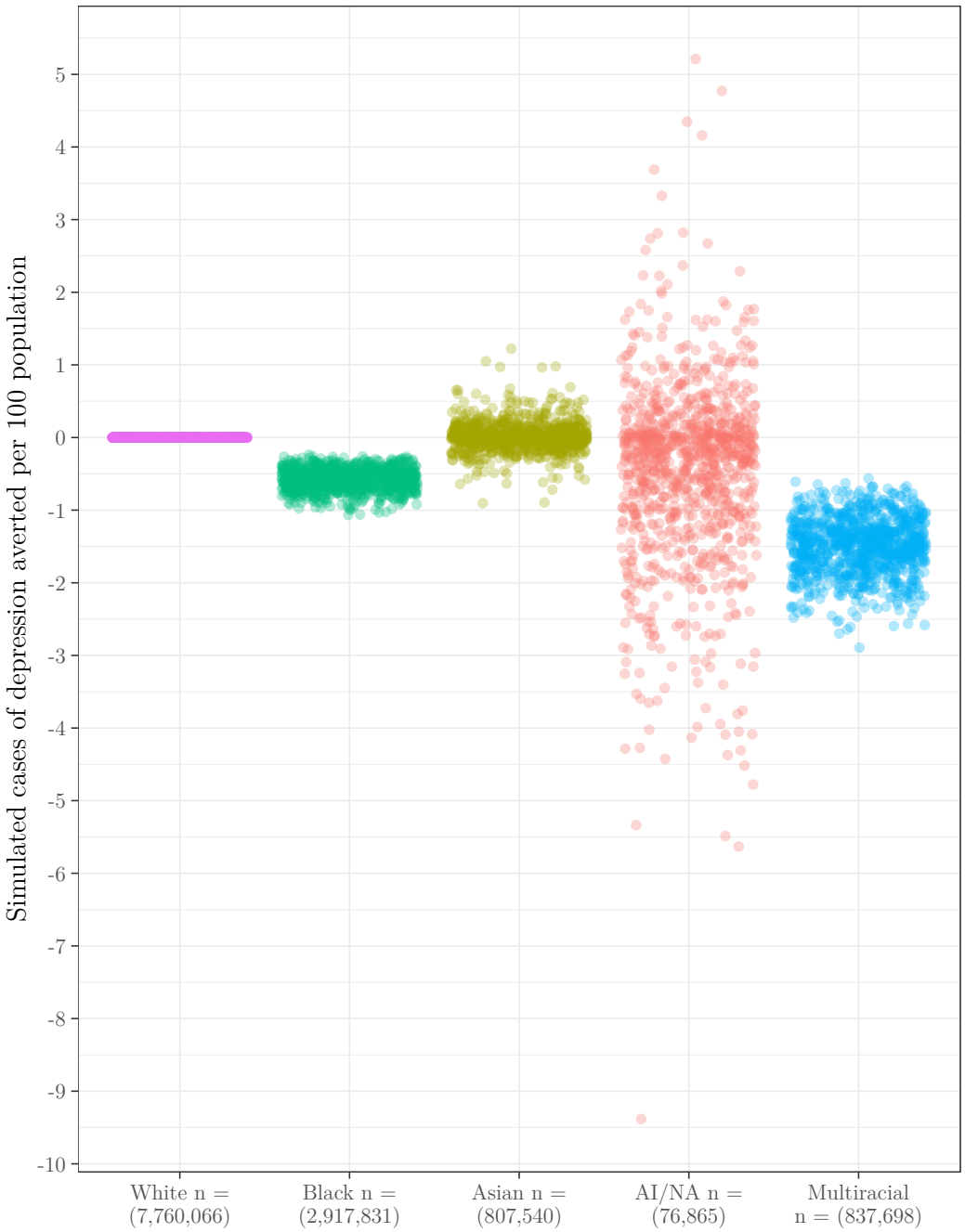


Figure 4.2: Simulated number of cases of depression averted per 100 population, results from 1,000 resampled datasets



4.4 Discussion

In this paper we illustrate a principled approach to estimating differences in the population impact of a hypothetical intervention in defined subgroups, as one way of identifying groups that might benefit the most from the intervention. Our analysis shows that there are substantial racial differences in the number of depression cases averted associated with an intervention to reduce disparities in exposure to ACEs, with the Multiracial population standing to benefit most from such an intervention. These differences are easily masked in subgroup effect analyses, where population sizes and distribution of exposures are not usually considered. However, as we have demonstrated, stochastic intervention models allows us to quantify the reduction in burden of disease across racial groups at a population level under altered exposure distributions.

A natural next step for practitioners interested in intervening would be to contextualize the results from this simulation with other potential interventions to reduce the rate of depression among Multiracial individuals, and potentially to estimate costs associated with such interventions in a cost-effectiveness analysis. Our methods can be extended to model different kinds of interventions — for example, a uniform reduction in the ACEs score rather than a shrinking of distributions — or an intervention reducing the ACE score of individuals within a given strata of socioeconomic position (SEP). This demonstration's modeled alteration of exposure patterns is just one example of a potential health equity-focused intervention, but it draws attention to the health benefits realized when inequities in exposure to ACEs are eliminated.

This study had a few limitations. First, high variability in the AI/NA group's risk ratio estimates from the underlying model are not eliminated with repeated resampling; future studies should make greater efforts to include larger proportions of vulnerable populations to improve inference. Second, our demonstration is purely hypothetical because it models intervening directly on ACEs; in reality ACE prevention strategies target determinants of ACEs such as family psychosocial or economic stress or caring adult presence [72]. Simulating interventions to modify determinants of ACEs would have more policy relevance and thus better align with calls for consequentialist epidemiologic research. Still, we believe simulating intervention on ACE scores is useful for demonstrating the limitations of focusing solely on identifying subgroup effects. Finally, our simulations do not specify a dependence between the intervened ACEs score and an individual's set of covariates. In reality, it is unlikely that the effect of the intervention would be random; instead, effects would likely be determined by an individual's SEP, environment, access to care, and other characteristics. Incorporating such individual-level covariate data into future analyses would improve the inference associated from this kind of simulation.

Plainly said, inequities in exposure to ACEs and prevalence of depression among the Multiracial population are concerning, yet Multiracial people are rarely studied or prioritized in interventions to reduce disparities in depression. Our analysis shows that racial inequities in exposure to ACEs are associated with a substantial population-level disparities in depression, and that the Multiracial population would benefit significantly from a reduction in ACEs

compared to other groups. We assert that strategies to reduce racial disparities in depression should focus on reducing exposure to childhood adversity, with a particular emphasis on the Multiracial population. Public health researchers and practitioners should employ rigorous methodologies to weigh different interventions and suitable target populations to remedy inequities in exposures and health outcomes. Others have described alternative approaches such as mediation analyses to answer similar questions [173]. Health equity will only be achieved when disparities are intervened upon, and stochastic substitution is one method among many that can help us make progress towards those aims.

Chapter 5

Conclusion

This goal of this dissertation was to describe the epidemiology of childhood adversity among Multiracial Americans, filling in gaps in evidence, and providing alternative research approaches to studying the health of the fastest-growing racial group in the US. These three papers are significant because they explicitly center the experience of the Multiracial population, an approach that is rarely taken in quantitative health research. In taking both a critical and computational perspective in my analyses, I hope that this dissertation provides theoretically-grounded evidence and actionable tools for public health practitioners working to address childhood adversity and health inequities in our society.

Chapter 2 began this dissertation's exploration of childhood adversity as a health risk for Multiracial Americans with descriptive analyses of patterns of ACEs in this population. I investigated drivers of high rates of ACEs in the Multiracial population, specifically testing a hypothesis that the high mean score in the Multiracial population was related to the fact that the largest group of Multiracial people in the US claim AI/NA ancestry, a group that also experiences high rates of childhood adversity. This chapter found that — consistent with previous literature — associations with ACEs were strongest for mental and behavioral health outcomes, and that this pattern was true for all racial groups studied. Multiracial AI/NA participants had higher mean ACEs score than White, Black, or Asian participants. However, there was no significant difference in mean scores between the two Multiracial groups and AI/NA participants, and no significant difference in ACE component scores except for sexual abuse and mother treated violently (for which Multiracial AI/NA participants had higher risk in only sexual abuse). These results do not support the hypothesis that high mean ACE scores in the Multiracial population are driven by the large proportion of Multiracial people with AI/NA ancestry, and suggest there may be other factors unique to the experience of Multiracial people that lead to elevated risk. We recommend that future studies repeat these analyses in larger datasets, and that these datasets should make detailed race variables available in public access versions to facilitate more Multiracial disaggregation studies.

In addition to high mean ACE scores, Multiracial adolescents and adults also have increased risk of physical, mental, and behavioral health outcomes: outcomes that are also strongly associated with ACEs. Building on the findings from chapter 2, one of the goals of

chapter 3 was to determine if these elevated rates of disease were linked to increased exposure to ACEs, and to compare the associations for Multiracial people with other racial groups. In particular, I examine adjusted associations by race with metabolic syndrome, hypertension, asthma, anxiety, depression, suicidal ideation, and problematic drug use by specifying a statistical interaction term between ACEs and race. This chapter found mixed results: while there were significant statistical interactions between ACEs and race in anxiety and problematic drug use, no one racial group experienced consistently stronger associations than others, and the strength and direction of interactive association varied by outcome, group, and the interaction scale used. These results suggest that ACEs interact with race in complex ways that do not reliably align with social constructivist theories of health, warranting further research into the most effective strategies to prevent ACEs and their sequelae.

The results of chapter 3 directly informed the aims of chapter 4, which were to highlight limitations of interaction analyses in racial disparities research, and to offer an alternative approach to identifying target subgroups for interventions to reduce racial disparities. Using adjusted models specified in chapter 3 regressing depression on an interaction between ACEs and race, I use stochastic substitution methods to simulate the population-level rate of cases of depression prevented if all racial groups' ACEs distributions approximated that of the White group. The simulation showed that while there were no significant differences in the strength of association between ACEs and depression for White and Multiracial participants, a health equity-focused intervention to prevent ACEs would disproportionately benefit the Multiracial population. This study shows that simulating stochastic interventions can be an important tool in health disparities research, and can identify large effects in smaller or overlooked populations, which may be important to redress health inequities.

While a primary aim of this dissertation was to describe childhood adversity as an important health risk for Multiracial people, questions still remain as to the reasons for elevated risk in this population. Epidemiologic data are unlikely to help fully explain these patterns; more qualitative and descriptive research is needed in order to understand drivers of this increased risk. Indeed, while structural and institutional racism are determinants of disparities in ACEs, it is still unclear how these macro-level forms of racism affect Multiracial people, or if Multiracial-monoracial ACEs disparities are due to something else entirely. Current prevailing ACEs prevention strategies could be more critical in approach; despite clear racial disparities in exposure to ACEs, the Centers for Disease Control and Prevention's 26-page ACEs prevention framework mentions race in exactly three sentences, and does not mention Multiracial people at all [72].

Perhaps more importantly, ACEs are only a single health risk that disproportionately affects this population. Far more research is needed to better characterize the health status of Multiracial people and subgroups in this population. Progress has been inhibited in no small part by a preference to ignore rather than explore Multiraciality, reliance on statistical methods that are not well-suited to small and complex populations, and datasets that suppress detailed race variables, preventing further subgroup disaggregation. Describing the social position of Multiracial Americans and how that position influences health will require a complex, nuanced understanding of social processes and structures unique to the Multiracial

experience that has not yet been well articulated in public health literature.

As the Multiracial population grows in size and visibility in the US, we must ask ourselves: will we continue to collectively prioritize the study of monoracial people at the expense of Multiracial people? If we do so, what are the consequences for population health, disparities, and inequities? What are the consequences for the social position of Multiracial people in a racially stratified society? That is a future that I hope to not imagine or experience. This dissertation is the beginning of a larger research agenda on monoracism and its effects on the health — and our understanding of the health — of Multiracial Americans. My hope is that these papers and research agenda spur the imaginations of others who are interested in building a more just, equitable, and inclusive society where all people are valued and are able to thrive.

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Appendix A

Crosswalk of topical domains, questions in Kaiser ACEs questionnaire, and questions in Add Health used to create ACEs score

Domain	**ACE Questionnaire**	**Add Health Measure Coding**
Emotional abuse	1. Did a parent or other adult in the household often... Swear at you, insult you, put you down, or humiliate you? or Act in a way that made you afraid that you might by physically hurt? Yes/No	**H4MA1** (Wave 4): Before your 18th birthday, how often did a parent or other adult caregiver say things that really hurt your feelings or made you feel like you were not wanted or loved? 1 = 1-10 times or more 0 = this never happened
Physical abuse	2. Did a parent or other adult in the household often... Push, grab, slap, or throw something at you? or Ever hit you so hard that you had marks or were injured? Yes/No	**H3MA3** (Wave 3): Before the time you started 6th grade, how often had your parents or other adult caregivers slapped, hit, or kicked you? 1 = 1-10 times or more 0 = this never happened
Sexual abuse	3. Did an adult or person at least 5 years older than you ever... Touch or fondle you or have you touch their body in a sexual way? or Try to actually have oral, anal, or vaginal sex with you? Yes/No	**H3MA4** (Wave 3): Before the time you started 6th grade, how often had your parents or other adult caregivers touched you in a sexual way, forced you to touch him or her in a sexual way, or forced you to have sexual relations? 1 = 1-10 times or more 0 = this never happened
Emotional neglect	4. Did you often feel that... No one in your family loved you or thought you were important or special? or Your family didn't look out for each other, feel close to each other, or support each other? Yes/No	**H1PR8** (Wave 1): How much do you think your family pays attention to you? 1 = quite a bit - very much 0 = not at all - somewhat

Domain	ACE Questionnaire	Add Health Measure Coding
Physical neglect	5. Did you often feel that... You didn't have enough to eat, had to wear dirty clothes, and had no one to protect you? or Your parents were too drunk or high to take care of you or take you to the doctor if you needed it? Yes/No	By the time you started 6th grade, how often had your parents or other adult care-givers: **H3MA1** (Wave 3): Left you home alone when an adult should have been with you? or **H3MA2** (Wave 3): Not taken care of your basic needs, such as keeping you clean or providing food or clothing? 1 = 1-10 times or more for either 0 = this never happened
Parental separation or divorce	6. Were your parents ever separated or divorced? Yes/No	**PA38 - PA54** (Wave 1 Parent) 1 = parents divorced or separated before participant turned 18 0 = parents not divorced or separated before participant turned 18
Mother treated violently	7. Was your mother or stepmother: Often pushed, grabbed, slapped, or had something thrown at her? or Sometimes or often kicked, bitten, hit with a fist, or hit with something hard? or Ever repeatedly hit over at least a few minutes or Threatened with a gun or knife? Yes/No	**PB20** (Wave 1 Parent): How much do you fight or argue with your current (spouse/partner)? 1 = a lot 0 = not at all - some

Domain	ACE Questionnaire	Add Health Measure Coding
Household substance abuse	8. Did you live with anyone who was a problem drinker or alcoholic or who used street drugs? Yes/No	**PC49E_2** (Wave 1 Parent): His/her biological father has alcoholism? or **PC49E_3** (Wave 1 Parent): His/her biological mother has alcoholism? or **H1TO52** (Wave 1): Are illegal drugs easily available to you in your home? 1 = yes to any 0 = no to all
Mental illness in household	9. Was a household member depressed or mentally ill or did a household member attempt suicide? Yes/No	**PA20** (Wave 1 Parent): In general, are you (main parent respondent) happy? or **PB16** (Wave 1 Parent): In general do you think (he/she) (main parent respondent's partner or spouse) is happy? or **H1SU6** (Wave 1): Have any of your family tried to kill themselves during the past 12 months? 1 = yes to PA20, yes to PB16, and no to H1SU6 0 = any other combination of responses

Domain	ACE Questionnaire	Add Health Measure Coding
Incarceration of household member	10. Did a household member go to prison? Yes/No	**H4WP3** (Wave 4): Has/did your biological mother ever spent/spent time in jail or prison? or **H4WP9** (Wave 4): Has/did your biological father ever spent/spend time in jail or prison? or **H4WP16** (Wave 4): Has/did your mother figure ever spent/spend time in jail or prison? or **H4WP30** (Wave 4): Has/did your father figure ever spent/spend time in jail or prison? 1 = yes to any 0 = no to all

Appendix B

Key questions used to construct Add Health covariate and outcome measures

Variable	**Add Health Questions**	**Coding**
Metabolic syndrome	Wave 4 and Biomarker File 1. Hypertension a) Systolic blood pressure ⩾ 130 mmHg (H4SBP), or b) Diastolic blood pressure ⩾ 80 mmHg (H4DBP), or c) Ever diagnosed with hypertension (H4ID5C), or d) Current hypertension medication use (medication file) 2. Waist circumference (> 102 cm for males, > 80 cm for females) (H4WAIST) 3. Triglycerides (top 3 deciles for males, top 2 for females) 4. High-density lipoprotein (bottom 2 deciles for males, bottom 3 for females) 5. Glycated hemoglobin (HbA1c) > 5.7%.	1 = exceeding cut-off in three or more of the five categories 0 = exceeding cut-off in fewer than three categories
Asthma	**H4ID5F** (Wave 4) Has a doctor, nurse or other health care provider ever told you that you have or had asthma, chronic bronchitis or emphysema? Yes/No	1 = yes 0 = no
Depression	**H4ID5H, H4MH18-27** (Wave 4) Have you taken prescription medication in the past 12 months for depression or stress or Has a doctor, nurse or other health care provider ever told you that you have or had depression? Yes/No	1 = yes to either 0 = no to both

Variable	Add Health Questions	Coding
Anxiety	**H4ID5J** (Wave 4) Has a doctor, nurse or other health care provider ever told you that you have or had: anxiety or panic disorder? Yes/No	1 = yes 0 = no
Suicidal ideation	**H4SE1** (Wave 4) During the past 12 months, have you ever seriously thought about committing suicide? Yes/No	1 = yes 0 = no
Problematic drug use	**H4TO63, H4TO65C-E, H4TO66** (Wave 4) Have you ever taken any prescription drugs that were not prescribed for you, taken prescription drugs in larger amounts than prescribed, more often than prescribed, for longer periods than prescribed, or taken prescription drugs that you took only for the feeling or experience they caused? or Have you ever used cocaine, crystal meth, or other drugs (not including marijuana or steroids)? or Have you ever injected (shot up with a needle) any illegal drug, such as heroin or cocaine? Yes/No	1 = yes to any 0 = no to all
Parental support	**H1PF1, H1PF4, H1PF5, H1NM14, H1WP10, H1PF23-25, HI1NF14, H1WP14** (Wave 1) Most of the time, your mother/father is warm and loving toward you You are satisfied with the way your mother/father and you communicate with each other Overall, you are satisfied with your relationship with your mother/father How close do you feel to your mother/father? How much do you think your mother/father cares about you? Very much, quite a bit, some, a little, none at all)	5 = very much 4 = quite a bit 3 = some 2 = a little 1 = none at all

Variable	**Add Health Questions**	**Coding**
Racial discrimination	**H4MH29** (Wave 4) What do you think was the main reason for being treated with less respect or courtesy than other people? National ancestry or origin, gender, race, age, religion, height or weight, shade of skin color, sexual orientation, education or income, physical disability, other	1 = national ancestry or origin, race, or shade of skin color 0 = all others