Examining the Role of Social Support and Neighborhood Deprivation in the Relationship Between Multiple ACEs and Health Risk Behaviors

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Examining the Role of Social Support and Neighborhood Deprivation in the Relationship
Between Multiple ACEs and Health Risk Behaviors

by

Marin L. Henderson-Posther

A dissertation submitted in partial fulfillment of the requirements for the degree of

Doctor of Philosophy
in
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Abstract

The accumulation of multiple adverse childhood experiences (ACEs) is associated with the disproportionate development of health risk behaviors (HRBs), such as smoking, substance use, physical inactivity, and risky HIV behaviors. The impact of neighborhood social inequities on the association between multiple ACEs and HRBs is not well known. This study aims to examine the impact of stressors associated with neighborhood deprivation on ACE-related HRBs, the potential protective factor of perceived social support (PSS), as well as better understand disproportionality experienced by racial/ethnic minorities. Through merging data from the 2010 Washington State Behavioral Risk Factor Surveillance System survey ($n = 5,447$) with the Neighborhood Deprivation Index (NDI), an NDI value is assigned based on participant zip code. Using structural equation modeling, this study demonstrates NDI mediates the relationship between multiple ACEs and HRBs, while both low and high PSS moderate this relationship. Additionally, the impact of exposure to multiple ACEs on health risk behaviors remained steady regardless of race/ethnicity. Acknowledging the contributions of neighborhood stressors and individual protective resources furthers the ACEs knowledgebase by providing a more integrated model of ACE-related disease production, improving explanatory mechanisms and clarifying the role of socio-structural factors in health disparities. Further, contextualizing the unique variances in ACE-related pathways depending on social and neighborhood factors enables more holistic interventions and preventative action at the community level, including policies targeting poverty, education, and housing conditions and increasing community social support assets.
Dedication

For my parents, my husband, Nick, and my children, Winnie and Rowen Wilder, whose very existence fills my heart.
Acknowledgements

Positionality Statement

Attending to positionality, grounded in ongoing self-reflection throughout the research process, is critical to conducting ethical, sound research (Holmes, 2020). In doing so, I am acknowledging that my position as a researcher is not “neutral” or “value-free,” and requires ongoing analysis of my motivation for pursuing this research (Holmes, 2020, p. 4). This practice clarifies both the paradigmatic influences on my methods, and maintains a sense of the real life experiences each “data point” contains, particularly when the topic of investigation is as sensitive and emotionally charged as childhood trauma. My impetus for pursuing adverse childhood experiences research started when I spent several years working as a mental health clinician with adults in high inequity settings. Experiences of severe and haunting childhood trauma – as well as engagement in health risk behaviors – were not uncommon among these individuals. However, many of my fellow social workers had a shared sense of disdain when discussing ACEs. For many of them, the ACEs measure held little practical application because of its omission of the numerous, intersecting systemic factors in our clients’ lives; it was devoid of place (Bruner, 2017).

While the ACEs measure continues to demonstrate critical research findings, many social workers feel this research can inadvertently dehumanize people, treating them as “risk factors” who must shoulder the burden of any negative health outcomes arising from their childhood trauma; that the research does not actively center peoples’ lived experiences (and resources) that also play a contributing role (Steptoe, 2019). The measure, and its related research, excludes the biopsychosocial, person-in-environment,
and strengths-based approaches social workers rely on. As an ACEs researcher and social worker, I am seeking to instill a new perspective on childhood trauma by investigating how multiple ACEs function in high inequity neighborhoods where several oppressive factors are present across one’s life (Hughes et al., 2017, e363). Similarly, in operationalizing a critical paradigm, my research approach attempts to clarify how systems of power can operate within the research process itself. This can be seen through a researcher’s use of identity labels, like sex, race, or sexual orientation, as “risk factors” or confounders, as it obscures the true systemic contributors, such as racism and economic oppression, and pathologizes people versus oppressive systems (Steptoe, 2019).

My positionality as a researcher also extends to acknowledging and being mindful of the use of language in the research process. For example, while using the term “ACE” is helpful shorthand for adverse childhood experiences, it can also distance readers from participant experiences. Thus, I would be remiss without reminding readers that the word ACE contains incredibly painful experiences from which many people struggle throughout their lives: physical abuse, sexual abuse, the neglect of a caregiver. Health risk behaviors are evidence – symptoms – of this ongoing struggle from accumulated trauma, and, similarly to the current investigation’s emphasis on the importance of reintroducing socio-structural context, we cannot “sterilize” research by separating this word from its inherent emotional context. In doing so, I am hopeful to honor and render visible the numerous survivors of childhood trauma and improve the utility of the ACEs measure.
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Chapter 1:

Introduction

The accumulation of four or more adverse childhood experiences (ACEs) – also referred to as “multiple ACEs” – is associated with the disproportionate development of many leading health problems, contributing to poorer health (Hughes et al., 2017, p. e356). Research continues to indicate that experiencing multiple ACEs has more damaging long-term effects than singular exposures and is consistently associated with prevalence in adulthood health risk behaviors (HRBs) (Goldstein et al., 2020; Hughes et al., 2017). Current biobehavioral theories posit that these HRBs, such as smoking, drinking, and/or risky HIV behaviors are commonly initiated as a coping device to deal with the stress from childhood trauma. When used throughout the life course, these behaviors contribute to the development of diseases like cancer, stroke, HIV/AIDS, emphysema, and heart disease. While numerous studies acknowledge multiple ACEs as a “tipping point” in the accumulation of childhood trauma, the pathways contributing to the association between multiple ACEs and the development of HRBs have been underexplored, particularly in examining the role of stressors associated with neighborhood social inequities. Consequently, there is a need for research identifying the factors explaining and influencing this relationship to potentially circumvent or intervene upon these behaviors (Steptoe, 2019).

This study aims to fill a gap in the ACEs knowledgebase by identifying specific and potentially amenable social and structural factors that explain and influence the increased prevalence of HRBs among those with multiple ACE exposures. In doing so, this study seeks to transform perspectives on ACE-related health behaviors to
contextualize and implicate systemically oppressive contributions versus the current and misplaced emphasis on individualized risk. Individualized risk tends to pathologize groups experiencing disparate outcomes and indirectly blames individuals for engaging in HRBs as a result of trauma they experienced through no fault of their own (Pauly et al., 2017; Steptoe et al., 2019; Krieger, 2001). Zooming out and situating individuals in their mezzo or macro-level context illuminates obstacles in the environment, as well as widens the scope of potential solutions to include systemic changes necessary for the eradication of ACEs (Krieger, 2001; Bruner, 2017). To better clarify these disparities in health outcomes, current research suggests health models “should be broadened to incorporate both stress and resource experiences simultaneously,” and also to unpack the multi-level determinants within the social environment in order to provide a more “integrative view” of health (Wickrama et al., 2015, p. 1109). In line with this, health research supports evidence of perceived social support (PSS) as a buoying resource and neighborhood deprivation as a deleterious mechanism in a multitude of health problems (Messer et al., 2006; Thoits, 2011; Dong et al., 2004; Jones et al., 2018).

Neighborhood deprivation refers to “the concentration of multiple markers of economic disadvantage” within a community (Messer et al., 2006, p. 1042). However, this construct conveys a much more sophisticated picture than just the socio-economic status of one’s community; it also captures various forms of social inequity within that community and the inherent associated stress. This includes how social status stratifies life and job opportunities, access to wealth and assets, and types of living conditions, all of which are important determinants of health (Messer et al., 2006, p. 1048). For those with multiple exposures to childhood trauma, the neighborhood context provides
important information about the absence or presence of embedded, chronic stressors, which may better explain why individuals with multiple types of trauma are more likely to develop HRBs. By encapsulating the numerous types of inequity characteristic of neighborhood deprivation and assessing this variable as a mediating force, we can parse out the structural contributions to stress accumulation from childhood trauma. Relatedly, by assessing the influence of someone’s perception of emotional and social support in their lives – having someone to consistently turn to when they are in need – we can begin to understand the impact of individual level forces interrupting the stress pathways from childhood trauma.

The relationship between high neighborhood deprivation and the occurrence of certain non-ACE related HRBs, such as prenatal drug use, as well as the influence of one’s social supports on ACE-related outcomes, make these variables plausible mechanisms on the pathway between multiple ACEs and increased HRBs (Messer et al., 2006; Bruner, 2017; Thoits, 2011). Thus, this study aims to test the hypothesis that neighborhood deprivation mediates the relationship between the accumulation of multiple ACEs and HRBs, specifically smoking, binge/heavy drinking, drinking and driving, physical inactivity, drug use, and risky HIV behavior, while PSS and race/ethnicity moderate this relationship. With the intention of extending current biologic ACE explanatory models, neighborhood deprivation widens the concept of contributing toxic stressors to systemic factors, and encapsulates the “socioeconomic mechanism” by which social inequity can increase health risk behaviors (Font & Maguire-Jack, 2016, p. 398). More specifically, the accumulation of multiple ACEs contributes to increased socioeconomic adversity, which includes toxic stress and reduced opportunities for health,
contributing to the adoption of health risk behaviors (Font & Maguire-Jack, 2016). Because the bulk of ACEs research focuses on individual-level explanations for health disparities, widening the scope to integrate the influence of both individual resources and societal barriers to health may better illuminate the ramifications of stacked inequities and provide a more holistic depiction of the role of the socio-structural environment (Bruner, 2017). Contextualizing the role of the environment offers new points of intervention at the community level, and it also changes the focus to *prevention* instead of remediating what has already happened, such as with typical clinical practices for handling child trauma (Bruner, 2017).

An overarching premise of this study is that toxic stress and reduced opportunities for health that are associated with neighborhood deprivation adversely influence HRB outcomes from ACEs (Melton-Fant, 2019, p. 1). Research indicates the ongoing, persistent adversities characteristic of high deprivation environments contribute to the development of toxic stress, creating a cumulative effect on stress embodiment, affecting psychological and physiological processes (Jones et al., 2018; p. 33; Wickrama et al., 2015; Goldstein et al., 2020). The stress induced by childhood trauma is rarely isolated; it often occurs within a larger structure of adversity and stress proliferation (Jones et al., 2018). While previous studies posit that the stress induced by childhood trauma, particularly multiple traumas, seems to predispose many survivors to seek out HRBs to cope or to numb symptoms (Felitti et al., 1998; Center for Substance Abuse Treatment, 2014), this does not parse out contributions from the neighborhood environment, a potentially significant source of stress. In neighborhoods with higher levels of deprivation, this greater exposure to chronic forms of inequity likely explains the greater
prevalence of health risks, and may also challenge the ability to buffer its impacts, contributing to higher likelihood of engaging in HRBs to cope (Bethell, 2017).

Also encapsulated within this premise is that recent studies indicate racial/ethnic minorities have disproportionately higher likelihood for experiencing multiple ACEs, increasing the likelihood they will develop health problems, as well as HRBs (Goldstein et al., 2020; APA, 2017, CDC, 2019). Not surprisingly, these groups are also more likely to live in areas of higher neighborhood deprivation because of structurally oppressive factors, including segregation and racial discrimination (Cox, Tice, & Long, 2019). Thus, with exploration of neighborhood deprivation and race/ethnicity in tandem, we broach the subject of how inequity, racism, and social processes function in exposure to multiple ACEs and its outcomes (Cole, 2009) and may create greater likelihood for stress embodiment processes, such as HRBs (Steptoe, 2019; Daftary, 2018). This necessitates exploration of neighborhood inequity on the causal pathway between multiple ACEs and development of HRBs, as well as exploring how race may moderate this relationship.

Another important contribution of this study is that social support plays an important role in moderating the influence of ACEs on health outcomes and can act as a protective resource. This study utilizes PSS, as research shows that a person’s perception of the supports they have available is a better indicator of coping and adaptation to stress than the “actual support an individual receives” (Simon et al., 2019, p. 2). It is well established PSS has significant importance following any type of stress exposure, as it can protect against psychological and physiological impairment (Thoits, 2011). For example, following trauma exposure, PSS is particularly critical in buffering the development of trauma-related symptoms, such as PTSD (Lee, 2019; Simon et al., 2019),
and the absence of PSS is one of the biggest risk factors for the chronicity of trauma symptoms (Simon et al., 2019). Social support also appears to have a relationship with the likelihood of developing alcohol and drug use problems, with reduced likelihood among those with higher self-rated support (Stockdale et al., 2007). Because health research continues to show social relationships have causal influence on mortality and overall health, including HRBs, it is important to investigate how PSS interacts with multiple ACE exposures to understand if it has buffering potential, as well as how it interrelates with neighborhood deprivation (Thoits, 2011; Wang et al., 2018).

A final significance of this study is that both neighborhood context and PSS may illuminate mechanisms contributing to positive health results. A critique of ACEs research involves the underlying assumption that people with multiple ACEs are destined for health problems, including engaging in higher risk behaviors. This assumption prevents us from examining if individuals with similar levels of exposure can have different outcomes (Steptoe, 2019). Because of the overall emphasis on risk assessment in ACEs research, there are still gaps in our understanding of the protective factors buffering the effects of ACEs. Thus, among those who do not report engaging in HRBs, it is important to explore if lower neighborhood deprivation and/or higher perceived social support might explain these outcomes even with exposure to multiple ACEs. Research devoted to identifying factors that contribute to health risk behaviors among those with multiple ACE exposures may help expose protective factors useful in intervention efforts and policy changes (Steptoe, 2019).

The findings in the present study are based on secondary data from the 2010 Washington State Behavioral Risk Factor Surveillance System (BRFSS), a cross-
sectional study (Washington State Department of Health, 2011). The BRFSS is a nationwide telephonic survey of adults aged 18 years or older that examines self-reported health status, health knowledge, preventative health behavior, chronic conditions, ACEs, and HRBs in various states depending on the year. This study also merges data from the Neighborhood Deprivation Index (NDI) with BRFSS data by using each participant’s zip code to assign an NDI value. The NDI was created using the 2013-2017 American Community Survey (ACS) data and is a 13-measure index quantifying deprivation for every census tract throughout the 50 states and Washington D.C. (“Neighborhood Deprivation Index Data,” 2020). The purpose of this study is to: (1) examine how exposure to multiple ACEs contributes to the development of health risk behaviors (smoking, binge/heavy drinking, physical inactivity, drinking and driving, drug use, and risky HIV behavior) (2) identify whether the association between exposure to multiple ACEs and resulting HRBs is explained or mediated by level of neighborhood deprivation (3) explore whether the relationship between exposure to child trauma and resulting HRBs is moderated by PSS, and (4) due to increased exposure to multiple ACEs among BIPOC individuals, examine if race/ethnicity moderates the relationship between exposure to multiple ACEs and HRBs, resulting in higher HRBs.
Chapter 2: 

Literature Review

Theoretical Orientation

While it is difficult to find critiques of the prevailing biomedical models for ACE-related morbidity, which use a combination of biobehavioral explanations, allostatic load, and epigenetics to demonstrate how ACEs “exert their influence” on adulthood health (Felitti et al., 1998; Campbell et al., 2016, p. 350), there are gaps in their explanatory power. Notably, biomedical models focus on individual-level contributions without accounting for community or macro-level processes that influence stress and health trajectories (Bowleg & Bauer, 2016), presumably critical influences in the interplay of biology and trauma (Pathak & Nichter, 2015; Krieger, 2001). The application of ecosocial theory has the potential to address these gaps in knowledge. Developed in 1994 by Nancy Krieger, a social epidemiologist, ecosocial theory integrates biological and social models of disease distribution and exposure to understand the complex interplay between how social inequities, in combination with other factors, can become biologically embodied and generate disease (Krieger, 2001). Ecosocial theory explains that the processes happening around an individual involve a complex interrelationship with an individual’s body, wherein the environment interacts with the body and the body with the environment in a connected, dynamic way. These social and biological components cannot be understood separately because it is their combination that results in impact (Krieger, 2001). With ACEs, this aspect accounts for both the biological stress processes induced by trauma and the compounding or synergistic influence of stressful environmental, racial and structural processes from neighborhood deprivation.
There are four interconnected parts to ecosocial theory that explicate the process of disease development and progression: embodiment; pathways to embodiment; the multilevel interaction of exposure, susceptibility, and resistance; and agency and accountability (Krieger, 2001, p. 672). Each component acts as a window for understanding some of the factors lacking in the biomedical frameworks, while also creating the possibility for improving understanding and developing intervention (Krieger, 2001). For example, “pathways to embodiment” reveals how simultaneous aspects of economic deprivation, discrimination, barriers to opportunities, and access to resources – occurring at multiple levels and across time – can compound the impacts of trauma, generating a greater likelihood for health problems (Krieger, 2001; Pathak & Nichter, 2015). Thus, the social distribution of ACE-related health problems, which are disproportionately represented in minorities and those in poverty, is seen as a “biological expression” of these inequities (Krieger, 2001, p. 672). Through this, we realize we cannot understand one’s biological processes – and, resultantly, disease development and progression – in isolation from the simultaneous processes of power, property, history, society, and an individual’s circumstances (Krieger, 2001).

Importantly, in accounting for the socio-structural bases of health that are overlooked in current biomedical explanations, biological and stress processes are contextualized. This is important particularly for HRBs, since the biomedical model can activate biases and blame toward individuals who develop these behaviors (Krieger, 2001). These ideas “emphasize individuals’ responsibility to ‘choose’ so-called ‘healthy lifestyles’ and to cope better with ‘stress’” instead of examining “economic and political determinants of health and disease, including structural barriers to people living healthy
lives” (Krieger, 2001, p. 670). This also explains how societies contribute to the exposure and generation of disease such as ACEs through the way disease – or health behavior – is conceptualized, ignoring some facets, such as oppression, while emphasizing others, such as individual differences in biological processes (Krieger, 2001). Widening the scope of impact to integrate the layered influences on an individual’s entire life course shifts the paradigm from a deterministic model to one that is modifiable (Buffardi et al., 2008), enabling more holistic interventions and preventative action at the community or societal level, in addition to the individual level (Bruner, 2017). From this theoretical orientation, this study explores the impact of social inequities in combination with biological components to understand variance in toxicity and pathways contributing to HRBs (Krieger, 2001). By working with these frameworks in tandem, we begin to understand the multiple interlocking pieces contributing to health disparities from ACEs and have a more nuanced framework that creates room for individual agency, such as the buffering or protective factors from PSS (Krieger, 2001).

Coupled with ecosocial theory, this study also utilizes a critical inquiry paradigm, which provides an important “interpretive framework” for ACEs research and aligns with many social work principles (Daftary, 2018, p. 10). This paradigm includes an explicit social justice orientation, an emphasis on promoting the voices of those who have been historically silenced and oppressed, understanding the pervasiveness of systems of privilege, and applying intersectional thinking (Daftary, 2018). Further, foregrounding ACEs research within a critical paradigm prevents reinscribing dominant narratives within ACEs risk-assessment by reconceptualizing how findings are framed:
The use of ‘vulnerable’ and ‘at risk’ decontextualizes the social determinants of health from broader public policy approaches and structural inequalities. Naming groups or populations as vulnerable and/or marginalized without acknowledgement of the structural conditions that contributed to inequitable health outcomes in the first place has the potential to further marginalize and stigmatize populations (Pauly et al., 2017, p. 506).

Thus, a critical paradigm illuminates how excluding discussion of socio-structural factors indirectly pathologizes inequity, discrimination, and poverty relative to neighborhood deprivation, and instead clarifies the pervasiveness of systems of privilege and how they perpetuate and maintain disparities (Steptoe, 2019). Importantly, this then shifts conceptualization of HRBs to being an “adaptation” to the embedded presence of toxic stress from multiple social and physiological disruptions versus individual pathology (Goldstein et al., 2020; Strine et al., 2012). Finally, a critical paradigm also operationalizes the action piece of research in being explicit about using research to create social change and challenge inequity (Steptoe, 2019).

**Multiple ACEs and Health Risk Behaviors**

Adverse childhood experiences refer to 8 types of childhood trauma occurring before the age of 18. ACEs criteria encompass three categories of child abuse, including physical, verbal, and sexual abuse, and five categories of household trauma, including divorce, domestic violence, living with someone who has been incarcerated, living with someone who has a drug or alcohol problem, and living with someone who has a mental illness. Cumulative exposure to ACEs is predictive of numerous adult health risks, including increased engagement in HRBs, increased morbidity and comorbidity, greater
prevalence of mental illness, suicide, injury and disability, as well as early mortality (Felitti et al., 1998; Campbell et al., 2016; Brown et al., 2009). ACEs research has shown childhood trauma is relatively common throughout the United States (Campbell et al., 2016; Felitti et al., 1998), and an individual who experiences at least one ACE is anywhere from 65% to 93% more likely to experience a second form of trauma (Felitti et al., 1998).

Research indicates there is a “threshold effect” at the accumulation of four or more types of childhood trauma, as it seems to induce substantially greater odds for all ACE-related health problems and mortality (Felitti et al., 1998; Brown et al., 2009; Campbell et al., 2016; CDC, 2019; Schüssler-Fiorenza Rose et al., 2016, p. 11). Additionally, those with multiple exposures to childhood trauma may experience significant social effects as well, such as chronic unemployment, limited educational attainment, financial struggles, and even houselessness (Font & Maguire-Jack, 2016; Topitzes et al., 2016). Yet, much of ACEs research focuses on generalized exposure with little emphasis on those with multiple trauma exposures. In a meta-analysis of odds associated with multiple types of childhood trauma, estimates indicate individuals with multiple ACEs are anywhere from 1.6 to 12.2 times more likely to initiate HRBs compared to those with no ACEs, depending on the HRB (Hughes et al., 2013; Campbell et al., 2016; Felitti et al., 1998). While those with four or more types of ACE exposures represent approximately 16% of the population (CDC, 2019), their differential odds place an undue likelihood for all detrimental health outcomes, with the majority of these individuals engaging in some form of HRB (Felitti et al., 1998). As a result, researchers
have indicated the need for identifying the mechanisms contributing to these worsened outcomes.

Because the stress associated with ACEs can affect several processing components in the brain, those with childhood trauma may feel an increased sense of loneliness, hypervigilence, emotional dysregulation, increased reactivity, disrupted interpersonal functioning, such as aggressiveness, and disrupted learning and memory (Herzog & Schmahl, 2018, p. 3; Goldstein et al., 2020). These impacts appear to worsen with multiple exposures to trauma and can lead to a heightened perception of stress, intensifying symptoms and “shaping subsequent behavioral responses,” such as HRBs (Jones et al., 2018, p. 37). Often referred to as biobehavioral explanations, researchers determined health risk behaviors like smoking or drinking are often initiated as a coping device due to their pharmacologic impact on the brain, such as improving mood and providing temporary stress relief (Felitti et al., 1998; Hughes et al., 2017; Campbell et al. 2016).

Health risk behaviors, in combination with stress response systems and epigenetic mechanisms, appear to be an important contributor to long-term health problems and early mortality among those with childhood trauma (Felitti et al., 1998). Research indicates there is a dose response relationship between childhood trauma and adult health behaviors, such that as exposure to trauma increases so does the frequency of developing these behaviors. For example, those with a higher number of ACEs are more likely to smoke in adolescence and are also more likely to smoke chronically in adulthood, contributing to the likelihood of diseases like emphysema and cancer (Felitti et al., 1998, p. 254).
Neighborhood Deprivation and Perceived Social Support

While we know individuals affected by four or more types of adverse childhood trauma have substantially greater odds of developing health risk behaviors, very little is known about the mechanisms influencing their development. However, the interrelation between neighborhood deprivation and social support resources provides promising insight, while also addressing some of the gaps in current explanations. Notably, biobehavioral explanations do not account for factors related to one’s physical and wider structural environmental context in the development of HRBs, such as neighborhood deprivation, which often involves chronic stressors.

As mentioned previously, neighborhood deprivation is the distillation of various forms of inequity (Messer et al., 2006). To assess deprivation, the Neighborhood Deprivation Index (NDI) provides a deprivation value, 1 through 5 (with 1 being least deprivation and 5 being most deprivation), based on 13 indicators, including disparities in income, wealth, employment, education, occupational status, home ownership, and housing conditions, such as insufficient plumbing (Messer et al., 2006; “Neighborhood Deprivation Index Data,” 2020). Ongoing public health research supports evidence of these neighborhood-level forces on a multitude of health outcomes, including cardiovascular health, premature birth, cancer, as well as specific health behaviors, like gambling and drug use during pregnancy (Groos et al., 2018; Messer et al., 2006). Both early life adversities, such as ACEs, and neighborhood deprivation are linked with ongoing, cumulative life adversities, “in a successively contingent manner, creating a stress trajectory or pathway,” much like a chain reaction (Wickrama et al., 2015, p. 1110; Jones et al., 2018, p. 33). Thus, this toxic “stress trajectory” leads to biologic stress
responses that not only hinder adaptive coping (Jones et al., 2018), leading to HRBs, but also increases the risk of many health problems (Wickrama et al., 2015).

Application of the NDI reveals how, “...class, status, and party (or power), contemporarily operationalized as occupation, education, and income, are differentially distributed and may influence opportunities for health and well-being” (Messer et al., 2006, p. 1056). Accordingly, we cannot fully understand the ACEs trajectory without inclusion of the neighborhood context, as it exposes the functioning of deeply embedded social and political processes and reduced opportunities for health (Strompolis et al., 2019; Daftary, 2019; Lee & Chen, 2018; Cox, Tice, & Long, 2019). This geographic measure of deprivation acts as a proxy for various social inequities and helps identify the role of collective disparity (Groos et al., 2018). For example, neighborhoods with a higher deprivation index are more likely to experience community disinvestment typically associated with conditions like substandard housing, higher unemployment, segregation, a higher number of people on public assistance, reduced access to health clinics, and lacking educational opportunities from poorly funded schools (Cox, Tice, & Long, 2019).

Deprivation factors can directly shape health behavior as well. For instance, research shows that those living in high deprivation environments are less likely to spend leisure time exercising in their neighborhood due to safety concerns and having less green space, thereby increasing the likelihood of inactivity (Pampel et al., 2010). Not surprisingly, factors measured by the NDI are known to have “profoundly compromising” influence on one’s health trajectory (Bruner, 2017, p. S126). We must
understand if the accumulation and toxicity of childhood trauma operates through these socioeconomic factors to better explain ACE-related health behaviors.

Recent ACE studies provide evidence that certain identifiers of SES, such as living below the poverty line, education level, and health care access, play an important role in mediating the relationship between childhood adversity and overall health. In a 2018 study by Jones et al., experiencing a difficult event in adulthood (such as incarceration, sexual assault, or caregiver burden), having low income status, as well as weak social support all mediated the relationship between exposure to ACEs and the development of mental illness. In another ACEs study, researchers demonstrated how adult SES (measured by income, health insurance status, marital status, and education level) plays a mediating role in numerous ACE-related health outcomes. They found that people who experience higher ACEs tend to be more likely to experience poverty in adulthood as well as experience other factors related to poverty, including being less likely to graduate from high school and less likely to be married (Font & Maguire-Jack, 2016). Thus, the impact of exposure to multiple adverse childhood experiences on the prevalence and severity of later health risk behaviors appears to be partially driven by socioeconomic status. As Jones et al. explain, “While income is a strong determinant of physical and mental health, its effects are largely indirect, operating through differential exposure to adverse conditions” (2018, p. 33).

Based on the findings of these studies, which demonstrate the significant, mediating role of socioeconomic factors following exposure to ACEs, particularly multiple ACEs (Font & Maguire-Jack, 2016; Jones et al., 2018), as well as studies demonstrating the correlation between poverty and worsened health outcomes (Messer et
al., 2006; Pampel et al., 2010; Font & Maguire-Jack, 2016), neighborhood deprivation is specifically examined as a mediator in this study. Using the NDI improves on this research by accounting for the “constellation” of factors comprising deprivation and inequity on the pathway to adult HRB outcomes (Messer et al., 2006, p. 1057). Study findings emphasize that the dimensions of deprivation within the NDI collectively and comprehensively encapsulate “diverse underlying theoretical concerns,” including things like “material well-being, prestige, and human capital,” which is more powerful than a single indicator of SES (Pampel et al., 2010, p. 350).

In addition to better understanding how one’s neighborhood environment can create toxic stress, understanding the impact of resources on one’s health is also important. Social support is a measurable resource with profound impact that may play a role in “interrupting” stress pathways (Wickrama, 2015; Thoits, 2011). Decades of research indicate social support is a critical protective component in buffering the development of trauma-related psychological symptoms and stressful life events and has important impact on overall well-being (Thoits, 2011). However, exposure to childhood adversity may “significantly” disrupt social support due to both the secrecy with which abuse often occurs and the fact that abuse typically involves the violation of a significant caregiver or familial relationship, which can diminish support-seeking following trauma (Center for Substance Abuse Treatment, 2014, p. 74). Additionally, while some research seems to indicate social support may be limited in high deprivation environments due to the pervasive number of stressors (Jones et al., 2018; Lee, 2019; Bruner, 2017), neighborhood deprivation does not preclude social support nor does a more affluent neighborhood guarantee it.
Recent ACEs studies back the idea that social support, such as PSS, plays an important role in the relationship between childhood adversity and overall health. In a study examining children with four or more ACEs, emotional support, in combination with two other forms of social support, reduced the prevalence of poor health by two-thirds (Bellis et al., 2018). Because good social support improves trauma-related health, while low social support explains the development of trauma-related symptoms and diminished coping ability, it follows that PSS may play an integral role in decreasing the likelihood of developing HRBs among those with multiple ACEs. Additionally, if low PSS and high NDI occur in concert, these factors together may help explain reduced likelihood of adaptive coping behaviors following ACEs.

Although resiliency research is common in ACEs studies with child populations, there is little research devoted to identifying what explains the absence of symptoms – and reducing the likelihood of developing HRBs – among adults with multiple ACEs. Many childhood ACEs studies and models show the importance of social connection, supportive relationships such as mentors or a trusted adult, and acquiring social and emotional skills to both improve and prevent the development of ACE-related health problems as the child ages (Bellis et al., 2018; Sege & Harper Browne, 2017). Since these factors comprise social support, and a large body of research shows the positive, buffering effect social support provides in preventing the development of trauma-related symptoms, good PSS could reduce the likelihood of developing HRBs, explaining more positive outcomes among those with multiple exposures (Jones et al., 2018).

As of evidence of this, certain social psychological factors associated with one’s social ties, such as social control and social influence, have proven helpful in explaining
engagement with health-promoting behaviors, though this depends on the person’s reference group (Thoits, 2011). Further, in accounting for the provision of social capital regardless of circumstance, good PSS may also work to buffer the harmful effects of structural inequities (Larkin, Felitti & Anda, 2014). While some research seems to indicate individual-level protective factors like social support do not buffer neighborhood-level stressors, such as witnessing a violent attack, this relationship must be examined with neighborhood-level SES stressors, such as deprivation (Stockdale et al., 2007). Similarly, there are numerous environmental factors that may be more conducive to adaptive coping because of lower stress and greater resources, including lower violence and crime, quality housing, and greater supply of living wage jobs. Thus, environmental factors characterizing lower neighborhood deprivation may offer stress reduction and a lower likelihood of engaging in HRBs.

**Social Work Implications**

This study challenges a typical deficits-based or “risk assessment” orientation toward a conceptualization of ACEs that is more holistic. Social work practice emphasizes a biopsychosocial approach in order to “simultaneously perceive the individual and the collective,” theorizing that the best way to address wellness is by attending to the whole person, including their community and available resources (Larkin, Felitti & Anda, 2014, p. 2). This situates a person’s life course within a systems context in order to understand the complex comingling of factors impacting health and the production of disease. A biopsychosocial orientation helps explain how one’s social environment in combination with biology, mental health and social networks, are all critical for healthy development, and that ACEs can impinge development by impacting
stress and coping systems (Larkin, Felitti & Anda, 2014). In a neighborhood with many forms of inequity, childhood trauma can lead to HRBs as an adaptation to stressful internal and external processes. Accordingly, this study reflects the next phase of ACEs research examining the unique interplay between internal and socio-environmental processes and how this influences ACE-related disparities (Larkin, Felitti & Anda, 2014).

In the same vein, this study solidifies social work’s role at the forefront of ACEs research by contextualizing the unique variances in ACE-related pathways depending on social and community factors. One of the biggest criticisms of ACEs is that the composite ACE score assumes universal interpretation and usefulness (Steptoe, 2019). The summation of types of adverse experiences is supposed to directly translate to inherent risk for various health, behavioral, and mortality outcomes. However, what is glaringly absent from this score is context: where one lives, one’s experiences of discrimination and oppression, one’s access to resources, etc. Tapping into social work’s unique commitment to examining the full scope of possible contributions to adversity, as well as resources, (Larkin, Felitti & Anda, 2014) renders visible the “multiple, interactive levels” that individuals are placed within (family, neighborhood, community, state, federal policies), and how these levels each reflect various resources or challenges depending on a variety of factors (Melton-Fant, 2019, p. 1). Social work has the objective to expose and promote action around the omnipresence of disparities, such as health inequities, including contextualizing inequities in terms of social, structural, and political processes and aiming to eliminate systems perpetuating them (Daftary, 2018). By examining how the intersection of access, discrimination, and systems of privilege impacts the toxicity of child adversity – investigated through both NDI and the
racial/ethnic moderator – this study emphasizes an approach to ACEs research that includes socio-structural impacts and developing methods to adequately address this variance in outcomes.

In tracking health inequities throughout the life course we see how many forms of systemic oppression combine to influence health starting in childhood. Therefore ACEs research practices that aim to illuminate contributions from poverty, racism, oppression, etc. that are encapsulated within neighborhood deprivation and the racial/ethnic moderator can contribute to policies that then work to “dismantle” these processes (Bowleg, 2017, p. 679). As Hughes et al. explain, “Sustained prevention gains might require a shift in focus to include the early drivers of poor health. Policies that capture the environmental and societal causes of adversity in childhood offer new opportunities to address ACEs rather than just their consequences” (2017, p. e363).

Consequently, establishment of pathways will enable guidance for the development of efficacious policies and social work practices to reduce childhood trauma’s “contribution” to national health problems and the Global Burden of Disease (Larkin, Felitti & Anda, 2014; Hughes et al., 2017, p. e363). ACEs contribute to 5 of the ten major mortality-related health problems in the United States (CDC, 2019), and the national costs associated with ACE-related health problems are currently estimated to be around $124 billion (CDC, 2017). While the ultimate objective is to eradicate childhood trauma, first we must better understand the mechanisms contributing to negative adult physical and mental health outcomes, such as the development of HRBs (Hughes et al., 2017). In turn, these findings can inform and enhance legislation and social programs to
reduce or intervene upon HRBs through both individual and community assets, which will have greater impact (Bellis et al., 2018).

For example, creating policies to focus on the eradication of poverty, such as through improving pay equity and instituting a federal living wage, as well as policies devoted to eliminating community disinvestment that could improve education and housing conditions may reduce neighborhood deprivation and related indicators (Cox, Tice, & Long, 2019). Policies and institutional practices aimed at eliminating systemic oppression will also be integral to this objective. Further, regardless of level of ACE exposure, social work can have an instrumental role in bolstering social support resources within communities through asset-based community development (ABCD), which enhances assets already existing within a neighborhood context (Bellis et al., 2018). Examples of ABCD include identifying community support figures, enhancing cultural connection, and creating opportunities for friendship and networking, which are all resiliency factors proven helpful in improving ACE-related health outcomes in child cohorts and may be equally beneficial to adults (Bellis et al., 2018). Also, the creation of community mentoring interventions that work to increase self-efficacy and accountability, factors essential to improving health-related behavior in adults with ACEs, may be effective interventions for preventing or reducing HRBs (Goldstein et al., 2020).
Chapter 3:

Methods

The purpose of this study is to: (1) examine how exposure to multiple ACEs contributes to the development of health risk behaviors (smoking, binge/heavy drinking, physical inactivity, drinking and driving, drug use, and risky HIV behavior) (2) identify whether the association between exposure to multiple ACEs and resulting HRBs is explained or mediated by level of neighborhood deprivation (3) explore whether the relationship between exposure to ACEs and resulting HRBs is moderated by PSS, and (4) due to increased exposure to multiple ACEs among BIPOC individuals, examine if race/ethnicity moderates the relationship between exposure to multiple ACEs and HRBs, resulting in higher HRBs.

Study Dataset

This study utilizes the 2010 Behavioral Risk Factor Surveillance System (BRFSS) data from Washington State, a population-based, cross-sectional study (Washington State Department of Health, 2011). The BRFSS is a nationwide random-digit dialing (RDD) telephonic survey of non-institutionalized adults ages 18 years or older (Ford et al., 2014). The BRFSS is conducted in rotating cycles in all 50 states at different points in time, and the number of adults surveyed in each geographic region depends on the size of the area, as well as available financial backing. Each year, approximately 400,000 interviews are conducted; participants do not receive any sort of incentive for participating in the BRFSS (CDC, 2018).

The BRFSS standardized protocol, available in both English and Spanish, investigates self-reported health status, health knowledge, preventive behaviors, chronic
conditions, and HRBs (CDC, 2018). The BRFSS has core interview questions administered in every state participating in a given year, and states also have the option to add state-relevant questions based on particular health needs (Purnell et al., 2012; CDC, 2018). Because only certain states complete the adverse childhood experiences module in a given year, this study uses the BRFSS survey data collected in 2010 when Washington State added the ACEs questionnaire (Washington State Department of Health, 2011). This State’s health-related data is used for the analysis in this study.

**Measures**

*Predictor: Adverse Childhood Experiences*

The Adverse Childhood Experiences (ACEs) survey consists of 11 questions that assess exposure to *three* types of child abuse, including physical, sexual, and emotional abuse, and *five* types of household dysfunction, including divorce, domestic violence, living with someone who was incarcerated, living with someone who has a drug or alcohol problem, and living with someone who has a mental illness, all occurring before the age of 18. Adhering to the CDC’s recommended data analysis approach for the ACEs measure, the three sexual abuse-related questions are collapsed to one, where a “yes” response to any of the three indicators reflects endorsement of sexual abuse (CDC, 2019; Schüssler-Fiorenza Rose et al., 2014; Font & Maguire-Jack, 2016, p. 392). Similarly, the two questions devoted to the assessment of living with a person with a substance problem are also collapsed to one, where a “yes” response to either of the two indicators reflects endorsement of living with a person with a substance problem. Accordingly, 8 categories of abuse and household dysfunction are created, and a cumulative exposure score ranging from 0-8 is calculated (Anda et al., 2010). Respondents without any ACE exposures are
excluded from the analysis because this study is examining the factors through which ACE exposure contributes to HRBs.

Because ACEs demonstrate strong positive skewness when kept as a categorical predictor (due to a much smaller percentage of people having multiple exposures, particularly in the higher ranges), and because this study is specifically interested in accumulated trauma beyond 3 ACEs versus individual ACEs – or the “threshold effect” at 4 or more ACES – ACE respondents are grouped into two categories: those with 4 or more ACEs, and those with 1-3 ACEs, and a dichotomized variable of multiple ACEs is entered for data analysis (Schüssler-Fiorenza Rose et al., 2016, p. 11). This matches the methodology of previous ACE-related health research studies focusing on multiple exposures (Schüssler-Fiorenza Rose et al., 2016; Hughes et al., 2018). Researchers found that when analyzing exposure, “there was not a significant difference” among 3 or fewer ACE exposures but “there were wide and overlapping confidence intervals (due to smaller numbers) at higher levels of ACEs,” creating the need for a “dichotomized ACE Score into 4 or more/less than 4 categories for analyses examining the effect of health and contextual factors” (Schüssler-Fiorenza Rose et al., 2016, p. 6-7).

The ACEs scale is a validated measure (Anda et al., 2010). It demonstrates high internal consistency with a Cronbach’s Alpha of 0.78 and also shows strong test-retest reliability (Ford et al., 2014; Campbell et al., 2016). Currently, 42 states have included the BRFSS ACEs survey as a state-added portion, but participating states vary annually.

**Outcome: Health Risk Behaviors**

The outcome variable, health risk behaviors (HRBs), consists of 7 self-reported items assessing problematic alcohol use, painkiller use to get high, marijuana use,
smoking status, drinking and driving, physical inactivity, and risky HIV behaviors. These items are all part of the core BRFSS interview protocol and are included in every data collection cycle.

One question is used to determine heavy/binge drinking: “Considering all types of alcoholic beverages, how many times during the past 30 days did you have X (X = 4 for men and X = 5 for women) or more drinks on an occasion?” Responses are dichotomized as 0 indicating no episodes of binge drinking and 1 indicating 1 or more episodes of binge drinking.

One question is used to determine painkiller use to get high: “During the past 30 days, on how many days did you use a painkiller to get high, like Vicodin, Oxycontin (sometimes called Oxy or OC), or Percocet (sometimes called Percs)?” Responses are dichotomized as 0 indicating no use of painkillers to get high and 1 indicating 1 or more days of using painkillers to get high.

Similarly, one question is used to determine marijuana use: “During the past 30 days, on how many days did you use marijuana or hashish (grass, hash, or pot)?” Responses are dichotomized as 0 indicating no use of marijuana and 1 indicating 1 or more days of using marijuana.

The BRFSS smoking section includes two questions to assess current smoking status: “Have you smoked at least 100 cigarettes in your entire life?” and “Do you now smoke cigarettes every day, some days, or not at all?” Respondents who answer yes to smoking 100 cigarettes and also smoke every day or some days are categorized as current smokers, dichotomized as 1. Those who have not smoked 100 cigarettes and do not smoke at all are categorized as nonsmokers (Purnell et al., 2012).
The drinking and driving BRFSS section includes one question: “During the past 30 days, how many times have you driven when you’ve had perhaps too much to drink?” Respondents who report 1 or more times are categorized as engaging in drinking and driving.

For the BRFSS exercise module, respondents are asked, “During the past month, other than your regular job, did you participate in any physical activities or exercises such as running, calisthenics, golf, gardening, or walking for exercise?” Responses are dichotomized as 0 for people answering “yes” to physical activity and 1 for people answering “no” and are physically inactive.

BRFSS standardized protocol explores risky HIV behaviors by providing a list of high risk HIV-related behaviors, such as injecting non-prescribed intravenous drugs or buying or receiving money for sex, and asking participants to indicate if any of the scenarios apply to them in the last year without disclosing specifically which apply (CDC, 2018). Respondents who endorse any of the scenarios are coded as engaging in high-risk HIV behavior, dummy coded 1.

**Mediator: Neighborhood Deprivation**

Neighborhood deprivation is utilized as a mediator in this study based on previous research demonstrating the association between exposure to ACEs and resulting adulthood SES indicators, as well as evidence of the mediating effect of certain SES factors (Font & Maguire-Jack, 2016; Jones et al., 2018). To assess neighborhood deprivation, this study merges the Neighborhood Deprivation Index (NDI) table with BRFSS data. Several researchers created the NDI by using the 2013-2017 American Community Survey (ACS) data, and it is a 13-measure index quantifying deprivation for
every census tract throughout the 50 states and Washington D.C. (“Neighborhood Deprivation Index Data,” 2020). The data table is publicly available online through the NIH research resources (NIH, 2021). The index captures the collection of factors indicative of deprivation, including:

1. Occupation (percent of households in management, business, science, or arts occupations; percent unemployed)
2. Wealth and income (percent of households with incomes below the poverty level; percent of households receiving dividends/interest/rental income; median household income; median home value; percent of households receiving public assistance)
3. Housing conditions (percent of female-headed households with children under 18; percent of housing units that are owner occupied; percent of households without a telephone; percent of households without complete plumbing facilities)
4. Education level (percent with a high school degree or higher; percent with a college degree or higher) (“Neighborhood Deprivation Index Data,” 2020).

The higher a given area’s deprivation level, the higher the value on the index, ranging from -2.5 to 1.9. These values are then categorized into quintiles with 1 = “least deprivation” to 5 = “most deprivation.” The NDI demonstrates high internal consistency with a Cronbach’s Alpha of 0.91 (Stoddard, 2013). Participant zip code is used to match the corresponding NDI value with each participant’s BRFSS data. Census tracts are converted to zip code using the U.S. Department of Housing & Development’s conversion chart (U.S. Department of Housing & Development, 2019). In the instance there are multiple census tracts within the same zip code and differing NDI values, the
mode NDI value is taken. The ICC (intra-class correlation) is used to ensure HRBs are not explained by the grouping variable.

**Moderators: Perceived Social Support and Being a Person of Color**

For the first moderator, this study assesses perceived social support (PSS) through the Emotional and Social Support module, a 1-question measure from the core protocol. This asks respondents, “How often do you get the social and emotional support you need?” with support from any source, including formal (caregiver, church, therapist, etc.) and informal (friend, partner, family, etc.). Response options include Always, Usually, Sometimes, Rarely, and Never. Responses are coded into three categories: 1 = always or usually receiving support, 2 = sometimes receiving support, and 3 = rarely or never receiving support. Despite this being a one-indicator measure, numerous studies have demonstrated its utility in ACEs research and beyond (Kobau et al., 2013; Brinker & Cheruvu, 2016; Edwards et al., 2016; Schüssler-Fiorenza Rose et al., 2016; Willet et al., 2015).

For the second moderator, the BRFSS demographic section provides information on the race/ethnicity variable (CDC, 2019). This moderator is dummy coded and categorized into the following groups: two categories of race/ethnicity, BIPOC or non-BIPOC. Previous research indicates this variable has associations with both ACEs and NDI and will be explored to see if it has moderating effects on the development of HRBs.

**Control Variables**

The BRFSS demographic section also provides information on relevant control variables. These include age (in years, 18 – 64); sex (male or female); and race/ethnicity (BIPOC or non-BIPOC) with the exception of the BIPOC moderation analysis model.
These control variables were chosen based on previous ACEs research, theoretical perspective, as well as improved model fit/explanatory power.

**Data Analysis Plan**

*Structural Equation Modeling Analysis*

In order to test confirmatory associations between variables of interest, this study uses structural equation modeling (SEM) in Amos (Kline, 2011). Prior to the analyses, it is critical to appropriately handle missing data (Byrne, 2016). Variables must be checked in SPSS to see if the missingness is systematic or “ignorable” through a missing values analysis and Little’s MCAR test (Kline, 2011, p. 55; Kim, 2021c). If data are missing at random (MAR) or missing completely at random (MCAR) and a relatively small subset of each variable (i.e. approximately less than 5%), this study uses single value imputation, or imputing the average score of the missing variable (Kline, 2011). While Amos offers FIML (full information maximum likelihood) to handle missing data, this option is not preferable for the current investigation because it does not allow bootstrapping to test the effect of the mediator, indices of multivariate normality, or modification indices to improve model fit (Byrne, 2016; Kim, 2021b, p. 12).

SEM has numerous benefits including the ability to test hypothesized associations between variables based on theory (Kline, 2011; Anson et al., 2013). Additionally, SEM provides more accurate effect sizes than other statistical procedures, and due to this study’s large sample size – which is beneficial for more complex models – results are expected to be “reasonably stable” (Kline, 2011, p. 12). SEM assesses covariance between variables with the objective of attempting to explain the highest amount of
variance in the model (Byrne, 2016; Kline, 2011). For the SEM analysis, the following steps are addressed:

1. **Specification.** As Kline (2011) describes, specification is the creation of a structural equation model that hypothesizes the relationship between variables, which defines the parameters. In this study’s hypothesized models, the exogenous variables include all control variables (race/ethnicity, sex, age), moderators, and Multiple ACEs while the endogenous variables are NDI and HRBs. SEM requires that all exogenous variables covary with one another, and that endogenous variables have residual error (Kim, 2021b). For each of the hypothesized models, the number of parameters and observations are calculated, recognizing the number of parameters cannot exceed the number of observations. Degrees of freedom are calculated using the following formula:

\[ df_M = p - q, \]

where \( p \) is observations and \( q \) is the estimated parameters (Kline, 2011).

2. **Identification.** Identification for structural equation models refers to the computer’s ability to come up with a “unique estimate” for each parameter in the model (Kline, 2011, p. 93). Models can be over-identified, just-identified, or under-identified depending on the degrees of freedom (Kim, 2021b). SEM models require over-identification – or that the degrees of freedom are greater than 0 – to calculate the chi-square statistic (Kline, 2011; Kim, 2021b). To achieve an over-identified model, insignificant parameters that are not directly involved in the hypothesis testing can be constrained to 0, such as covariances, or insignificant paths can be deleted (Kim, 2021b).

3. **Estimation.** Maximum likelihood (ML) is the most common SEM measurement for model fit, or “how well the model explains the data,” and is the chosen method to estimate parameters for this study’s hypothesized models (Kline, 2011, p. 93;
Kim, 2021b). The assumptions for ML include multivariate normality and having continuous endogenous variables (Kim, 2021b, p. 19). Additionally, bootstrap resampling with 5,000 resamples and 95% bias corrected confidence intervals are used for all analyses. For the mediation analysis (Figure 1), bootstrap resampling indicates whether the mediation effect is significant and shows indirect effects. For the moderation models, multigroup SEM analyses test for moderation effects, as both the PSS and BIPOC variables are categorical (Figure 2 & Figure 3) (Kim, 2021d).

4. Model Evaluation and Respecification. Multiple goodness-of-fit indices, such as chi-square ($X^2 > .05$), comparative fit index (CFI ≥ .95), and Root mean square error of approximation (RMSEA ≤ .05) are used in concert to evaluate model validity because chi-square tends to be highly influenced by a large sample size (Kline, 2011; Kim, 2021b). With multigroup analysis, Amos does not provide individual goodness-of-fit indices for each group, but rather the “global model fit for the entire multiple group path model” (Kim, 2021, p. 19). Accordingly, if the unconstrained model demonstrates good model fit through goodness-of-fit indices, it can be assumed the hypothesized path model has “suitable” model fit for all groups (Kim, 2021, p. 15). Through the use of modification and model fit indices, as well as examination of path coefficients, the models can be repecified to improve overall fit/explanatory power. However, it is important to note these indices should be interpreted and utilized alongside relevant theory (Kim, 2021c).

The following hypotheses are examined in this study:
Mediation

Hypothesis I. The effect of multiple ACEs on HRBs is mediated by level of NDI, such that the effect of multiple ACEs on HRBs is fully or partially explained with increasing values of NDI (Figure 1).

1A: There is a positive direct effect of multiple ACEs on HRBs.
1B: There is a positive direct effect of multiple ACEs on NDI.
1C: NDI has a positive effect on HRBs, such that as NDI increases so do HRBs.
1D: Multiple ACEs lead to higher NDI, which leads to HRBs.

Figure 1

*Path Model Demonstrating Mediation of NDI on ACEs and HRBs*
Moderation

Hypothesis 2. The effect of Multiple ACEs on HRBs is moderated by level of PSS, such that as level of PSS decreases, resulting HRBs will increase (Figure 2)

2A: High PSS will weaken the effect of multiple exposures to child trauma on resulting health risk behaviors.

2B: Low PSS will strengthen the effect of multiple exposures to child trauma on resulting health risk behaviors.

Figure 2

*Path Model Demonstrating Moderation of PSS on ACEs and HRBs*
Hypothesis 3: In the relationship between multiple ACEs and NDI on HRBs (Figure 3),

3A: Being BIPOC has a positive direct effect on multiple ACEs.

3B: Being BIPOC has a positive direct effect on NDI.

3c: The effect of multiple ACEs on HRBs is moderated by being BIPOC, such that individuals who are BIPOC will demonstrate increased HRBs.

Figure 3

Path Model Demonstrating Moderation of Race/Ethnicity on ACEs and HRBs
Chapter 4:

Results

Preliminary Data Analysis

**Missing Data.** Most of the variables in the present study were not missing data due to the interview structure of the BRFSS survey, where interviewers are directly interviewing respondents, minimizing system-missing data. Little’s MCAR test was run on all variables and was not significant, indicating missing data is not systematic. In the ACEs module, the BRFSS codes a “refused” or “don’t know” response as a “no” response. For the PSS variable, a relatively small portion of respondents reported “don’t know” or “refused” (n = 51). Because of the small proportion of cases (less than 1%), single value imputation was applied (mode = 3 or a response of “usually” receiving PSS). Similarly, the use of painkillers variable demonstrated a very small portion of missing cases (n = 57), as well as the marijuana use variable (n = 27), and single value imputation (mode = 0 or a “no” response for use) was also applied. The physical inactivity variable contained a small number of “don’t know/refused” responses (n = 12) and single value imputation (mode = 0 or a “yes” response for exercising in the last 30 days) was applied. For the BIPOC variable, single value imputation was not appropriate in inferring a respondent’s race, so listwise deletion was applied to respondents who endorsed a “refused” or “don’t know” to racial identity (n = 82). The NDI variable demonstrated a proportion of missing data (n = 333) attributable to some zip codes not having an assigned NDI value in the NDI table (researchers were not able to calculate an NDI for every area). As a result, listwise deletion was applied to the missing cases. Lastly, because the risky HIV behavior question was considered to be an important factor
comprising the HRB variable and was only asked of those younger than 65 years of age, the sample was restricted to individuals aged 18 to 64, eliminating 2,173 respondents. The resulting sample size is \( n = 5,447 \).

**Normality of the Data.** Because most of the variables in the dataset are dichotomous or ordinal level data, graphical observations of the variables through histograms tended to show skewness with positive kurtosis, which is not uncommon for these types of response options (Byrne, 2016). For example, with the HRB variable, most respondents reported no health risk behaviors, creating a positive skew. Similarly, most respondents reported usually or always receiving PSS. However, with SEM, emphasis is on multivariate normality with particular weight on multivariate kurtosis because this most greatly affects tests of variance and covariance and the use of the Maximum Likelihood method for estimation of fit (Byrne, 2016). Amos provides tests to assess for normality, including kurtosis, critical ratio, and Mahalanobis distance. The main mediation path model for this study demonstrates relatively low multivariate kurtosis (multivariate kurtosis = .630, C.R. = 2.373), where normal distribution is reflected in kurtosis values of < 5.00 (Byrne, 2016; Kim, 2021c). Examination of the Mahalanobis distance \( (d^2) \) score helps assess multivariate outliers for individual cases, which indicated no serious departure in value from surrounding cases (Kim, 2021c).

Next, because all variables are observed variables, zero-order correlation analyses help check for issues of collinearity and show significant relationships between variables. All variables in the sample demonstrate tolerance of < .10 and VIF of > 4, signifying collinearity is not an issue (Field, 2009). Tables 1, 2 and 3 show correlations for each of the observed variables in the path model (ACEs, NDI, and HRBs). Table 4 demonstrates
the correlations, means and standard deviations among the observed variables, including both moderators, PSS and BIPOC. All variables are significantly correlated.

Table 1.

Zero-Order Correlation Matrix of Health Risk Behavior Variables

<table>
<thead>
<tr>
<th></th>
<th>1.</th>
<th>2.</th>
<th>3.</th>
<th>4.</th>
<th>5.</th>
<th>6.</th>
<th>7.</th>
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</thead>
<tbody>
<tr>
<td>1. Marijuana Use</td>
<td></td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Binge Drinking</td>
<td>.18***</td>
<td></td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Current Smoker</td>
<td>.16***</td>
<td>.14***</td>
<td></td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Risky HIV Behavior</td>
<td>.11***</td>
<td>.09***</td>
<td>.06***</td>
<td></td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Drinking and Driving</td>
<td>.07***</td>
<td>.28***</td>
<td>.00</td>
<td>.03*</td>
<td></td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>6. Physical Inactivity</td>
<td>.01</td>
<td>-.02</td>
<td>.12***</td>
<td>.00</td>
<td>-.04*</td>
<td></td>
<td>1.00</td>
</tr>
<tr>
<td>7. Used Painkillers to Get High</td>
<td>.16***</td>
<td>.06***</td>
<td>.05***</td>
<td>.09***</td>
<td>.07***</td>
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</table>

*p < 0.05, **p < 0.01, ***p < 0.001

Table 2.

Zero-Order Correlation Matrix of Adverse Childhood Experiences Variables

<table>
<thead>
<tr>
<th></th>
<th>1.</th>
<th>2.</th>
<th>3.</th>
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<th>6.</th>
<th>7.</th>
<th>8.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Sexual Abuse</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Physical Abuse</td>
<td>.16***</td>
<td></td>
<td></td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Emotional Abuse</td>
<td>.09***</td>
<td>.37***</td>
<td></td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Domestic Violence</td>
<td>.11***</td>
<td>.37***</td>
<td>.23***</td>
<td></td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Household Member Substance Use</td>
<td>.05***</td>
<td>.13***</td>
<td>.09***</td>
<td>.22***</td>
<td></td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Household Member Mental Illness</td>
<td>.12***</td>
<td>.14***</td>
<td>.12***</td>
<td>.12***</td>
<td>.16***</td>
<td></td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>7. Household Member Incarceration</td>
<td>.08***</td>
<td>.12***</td>
<td>.07***</td>
<td>.15***</td>
<td>.21**</td>
<td>.10***</td>
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<tr>
<td>8. Parental Separation/Divorce</td>
<td>.01</td>
<td>.08***</td>
<td>-.03*</td>
<td>.19***</td>
<td>.09**</td>
<td>.03*</td>
<td>.11***</td>
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*p < 0.05, **p < 0.01, ***p < 0.001
Table 3.

Zero-Order Correlation Matrix of Neighborhood Deprivation Index Variables

<table>
<thead>
<tr>
<th></th>
<th>1.</th>
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<th>9.</th>
<th>10.</th>
<th>11.</th>
<th>12.</th>
<th>13.</th>
</tr>
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<tbody>
<tr>
<td>1. Percent of Households Receiving Dividends, Interest, Rent Income</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>2. Percent of Households Receiving Public Assistance</td>
<td>-.68**</td>
<td>1.00</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>3. Median Household Income</td>
<td>.73**</td>
<td>-.70**</td>
<td>1.00</td>
<td></td>
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</tr>
<tr>
<td>4. Median Home Value</td>
<td>.74**</td>
<td>-.56**</td>
<td>.80**</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>5. Percent of Households in Management, Business, Science or Arts Occupations</td>
<td>.75**</td>
<td>-.66**</td>
<td>.73**</td>
<td>.79**</td>
<td>1.00</td>
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<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Percent of Female-Headed Households with Kids Under 18</td>
<td>-.56**</td>
<td>.64**</td>
<td>-.43**</td>
<td>-.42**</td>
<td>-.53**</td>
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<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>7. Percent of Housing Units that are Owner Occupied</td>
<td>.47**</td>
<td>-.49**</td>
<td>.53**</td>
<td>.18**</td>
<td>.21**</td>
<td>-.33**</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Percent of Households Without a Phone</td>
<td>-.30**</td>
<td>.35**</td>
<td>-.32**</td>
<td>-.21**</td>
<td>-.23**</td>
<td>.22**</td>
<td>-.30**</td>
<td>1.00</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>9. Percent of Households Without Complete Plumbing Facilities</td>
<td>-.06*</td>
<td>.17**</td>
<td>-.18**</td>
<td>-.07**</td>
<td>-.06*</td>
<td>-.04</td>
<td>-.11**</td>
<td>.20**</td>
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</tr>
<tr>
<td>10. Percent with a High School Degree or More</td>
<td>.58**</td>
<td>-.67**</td>
<td>.51**</td>
<td>.47**</td>
<td>.63**</td>
<td>-.56**</td>
<td>.25**</td>
<td>-.24**</td>
<td>-.09**</td>
<td>1.00</td>
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<tr>
<td>11. Percent with a College Degree or Higher</td>
<td>.74**</td>
<td>-.63**</td>
<td>.71**</td>
<td>.84**</td>
<td>-.92**</td>
<td>-.50**</td>
<td>.09**</td>
<td>-.21**</td>
<td>-.08**</td>
<td>.60**</td>
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<td></td>
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<tr>
<td>12. Percent Below Poverty Level</td>
<td>-.60**</td>
<td>.78**</td>
<td>-.64**</td>
<td>-.46**</td>
<td>-.54**</td>
<td>.62**</td>
<td>-.51**</td>
<td>.35**</td>
<td>.12**</td>
<td>-.61**</td>
<td>-.49**</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>13. Percent Unemployed</td>
<td>-.37**</td>
<td>.47**</td>
<td>-.44**</td>
<td>-.37**</td>
<td>-.40**</td>
<td>.34**</td>
<td>-.28**</td>
<td>.16**</td>
<td>.09**</td>
<td>-.29**</td>
<td>-.37**</td>
<td>.41**</td>
<td>1.00</td>
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</table>

*p < 0.05, **p < 0.01, ***p < 0.001
### Correlations, Means, and Standard Deviations for Observed Variables

<table>
<thead>
<tr>
<th></th>
<th>1.</th>
<th>2.</th>
<th>3.</th>
<th>4.</th>
<th>5.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. ACEs</td>
<td>1.00</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. HRBs</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>3. PSS</td>
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<td>.16***</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>4. NDI</td>
<td>.08***</td>
<td>.07***</td>
<td>.07***</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>5. BIPOC</td>
<td>.04**</td>
<td>.04</td>
<td>.10***</td>
<td>.08***</td>
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</table>

**Range**

<table>
<thead>
<tr>
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<th>(1-2)</th>
<th>(0-7)</th>
<th>(1-3)</th>
<th>(1-5)</th>
<th>(1-2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>1.30</td>
<td>.69</td>
<td>1.27</td>
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<td>BIPOC</td>
<td>1.34</td>
<td>.75</td>
<td>1.40</td>
<td>2.82</td>
<td>--</td>
</tr>
<tr>
<td>Non-BIPOC</td>
<td>1.29</td>
<td>.68</td>
<td>1.24</td>
<td>2.65</td>
<td>--</td>
</tr>
<tr>
<td>High PSS</td>
<td>1.27</td>
<td>.62</td>
<td>--</td>
<td>2.64</td>
<td>1.13</td>
</tr>
<tr>
<td>Medium PSS</td>
<td>1.36</td>
<td>.89</td>
<td>--</td>
<td>2.77</td>
<td>1.21</td>
</tr>
<tr>
<td>Low PSS</td>
<td>1.48</td>
<td>1.11</td>
<td>--</td>
<td>2.92</td>
<td>1.25</td>
</tr>
</tbody>
</table>

**Standard Deviation**

|       | .46  | .91  | .57  | 1.21  | .36  |

*p < 0.05, **p < 0.01, ***p < 0.001

**Power Analysis.** To assess sufficient power for the analyses, a power analysis is conducted using the RMSEA statistic (Preacher & Coffman, 2006). To assist with this, Preacher & Coffman (2006) created a website that generates R code based on one’s SEM model statistics, which is then submitted to Rweb for a power calculation (Kim, 2021c).

For the proposed mediation model, inputting a sample size of 5,447, $\alpha = .05$, 2 degrees of freedom, RMSEA of .007 from the proposed model, and an alternative RMSEA of .08 provides a power estimate of 1. This indicates suitable power for testing the covariance of the SEM model (Preacher & Coffman, 2006). Similarly, to test the power for the PSS moderation model, inputting a sample size of 5,447, $\alpha = .05$, 9 degrees of freedom, RMSEA of .000 from the proposed model, and an alternative RMSEA of .08 provides a power estimate of 1 (Preacher & Coffman, 2006). Finally, to test the power for the BIPOC moderation model, inputting a sample size of 5,447, $\alpha = .05$, 2 degrees of
freedom, RMSEA of .000 from the proposed model, and an alternative RMSEA of .08 provides a power estimate of 1 (Preacher & Coffman, 2006).

**Sample Characteristics.** Table 5 shows descriptive statistics of the 2010 Washington State BRFSS sample (n = 5,447), which indicate the majority of participants are female (61.8%), white (84.7%), age 50 years or older (52.1%), and live in average deprivation neighborhoods (32.1%). Table 6 provides frequency statistics of Adverse Childhood Experiences among sample respondents. Only those with at least 1 ACE were included in the analysis. The majority of individuals reported 1-3 ACE exposures (70.1%), while nearly a third of the sample reported exposure to 4 or more types of childhood trauma (29.9%). The most common type of ACE exposure was emotional abuse (55.3%) closely followed by living with a household member who used substances, including problematic alcohol use, illegal street drugs or abusing prescription medication (49.8%). Table 7 provides type and prevalence of the seven health risk behavior variables measured in the 2010 Washington BRFSS sample. Over half of respondents report no health risk behaviors (53.7%), and just under half of respondents endorse at least one of the seven health risk behaviors surveyed (46.2%). Being a current smoker was the most common health risk behavior (20.3%), closely followed by physical inactivity (17.7%) and at least one episode of binge drinking (16%) in the last 30 days.
Table 5.

Descriptive Statistics in the 2010 Washington BRFSS Sample (n = 5,447)

<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>Percent (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>2082</td>
<td>38.2</td>
</tr>
<tr>
<td>Female</td>
<td>3365</td>
<td>61.8</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18-34</td>
<td>833</td>
<td>15.3</td>
</tr>
<tr>
<td>35-49</td>
<td>1776</td>
<td>32.6</td>
</tr>
<tr>
<td>50-64</td>
<td>2838</td>
<td>52.1</td>
</tr>
<tr>
<td><strong>Race/Ethnicity</strong></td>
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<td></td>
</tr>
<tr>
<td>BIPOC</td>
<td>835</td>
<td>15.3</td>
</tr>
<tr>
<td>Black</td>
<td>85</td>
<td>1.6</td>
</tr>
<tr>
<td>Other Race (Asian, Hawaiian, Native American, Other)</td>
<td>232</td>
<td>4.3</td>
</tr>
<tr>
<td>Multiracial</td>
<td>195</td>
<td>3.6</td>
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<tr>
<td>Hispanic</td>
<td>323</td>
<td>5.9</td>
</tr>
<tr>
<td>Non-BIPOC</td>
<td>4612</td>
<td>84.7</td>
</tr>
<tr>
<td><strong>Perceived Social Support</strong></td>
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<td></td>
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<tr>
<td>High</td>
<td>4343</td>
<td>79.7</td>
</tr>
<tr>
<td>Medium</td>
<td>763</td>
<td>14.0</td>
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<tr>
<td>Low</td>
<td>341</td>
<td>6.3</td>
</tr>
<tr>
<td><strong>Neighborhood Deprivation Level</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 – Least Deprivation</td>
<td>1131</td>
<td>20.8</td>
</tr>
<tr>
<td>2 – Below Average Deprivation</td>
<td>1287</td>
<td>23.6</td>
</tr>
<tr>
<td>3 – Average Deprivation</td>
<td>1751</td>
<td>32.1</td>
</tr>
<tr>
<td>4 – Above Average Deprivation</td>
<td>791</td>
<td>14.5</td>
</tr>
<tr>
<td>5 – Most Deprivation</td>
<td>487</td>
<td>8.9</td>
</tr>
</tbody>
</table>
Table 6.

*Frequency Statistics of Adverse Childhood Experiences in the 2010 Washington BRFSS Sample (n = 5,447)*

<table>
<thead>
<tr>
<th>Type of ACE</th>
<th>N</th>
<th>Percent (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sexual Abuse</td>
<td>1429</td>
<td>26.2</td>
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<tr>
<td>Physical Abuse</td>
<td>1648</td>
<td>30.3</td>
</tr>
<tr>
<td>Emotional Abuse</td>
<td>3011</td>
<td>55.3</td>
</tr>
<tr>
<td>Parental Domestic Violence</td>
<td>1478</td>
<td>27.1</td>
</tr>
<tr>
<td>Household Member Substance Use</td>
<td>2713</td>
<td>49.8</td>
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<tr>
<td>Household Member Mental Illness</td>
<td>1817</td>
<td>33.4</td>
</tr>
<tr>
<td>Household Member Incarceration</td>
<td>485</td>
<td>8.9</td>
</tr>
<tr>
<td>Parental Separation or Divorce</td>
<td>2399</td>
<td>44.0</td>
</tr>
<tr>
<td>1-3 ACE Exposures</td>
<td>3821</td>
<td>70.1</td>
</tr>
<tr>
<td>4 or More ACE Exposures</td>
<td>1626</td>
<td>29.9</td>
</tr>
</tbody>
</table>

*Note.* Only participants with at least 1 ACE were examined in this study.

Table 7.

*Type and Prevalence of Health Risk Behaviors in the 2010 Washington BRFSS Sample (n = 5,447)*

<table>
<thead>
<tr>
<th>Health Outcome</th>
<th>N</th>
<th>Prevalence in Sample (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marijuana Use (Last 30 Days)</td>
<td>431</td>
<td>7.9</td>
</tr>
<tr>
<td>Binge Drinking (Last 30 Days)</td>
<td>874</td>
<td>16</td>
</tr>
<tr>
<td>Current Smoker</td>
<td>1107</td>
<td>20.3</td>
</tr>
<tr>
<td>Engaged in Risky HIV Behavior (Last 12 Months)</td>
<td>186</td>
<td>3.4</td>
</tr>
<tr>
<td>Drinking and Driving (Last 30 Days)</td>
<td>152</td>
<td>2.8</td>
</tr>
<tr>
<td>Physical Inactivity (Last 30 Days)</td>
<td>963</td>
<td>17.7</td>
</tr>
<tr>
<td>Used Painkillers to Get High (Last 30 Days)</td>
<td>49</td>
<td>1</td>
</tr>
<tr>
<td>0 Health Risk Behaviors</td>
<td>2926</td>
<td>53.7</td>
</tr>
<tr>
<td>1 Health Risk Behavior</td>
<td>1608</td>
<td>29.5</td>
</tr>
<tr>
<td>2 Health Risk Behaviors</td>
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<td>12.0</td>
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<td>3 Health Risk Behaviors</td>
<td>204</td>
<td>3.7</td>
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<td>4 Health Risk Behaviors</td>
<td>49</td>
<td>.9</td>
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<td>5 Health Risk Behaviors</td>
<td>7</td>
<td>.1</td>
</tr>
<tr>
<td>6 Health Risk Behaviors</td>
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<td>0</td>
</tr>
<tr>
<td>7 Health Risk Behaviors</td>
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<td>0.0</td>
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</table>
Preliminary Mediation and Moderation Analyses. To provide suitable backing for the hypothesized SEM models, separate mediation and moderation analyses were conducted in SPSS using PROCESS (Hayes, 2018). Table 8 and Figure 4 provide the results of the PROCESS mediation analysis of neighborhood deprivation on the relationship between exposure to multiple ACEs and health risk behaviors, including control variables. As hypothesized, neighborhood deprivation is a mechanism explaining the relationship between exposure to multiple ACEs and increased health risk behaviors. Exposure to multiple ACEs in childhood is significantly and positively associated with living in a high deprivation environment in adulthood ($\beta = .101, p = .005$), which in turn increases health risk behaviors ($\beta = .046, p = .000$). There is a significant direct effect from exposure to multiple ACEs on health risk behaviors ($\beta = .243, p = .000$), as well as a significant indirect effect of multiple ACEs on health risk behaviors ($\beta = .005, 95\% \text{ CI} = .001 - .009$), as the 95% confidence intervals do not contain 0. Thus, neighborhood deprivation significantly mediates the association between multiple ACEs and HRBs.

Table 8.

Results of the PROCESS Mediation Analysis of Neighborhood Deprivation on the Relationship Between Adverse Childhood Experiences and Health Risk Behaviors (n = 5,447)

<table>
<thead>
<tr>
<th>Path</th>
<th>Coefficient</th>
<th>SE</th>
<th>t</th>
<th>p value</th>
<th>95% CI</th>
<th>R²</th>
<th>F (p)</th>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>LLCI</td>
<td>ULCI</td>
<td></td>
</tr>
<tr>
<td>$c$</td>
<td>.247</td>
<td>.026</td>
<td>9.37</td>
<td>.000</td>
<td>.196</td>
<td>.299</td>
<td>.041</td>
</tr>
<tr>
<td>$a$</td>
<td>.101</td>
<td>.036</td>
<td>2.83</td>
<td>.005</td>
<td>.031</td>
<td>.172</td>
<td>.010</td>
</tr>
<tr>
<td>$b$</td>
<td>.046</td>
<td>.010</td>
<td>4.62</td>
<td>.000</td>
<td>.027</td>
<td>.066</td>
<td>.045</td>
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<tr>
<td>Direct effect ($c'$)</td>
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<td>.026</td>
<td>9.21</td>
<td>.000</td>
<td>.191</td>
<td>.294</td>
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</tr>
<tr>
<td>Indirect effect</td>
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<td>.002</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>.001</td>
<td>.009</td>
</tr>
</tbody>
</table>

*p < 0.05, **p < 0.01, ***p < 0.001. SE, standard error; CI, confidence interval; LLCI, lower limit confidence interval; ULCI, upper limit confidence interval. Coefficients are unstandardized.
Figure 4.

Model of the Mediating Role of Neighborhood Deprivation on the Relationship Between Adverse Childhood Experiences and Health Risk Behaviors

![Diagram showing the mediation model]

Note. *p < 0.05, **p < 0.01, ***p < 0.001. ACEs, Adverse Childhood Experiences; NDI, Neighborhood Deprivation Index; HRBs, Health Risk Behaviors. Coefficients are unstandardized.

Table 9 shows the results of the PROCESS moderation analysis conducted in SPSS for the three levels of PSS (low, medium, and high). For a multicategorical moderator, PROCESS uses pairwise comparisons of the moderator to test for interaction effects with the predictor (Hayes, 2018). Results show a significant interaction between multiple ACEs and PSS, specifically between the low and high PSS groups ($\beta = .257$, $p < .01$, 95% CI = .062 – .453), indicating a moderating effect. Additionally, PROCESS provides the conditional effects of ACEs at values of PSS (Table 10), which show the
differences in effect between low ($\beta = .433, p < .001, 95\% \text{ CI} = .247 – .620$) and high PSS ($\beta = .176, p < .001, 95\% \text{ CI} = .117 – .235$). Using pick-a-point analysis, Figure 5 graphically represents the conditional effects of ACEs on health risk behaviors for the three values of PSS.

Table 9.

Results of the PROCESS Moderation Analysis of Perceived Social Support on the Relationship Between Adverse Childhood Experiences and Health Risk Behaviors ($n = 5,447$)

<table>
<thead>
<tr>
<th>Model</th>
<th>Estimate</th>
<th>SE</th>
<th>t</th>
<th>p value</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>LLCI</td>
</tr>
<tr>
<td>Intercept</td>
<td>1.220</td>
<td>.088</td>
<td>13.755</td>
<td>&lt; .001</td>
<td>1.037</td>
</tr>
<tr>
<td>Multiple ACEs</td>
<td>.176</td>
<td>.030</td>
<td>5.869</td>
<td>&lt; .001</td>
<td>.117</td>
</tr>
<tr>
<td>Medium vs. High PSS</td>
<td>.090</td>
<td>.104</td>
<td>.872</td>
<td>.383</td>
<td>-.113</td>
</tr>
<tr>
<td>Low vs. Medium PSS</td>
<td>.033</td>
<td>.177</td>
<td>.187</td>
<td>.852</td>
<td>-.314</td>
</tr>
<tr>
<td>Low vs. High PSS</td>
<td>.057</td>
<td>.154</td>
<td>.371</td>
<td>.711</td>
<td>-.245</td>
</tr>
<tr>
<td>ACEs * Medium vs. High PSS</td>
<td>.119</td>
<td>.073</td>
<td>1.630</td>
<td>.103</td>
<td>-.024</td>
</tr>
<tr>
<td>ACEs * Low vs. Medium PSS</td>
<td>.139</td>
<td>.116</td>
<td>1.195</td>
<td>.232</td>
<td>-.089</td>
</tr>
<tr>
<td><strong>ACEs * Low vs. High PSS</strong></td>
<td><strong>.257</strong></td>
<td><strong>.101</strong></td>
<td><strong>2.582</strong></td>
<td><strong>.009</strong></td>
<td><strong>.062</strong></td>
</tr>
</tbody>
</table>

*Note*. SE, standard error; CI, confidence interval; LLCI, lower limit confidence interval; ULCI, upper limit confidence interval. All estimates are standardized. $R^2 = .062, F(8, 5,438) = 44.806, p = .000$.

Table 10.

The Conditional Effects of ACEs on HRBs at values of the PSS Moderator

<table>
<thead>
<tr>
<th>Level of PSS</th>
<th>Effect</th>
<th>SE</th>
<th>t</th>
<th>p value</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>LLCI</td>
</tr>
<tr>
<td>Low (Never, Rarely)</td>
<td>.433</td>
<td>.095</td>
<td>4.554</td>
<td>&lt; .001</td>
<td>.247</td>
</tr>
<tr>
<td>Medium (Sometimes)</td>
<td>.295</td>
<td>.066</td>
<td>4.434</td>
<td>&lt; .001</td>
<td>.164</td>
</tr>
<tr>
<td>High (Always, Usually)</td>
<td>.176</td>
<td>.030</td>
<td>5.869</td>
<td>&lt; .001</td>
<td>.117</td>
</tr>
</tbody>
</table>

*Note*. ACEs, Adverse Childhood Experiences; PSS, Perceived Social Support
Figure 5

Conditional Effects of ACEs on HRBs at Values of the PSS Moderator

Note. ACEs, Adverse Childhood Experiences; PSS, Perceived Social Support; NDI, Neighborhood Deprivation Index
**SEM Analyses in Amos.** To assess the mediation effect of neighborhood deprivation on the relationship between the accumulation of multiple ACEs and HRBs, a mediation path model was run in Amos including control variables. The Maximum Likelihood method for estimating parameters was utilized, as well as bootstrapping of 5,000 resamples with 95% bias corrected confidence intervals to assess the effect of the mediator (Kim, 2021c). The initial hypothesized model showed fairly good overall fit (RMSEA = .036, $X^2 (2) = 16.267, p = .000, CFI = .971$). In the process of re-specifying to improve model fit, modification indices with values greater than 10 were used to add a path between Age and NDI (Kim, 2021c). Additionally, to create an over-identified, parsimonious model, insignificant paths among the control variables were constrained to 0, specifically between Age and Sex and Sex and BIPOC (Kim, 2021b). The resulting structural model shows excellent fit (RMSEA = .007, $X^2 (3) = 3.742, p = .291, CFI = .998$) and multivariate normality (kurtosis = .630, C.R. = 2.373). As found previously in the PROCESS mediation analysis, the accumulation of multiple ACEs has a significant positive association with the development of an array of health risk behaviors ($\beta = .123, p < .001, 95\%$ bias corrected CI = .095 – .150), as well as a significant direct effect on neighborhood deprivation. Neighborhood deprivation also demonstrates a significant direct effect on health risk behaviors ($\beta = .061, p < .001, 95\%$ bias corrected CI = .036 – .087). Additionally, multiple ACEs are positively associated with HRBs through neighborhood deprivation. As Table 11 shows, ACEs demonstrate significant indirect effects through NDI to HRBs ($\beta = .002, p = .003, 95\%$ bias corrected CI = .001 – .005).
Table 11.
Standardized Direct, Indirect and Total Effects from the Amos Mediation Analysis of Neighborhood Deprivation on the Relationship Between ACEs and HRBS

<table>
<thead>
<tr>
<th>Pathway</th>
<th>Direct Effects</th>
<th>p</th>
<th>Indirect Effects</th>
<th>p</th>
<th>Total Effects</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multiple ACEs → HRBs</td>
<td>.123</td>
<td>.000</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Multiple ACEs → NDI</td>
<td>.040</td>
<td>.001</td>
<td>--</td>
<td>--</td>
<td>.040</td>
<td>.001</td>
</tr>
<tr>
<td>NDI → HRBs</td>
<td>.061</td>
<td>.000</td>
<td>--</td>
<td>--</td>
<td>.061</td>
<td>.000</td>
</tr>
<tr>
<td>Multiple ACEs → NDI → HRBs</td>
<td>--</td>
<td>--</td>
<td>.002</td>
<td>.003</td>
<td>.125</td>
<td>.000</td>
</tr>
</tbody>
</table>

**Note.** ACEs, Adverse Childhood Experiences; NDI, Neighborhood Deprivation Index; HRBs, Health Risk Behaviors

To analyze the moderating effect of PSS, and to understand which path, if any, PSS moderates, a multigroup analysis approach was employed using Amos for the SEM path model (Figure 2), including control variables. Again, the Maximum Likelihood method for estimating parameters was utilized, as well as bootstrapping of 5,000 resamples with 95% bias corrected confidence intervals. The “global model fit” of the unconstrained model demonstrates excellent model fit, indicating the path model is appropriate for all three groups (RMSEA = .000, $X^2 (9) = 7.686$, $p = .566$, CFI = 1.00) (Kim, 2021d, p. 15). By utilizing the chi-square difference test of the nested models, which compares the unconstrained SEM model to a constrained model in which all paths are constrained to 1, the delta change in chi-square shows at least one path coefficient significantly differs for the three values of PSS ($\Delta X^2 (\Delta df = 16) = 27.047$, $p = .041$).

Follow-up local chi-square tests help determine which path is significant. In this approach, each individual path coefficient is constrained to be equal across the levels of PSS (Kim, 2021d). Results show that the pathway between exposure to multiple ACEs and HRBs is significantly different among levels of PSS ($\Delta X^2 (\Delta df = 2) = 7.545$, $p = .023$), indicating moderation. To determine which of the PSS groups significantly differ
along the multiple ACEs to HRBs pathway, secondary local chi-square tests are
conducted with separate pairwise comparisons between levels of PSS – low vs. high, low
vs. medium, and high vs. medium – by constraining each of these pathways to be equal
(Kim, 2021d). Similarly to the results in the PROCESS Macro, findings indicate a
significant delta change in chi-square between the low and high PSS groups ($\Delta \chi^2 (\Delta df = 1) = 5.462, p = .019$) with marginal level significance between the high and medium PSS
groups ($\Delta \chi^2 (\Delta df = 1) = 2.714, p = .099$). The delta change in chi-square was not
significant between the medium and low levels of PSS ($p > .10$). Table 13 provides the
standardized coefficients of the unconstrained model for each pairwise comparison of
perceived social support.

Table 12.

Model Comparisons from the Multi-Group Analysis of the PSS Variable

<table>
<thead>
<tr>
<th>Model</th>
<th>$\chi^2$ Value</th>
<th>$df$</th>
<th>$p$ value</th>
<th>$\Delta \chi^2$</th>
<th>$\Delta df$</th>
<th>$\Delta \chi^2$ $p$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unconstrained</td>
<td>7.686</td>
<td>9</td>
<td>.566</td>
<td>--</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Fully Constrained</td>
<td>34.732</td>
<td>25</td>
<td>.093</td>
<td>27.047</td>
<td>16</td>
<td>.041</td>
</tr>
<tr>
<td>Constrained: ACEs to HRBs</td>
<td>15.231</td>
<td>11</td>
<td>.172</td>
<td>7.545</td>
<td>2</td>
<td>.023</td>
</tr>
<tr>
<td>Constrained: ACEs to NDI</td>
<td>8.104</td>
<td>11</td>
<td>.704</td>
<td>.418</td>
<td>2</td>
<td>.811</td>
</tr>
<tr>
<td>Constrained: NDI to HRBs</td>
<td>8.133</td>
<td>11</td>
<td>.701</td>
<td>.447</td>
<td>2</td>
<td>.800</td>
</tr>
</tbody>
</table>

Note. ACEs, Adverse Childhood Experiences; NDI, Neighborhood Deprivation Index; HRBs, Health Risk Behaviors

Table 13.

Standardized Estimates from the Multi-Group Analysis Pairwise Comparisons of
Perceived Social Support (PSS) on the Pathway Between Multiple ACEs and HRBs

<table>
<thead>
<tr>
<th>Pathway</th>
<th>Low PSS Estimate</th>
<th>Medium PSS Estimate</th>
<th>High PSS Estimate</th>
<th>$\Delta \chi^2$ $p$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multiple ACEs $\rightarrow$ HRBs</td>
<td>.212</td>
<td>--</td>
<td>.089</td>
<td>.019</td>
</tr>
<tr>
<td></td>
<td>--</td>
<td>.143</td>
<td>.089</td>
<td>.099</td>
</tr>
<tr>
<td></td>
<td>.212</td>
<td>.143</td>
<td>--</td>
<td>.316</td>
</tr>
</tbody>
</table>

Note. ACEs, Adverse Childhood Experiences; HRBs, Health Risk Behavior
Because the direct effect of the BIPOC variable on NDI and its covariance with ACEs cannot be assessed in the Amos multigroup moderation analysis, standardized estimates from the NDI mediation analysis are utilized to better understand the significant associations of this variable. As Table 14 demonstrates, people of color in this sample have slightly higher exposure to multiple forms of child trauma and are also more likely to live in higher deprivation environments, as hypothesized.

Table 14.

**Standardized Estimates of the BIPOC Variable from the Amos Analysis of Neighborhood Deprivation on the Relationship Between ACEs and HRBS**

<table>
<thead>
<tr>
<th>Pathway</th>
<th>Estimate</th>
<th>SE</th>
<th>C.R.</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multiple ACEs ↔ BIPOC</td>
<td>.006</td>
<td>.002</td>
<td>2.915</td>
<td>.004</td>
</tr>
<tr>
<td>BIPOC → NDI</td>
<td>.043</td>
<td>.046</td>
<td>3.152</td>
<td>.002</td>
</tr>
</tbody>
</table>

*Note.* ACEs, Adverse Childhood Experiences; NDI, Neighborhood Deprivation Index; HRBs, Health Risk Behavior; BIPOC includes Black, Indigenous, and People of Color

Similar to the PSS moderation analysis, a multigroup path analysis using the dichotomous BIPOC variable helps assess if there is a moderating effect of race/ethnicity on the pathway between the accumulation of multiple ACEs and HRBs. This analysis uses the Maximum Likelihood method for estimating parameters, as well as bootstrapping of 5,000 resamples with 95% bias corrected confidence intervals. The “global model fit” of the unconstrained model demonstrates excellent model fit, indicating the path model is appropriate for both groups, BIPOC and non-BIPOC (RMSEA = .000, \( \chi^2 \) (2) = 1.136, \( p = .567 \), CFI = 1.00) (Kim, 2021d, p. 15). By utilizing the chi-square difference test of the nested models, which compares the unconstrained model to the constrained model, the delta change in chi-square shows that none of the path coefficients significantly differ for the two values of race/ethnicity (\( \Delta \chi^2 (\Delta df = 3) = \)
2.60, \( p = .457 \) as shown in Table 15. This indicates being BIPOC does not moderate the effect of multiple ACEs on HRBs, nor any other pathway. Thus, follow-up local chi-square difference tests are not necessary.

Table 15.

<table>
<thead>
<tr>
<th>Model</th>
<th>( \chi^2 ) Value</th>
<th>( df )</th>
<th>( p ) value</th>
<th>( \Delta \chi^2 )</th>
<th>( \Delta df )</th>
<th>( \Delta \chi^2 ) ( p ) value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unconstrained</td>
<td>1.136</td>
<td>2</td>
<td>.567</td>
<td>--</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Fully Constrained</td>
<td>3.736</td>
<td>5</td>
<td>.588</td>
<td>2.60</td>
<td>3</td>
<td>.457</td>
</tr>
</tbody>
</table>

Note. BIPOC includes Black, Indigenous, and People of Color

To better understand this result and any potential differences between racial groups within the BIPOC variable, the BIPOC variable was further broken down into four racial categories based on available data: Black, Other (Asian, Hawaiian, Native American, and Other), Multiracial, and Hispanic. A multigroup analysis was conducted for the five racial categories (White, Black, Other Race, Multiracial, and Hispanic) using the Maximum Likelihood method for estimating parameters, as well as bootstrapping of 5,000 resamples with 95% bias corrected confidence intervals. By utilizing the chi-square difference test of the nested models, the delta change in chi-square shows that none of the path coefficients significantly differ for the five groups of race/ethnicity (\( \Delta \chi^2 (\Delta df = 12) = 18.401, p = .104 \) as shown in Table 16. This indicates the invariance of race/ethnicity along the multiple ACEs to HRBs, ACEs to NDI, and NDI to HRBs pathways.
Table 16.

*Model Comparisons from the Multi-Group Analysis of Multiple Races/Ethnicity*

<table>
<thead>
<tr>
<th>Model</th>
<th>$X^2$ Value</th>
<th>df</th>
<th>$p$ value</th>
<th>$\Delta X^2$</th>
<th>$\Delta df$</th>
<th>$\Delta X^2$ $p$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unconstrained</td>
<td>4.282</td>
<td>5</td>
<td>.510</td>
<td>--</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Fully Constrained</td>
<td>22.683</td>
<td>17</td>
<td>.160</td>
<td>18.401</td>
<td>12</td>
<td>.104</td>
</tr>
</tbody>
</table>

*Note.* Multiple Races/Ethnicity includes comparisons between Black, Other, Multiracial, Hispanic, and White groups
Chapter 5:

Discussion

This study both corroborates and builds upon previous child trauma research to help explain health differences for those with multiple exposures to ACEs. Specifically, this research helps reveal the functioning of socio-structural stress pathways and the protective effect of social resources, providing points of entry for interrupting the “cascade” of social and health outcomes often resulting from multiple exposures to child trauma (Font & Maguire-Jack, 2016; Jones et al., 2018, p. 36). As expected, the findings in this study confirm how multiple ACEs contribute to the development of an array of health risk behaviors in adulthood, including drug use, problematic drinking, risky HIV behavior, drinking and driving, smoking, and physical inactivity. The threshold of experiencing four or more types of trauma is associated with an increased development of HRBs, matching previous findings (Felitti et al., 1998; Hughes et al., 2018).

The Mediating Effect of Neighborhood Deprivation

Multiple ACEs also demonstrate a significant pathway to adulthood neighborhood deprivation. In this sample, individuals with four or more ACEs were living in a more deprived environment compared to individuals with fewer exposures. This finding supports previous research indicating that those with higher levels of ACEs tend to have lower adulthood SES (Jones et al., 2018), including reduced educational attainment (Font & Maguire-Jack, 2016) and a greater likelihood for unemployment (Topitzes et al., 2016). The significance of exposure to multiple ACEs contributing to neighborhood deprivation clarifies that the association between multiple experiences of child trauma and poverty correlates may be wide spanning, demonstrated by the number of poverty measures
encapsulated within neighborhood deprivation. The indicators of social inequity within NDI reach beyond the current understanding of associations with multiple ACEs, such as greater proclivity for receiving public assistance, living in substandard housing conditions, and fewer opportunities for wealth and asset accrual (i.e. not owning a home, lower home values, being employed in non-management positions). This provides new insight into the kinds of social inequities researchers and clinicians should to be attuned to in examining the associations and/or chain effects of multiple ACEs. In stress proliferation research, it is hypothesized that exposure to multiple types of child trauma predisposes individuals to increased adversity in adulthood, such as living in high deprivation environments, by reducing educational attainment and contributing to "lower socioeconomic achievement" (Burton-Jeangros et al., 2015; Font & Maguire-Jack, 2016).

Further, examining the specific connection between community-level deprivation factors and ACE-related health behaviors is a relationship that has not been well studied. While research supports the "graded" or "dose response" relationship between ACEs and health risks, it has been unclear exactly why those with multiple exposures have worsened risk for HRBs and if other factors may contribute to this relationship (Felitti et al., 1998). While small in its effects, the mediation of neighborhood deprivation potentially clarifies some of the "mechanisms through which ACEs accumulate adversity in adulthood" – mechanisms that influence a trajectory of social and health consequences (Jones et al., 2018, p. 36). The significant indirect effect of neighborhood deprivation may help explain the relationship between worsened health outcomes among those with multiple childhood trauma exposures and implicates surrounding environmental and community-level stressors in exacerbating the effects of child trauma. These findings
demonstrate neighborhood deprivation plays a contributing role in both the “prevalence and severity” of HRBs among those with four or more types of child trauma (Laraia et al., 2012, p. 3).

The way neighborhood deprivation functions on this pathway may take a variety of forms, as stress research has shown that “disadvantaged social position is both a source of adversity and a drain on the capacity to cope” (Pampel et al., 2010, p. 352). As mentioned previously, the overwhelming number of severe daily stressors typically present in highly deprived neighborhoods, including factors not measured in the NDI, like environmental pollutants, high incarceration rates, hazardous working conditions, and crime, can trigger engagement in health risk behaviors (Ellis, 2017). This can happen for two main reasons: First, risk behaviors often provide mood regulation (albeit temporarily) for chronic stress, which may explain why both neighborhood deprivation and severe childhood trauma are characterized by heightened risk of maladaptive coping (Pampel et al., 2010; Jones et al., 2018, p. 37). Coupled with this, low-income settings offer fewer incentives for long-term investment in health, such as shorter lifespan and limited wealth accrual, encouraging an orientation toward “short-term gain,” which tends to foster a perception that the risks of these behaviors matter less (Pampel et al., 2010, p. 356). Thus, stress combined with limited incentives in high deprivation environments affect individuals’ overall motivation for healthy behavior and the benefit of avoiding health risks (Pampel et al., 2010).

Secondly, neighborhoods with high deprivation can reduce individuals’ ability to access resources to achieve health, which directly shapes behavior. For example, low-income environments can “cultivate” unhealthy behaviors through direct obstacles in the
environment, such as food deserts, advertisements for drinking or tobacco, higher percentage of liquor stores, lack of access to health clinics, or norms around smoking and inactivity (Pampel et al., 2010, p. 360; Laraia et al., 2012). The impact of social inequity on both motivation and reduced resources to achieve health increases and causes differential outcomes in health risk behaviors among those in high deprivation environments. For many of those who have experienced multiple childhood traumas, stress response systems are already heightened and it is presumed that the added psychological and material barriers inherent to neighborhood deprivation negatively impact health behaviors.

While small in its indirect effects, the significance of neighborhood deprivation mediating the relationship between exposure to multiple ACEs and HRBs is not limited to a better understanding of the prevalence of specific health risk behaviors among those with multiple types of trauma. Rather, it also sheds light on how socioeconomic mechanisms function in the resulting “cascade” of ill health often triggered by HRBs, as HRBs, like smoking and problematic drinking, are common causes for ACE-related diseases (Hughes et al., 2017; Font & Maguire-Jack, 2016, p. 398; Jones et al., 2018, p. 36). In health studies examining the association between neighborhood deprivation and increased prevalence of diabetes and cardiometabolic risk, findings show that neighborhood deprivation exerts influence on the severity of these diseases through “risk factors that are most influenced by behaviors” (Laraia et al., 2012, p. 8). This means that high neighborhood deprivation shapes health behavior, like sedentary lifestyle and smoking, in ways that foster the development, “accelerate” the impact, and impede the management of these diseases, such as through resource deficits, ongoing stressors, and
social cues (Laraia et al., 2012, p. 9). This same understanding can be applied to ACE-related behaviors and their resulting health problems, and may also explain the significantly increased risk of chronic conditions among those with multiple ACEs. As the results show, for those with multiple ACEs living in deprived environments, both neighborhood deprivation and ACEs have separate and combined influential risk on HRBs, which creates greater likelihood of dealing with health problems from these behaviors, such as heart disease, obesity, diabetes, and cancer (CDC, 2019).

However, because of the persisting significant direct effect of multiple ACEs contributing to HRBs while including neighborhood deprivation, this could indicate other unmeasured mechanisms are operating within this relationship, such as additional adverse events (i.e. houselessness, incarceration, etc.) (Jones et al., 2018). It is also possible that the perceived stress and psychological impact of neighborhood deprivation are more important determinants of resulting health risks than the NDI value itself – that an individual’s experience of neighborhood deprivation and any resulting psychological symptoms are a better proxy for underlying mechanistic functioning on ill health. This was shown to be true in a study exploring the effect of neighborhood disorder on health, where findings indicated that the psychological effects of neighborhood deprivation, such as feelings of distress, fully explained resulting incidence of obesity (Pampel et al., 2011, p. 362). It is also possible some individuals may be better equipped to handle the stress of neighborhood deprivation through different resources or better quality medical and therapeutic care, which is not parsed out in the NDI value (Roux & Mair, 2010).

Alternatively, the persisting direct effect could also point to the powerful stress effects of multiple ACEs that cause dysregulation in numerous physiologic processes,
particularly psychological impacts such as anxiety and depression that are implicated in biobehavioral explanations and can appear quite early in youth (Jones et al., 2018, p. 37). We also know those who experience multiple types of child trauma are much more likely to experience adulthood mental health impairment (Jones et al., 2018), signaling child trauma plays a large role in mental illness across the life course. Understanding the amount ACE-related health risks are attributable to child and adulthood psychological symptoms would be helpful in better understanding this pathway.

The Moderating Effect of Perceived Social Support

This study also clarifies the importance of perceived social and emotional support on the relationship between the accumulation of multiple ACEs and the subsequent development of health risk behaviors. Previous studies have shown the benefits of various types of social support for child trauma during youth (Bellis et al., 2018; Narayan et al., 2021), but the particular benefit for those with multiple ACE exposures and its potential to reduce engagement with HRBs have not been fully explored. People with high levels of PSS, or those who always or usually receive the social and emotional support they need, regardless of the type of support, showed a protective “buffering” effect from ACE toxicity, compared to those with low PSS who rarely or never receive social and emotional support. This was true for those with any ACE exposure but was particularly apparent in reducing the effect of ACEs on HRBs among those with multiple ACE exposures. Those with low PSS and 4 or more ACEs demonstrated almost double the HRB rates as those with high PSS and 4 or more exposures. Also, the gap between those with 1-3 ACE exposures and 4 or more ACEs and resulting HRBs was much wider for the low PSS group than for the high PSS group.
These findings are consonant with previous research that shows only high levels of PSS are effective in buffering the effects of high perceived stress situations (Ioannou et al., 2019). Given childhood trauma is an incredibly high stress experience – sometimes chronic – with a multitude of potentially severe ramifications (child removal, foster placement, loss of a trusted caregiver, injury, etc.), it follows that high levels of PSS would be required to buffer its impacts. High levels of PSS are shown to improve self-esteem and decrease depression symptoms by increasing feelings of self-worth and sense of belonging, all critical components in coping with trauma and trauma recovery. It also appears that high PSS has a circular relationship with self-esteem, wherein PSS boosts self-esteem, and higher self-esteem leads to a higher likelihood of using those social supports (Ioannou et al., 2019).

Because PSS did not moderate the pathways between multiple ACEs and NDI or NDI to HRBs, these results could indicate individual-level social resources are not effective in preventing the SES adversity associated with exposure to multiple ACEs, nor effective in buffering stressful community or structural experiences, which matches previous research (Stockdale et al., 2007). However, PSS does demonstrate benefit for those with any childhood trauma exposure regardless of neighborhood characteristics, especially those with 4 or more exposures. Clarifying that high social support plays a role in reducing engagement with health risk behaviors among those with multiple exposures potentially lays the groundwork for evidence-based intervention efforts. However, clinicians should be aware that these approaches may have little impact on the chronic environmental stressors associated with neighborhood deprivation.
The Insignificant Moderating Effect of Being BIPOC

As mentioned previously, racial category is used in this study as a proxy for the functioning of the “disproportionate distribution” of people of color toward experiencing “chronic, negative environmental, social, and psychological stressors, as well as the greater availability of environmental sources of unhealthy behaviors” that could increase the toxicity of multiple ACEs and result in higher HRBs (Jackson et al., 2010, p. 938). While being a person of color in this sample was associated with having had greater exposure to multiple ACEs and living in a more deprived environment, which matches previous research (Ellis, 2017), being BIPOC was not a significant moderator of the relationship between multiple ACEs and HRBs, nor any other pathway.

Because this study’s BIPOC race/ethnicity variable contains many diverse groups, including Black, Hispanic, Other Races, and Multiracial individuals, it was important to further parse out the BIPOC variable into individual racial/ethnic groups to understand if treating race in a monolithic way contributed to the lack of a statistically significant finding. Doing so better attends to potential dimensions of social inequity (and its potential effects on HRBs) that exist between different racial groups. However, even after parsing out the BIPOC race/ethnicity category, the moderation effect was still not statistically significant. This means that in this sample, the toxicity of multiple ACEs functioned similarly, regardless of an individual’s race/ethnicity, in producing resulting health risks.

The lack of a statistically significant effect of race/ethnicity could shed important light on ACE-related premature mortality rates among people of color. Because people of color with exposure to multiple ACEs demonstrate higher rates of premature mortality
compared to non-BIPOC individuals (Brown et al., 2009), the findings suggest this does not appear to be due to disproportionately increased engagement with health risk behaviors. Further research is needed to understand if this mortality differential is instead attributable to the physiologic effect of oppressive factors, such as racism, that may comingle with multiple ACEs (Barr, 2014; Krieger, 2001). Additionally, despite increased risk of exposure to multiple ACEs and a higher proclivity of experiencing neighborhood deprivation, it would be helpful to explore if BIPOC individuals may possess particular cultural, social, or religious influences that have a protective effect on engagement with health risk behaviors over the life course, particularly during critical developmental periods in youth (Park et al., 2018).

**Future Research**

There are important next steps in better understanding the full health effects of neighborhood deprivation, social inequity, and available resources. Because the indirect effect of NDI on the relationship between multiple ACEs and HRBs is small, it would be helpful to explore if including the most common ACE-related health effects along with health risk behaviors would increase this indirect effect, such as including poor self-rated health, diabetes, heart disease, mental illness, and early mortality (Ellis, 2017; Felitti et al., 1998). Future research would benefit from exploring this relationship.

Additionally, NDI acts as a composite measure that emphasizes the physical and economic barriers within the environment (i.e. poor housing conditions, income level, etc.), but it does not measure neighborhood-level social components, such as social connection/cohesion among residents, feelings of safety, or norms, which are important aspects of health (Roux & Maira, 2010). While the material and social attributes of a
neighborhood can go hand in hand, it would be helpful to explore how an individual’s social connections within a neighborhood, if perceived as strong, can interact or buffer some of the physical barriers within the environment. By factoring in social resources, this also presents a more holistic understanding of one’s neighborhood experience and available assets versus solely focusing on stressors such as deprivation. Because high PSS buffered the effects of multiple ACEs on HRBs but not the pathway between NDI to HRBs, it is possible neighborhood-level social support may be a better resource in moderating this pathway. There is evidence to indicate social networks play an important role in health behaviors and are responsible for about 10% of the SES gradient in health behaviors (Pampel et al., 2011, p. 363). For example, researchers have noted that low SES is not predictive of obesity levels when community social cohesion is perceived as high, and that individuals in communities with strong religious social capital have reduced severity of some health risk behaviors, like smoking (Pampel et al., 2011).

In continuing to piece apart how social inequity contributes to heightened ACE-related health risks, it is imperative for future research to prioritize marginalized populations most affected by ACEs. Recent research has shown women, people of color, and LGBTQ-identifying individuals have a much greater likelihood of being exposed to multiple types of adverse childhood experiences, which then increases the likelihood of facing significant resulting health disparities (Giano et al., 2020). It seems plausible that these increased exposures and worsened outcomes are partly due to embedded socially oppressive factors that increase stress exposure and can exacerbate child trauma (Barr, 2014). Attending to the varying intersectionality of an individual – understanding how aspects of race, sex, sexuality, and SES all comingle with ACE exposure – is necessary in
order to facilitate effective and efficient community and individual prevention and supports for ACEs. Among those exposed to childhood trauma, it would be helpful to examine if experiences such as discrimination and segregation further influence adulthood health outcomes. This way, researchers can continue to disentangle and “dismantle” the deeper layers of socio-structural influence, extending the current investigation’s contributions (Bowleg, 2017, p. 679). Without examining these factors as potential moderators of health disparities we risk treating systemic oppression as “neutral” in the ACEs health gradient.

As an example of this, it is uncertain if urban and rural NDI function similarly among those with ACE exposures, and if the type of deprivation differs depending on race, as this was not teased apart in the NDI analysis. Previous research indicates urban and rural NDI tend to have differential mediation effects on health depending on race, because, for example, Black individuals face higher rates of segregation in urban settings, while Native Americans often face difficulty accessing culturally competent care in more rural settings (Wong et al., 2020). By using a more complex lens to analyze the effects of potential mediating social inequities, we can achieve a better understanding of why these groups are exposed to higher rates of ACEs and have worsened impacts instead of controlling for these differences.

Lastly, in addition to ushering in a more integrated framework for conceptualizing ACEs, an important byproduct of this work involves a call for the development or application of new theoretical perspectives specific to child trauma that more explicitly identify and cultivate resources and assets. This includes incorporating a more culturally focused lens that attends to the potential protective aspects of one’s culture, as this is a
neglected area within ACEs research. Theory lays the groundwork for rendering visible the systemic processes that give rise to, maintain, and exacerbate issues such as child trauma. It also helps cultivate new perspectives with which we approach research, craft hypotheses, collect data, and develop interpretations. Theory that pulls from strengths-based and asset-based approaches can offer a transformative standpoint to ACEs that aims to shift deficits-based terminology and ideology, while emphasizing the discovery of existing resources within communities. Through this, we have a more holistic image of individuals and their surrounding systems that offers a new direction for this research, which is central to both preventing and understanding child trauma.

**Implications for Social Work Practice**

The findings from this study support specific social work practice implications around neighborhood deprivation and social support. First, interventions specific to neighborhood deprivation will likely require multifaceted approaches targeted across the life course that tackle both individual and mezzo-level factors associated with poverty. For example, among youth exposed to child trauma, focusing on providing quality educational opportunities and reducing school dropout may be a potent force in interrupting the pathway by which ACEs, operating through neighborhood deprivation, contribute to worsened health (Font & Maguire-Jack, 2016, p. 398). Similarly, in adulthood, efforts for maintaining employment, sourcing jobs with living wages, and providing opportunities for career advancement may be critical in altering this trajectory (Font & Maguire-Jack, 2016).

However, attention must also be focused on identifying assets and cultivating community-based approaches within neighborhoods that have multiple, chronic stressors,
such as increasing the availability of affordable, quality housing. Along with this, as mentioned previously, federal policies addressing factors associated with poverty will also be necessary to reduce the toxic stress and reduced health opportunities within poor neighborhoods. These may include instituting a living wage, targeting gender pay inequity, making higher education more affordable, and offering a more comprehensive social safety net for families (Cox, Tice, & Long, 2016). Policies devoted to interrupting the contribution of institutional oppression for marginalized groups may work to prevent disproportionate exposure to ACEs and resulting health issues.

The findings from this study also demonstrate the benefit of providing social support opportunities for those exposed to child trauma. Implementing social support approaches in clinical work could potentially bridge intervention and preventative action, including targeted approaches during critical developmental periods in youth (Bellis et al., 2018), as well as interrupting the intergenerational transmission of ACEs (Narayan et al., 2021). Because youth is the most common period for the initiation of ACE-related HRBs, which can become more entrenched in adulthood, targeting social support interventions in childhood or adolescence may be most effective (Jones et al., 2018). Studies indicate that among young children exposed to trauma, promoting strong, supportive social relationships as early as 5 years old leads to a reduced likelihood of engagement with risky behaviors and a reduced likelihood for experiencing additional adverse events (Narayan et al., 2021). While specific types of PSS approaches need to be tested for efficacy, finding ways to build social support into schools may be opportune because of this unique window in youth, as well as the ability to reach a wider number of individuals. Approaches may include mentorship programs, teacher relationships, or therapeutic
relationships. Additional types of social support that have been found to be successful in child cohorts include providing opportunities to make friendships, having access to a trusted adult, and cultural engagement (Bellis et al., 2018).

Another critical benefit of creating and implementing evidence-based social support approaches in clinical social work is that they have the potential to address the intergenerational transfer of risk for ACEs. Among parents with multiple ACEs, PTSD is a known mediator of the intergenerational transmission of child trauma (Narayan et al., 2021), and lack of PSS is the biggest risk factor for the development and chronicity of PTSD symptoms (Ioannou et al., 2019). Conversely, high social support is known to buffer the effects of PTSD. Conceptualizing social support as a tool that serves both intervention and prevention purposes acknowledges and attends to this broader cycle of childhood trauma.

However, it is important for clinicians to apprehend some of the possible challenges to existing social support networks among those with multiple types of trauma. For example, ACEs can create a trajectory of maladaptive coping and interpersonal dysregulation starting in youth, such as a greater likelihood for interpersonal conflict and reduced support systems, including smaller support networks (Jones et al., 2018). Additionally, research shows individuals in high stress situations can have difficulty accessing social supports or may “underestimate” the supports they have available both in efficacy and number because of the traumatic situation (i.e. perception that the supports may not know how to handle or understand the intensity of the trauma) (Ioannou et al., 2019, p. 7). To better address these challenges, when an individual has a history of child trauma clinicians may find utility in social support network mapping to locate existing
supportive relationships, identify the types of support the individual currently experiences within their network, and areas where social support can be built. This mapping has been specifically used to reduce engagement with risky behaviors, although not specifically related to child trauma (Columbia University, 2015). Future research may benefit from developing specific evidence-based practices for increasing social support among those with a history of childhood trauma.

**Limitations**

There are several limitations to the current study. First, ACEs survey questions are “retrospective and self-reported” and thus have the potential influence of recall bias (Bellis et al., 2018, p. 10). However, research indicates recall bias error tends to be related to underreporting traumatic experiences versus incorrectly endorsing a trauma that did not occur (Ege et al., 2015; Hardt & Rutter, 2004; Mersky, Topitzes, & Reynolds, 2013). Relatedly, because this study is not able to assess the duration or extent of an individual’s trauma, as this is not included in the ACEs measure, this study may underestimate an individual’s experience with ACEs. Further, this study uses cross-sectional data, meaning findings can only be interpreted as indicating correlation not causation (Burke et al., 2011; Mersky, Topitzes, & Reynolds, 2013).

Because BRFSS participants are included at random from a specific geographic area of Washington State, this does not guarantee the full spectrum of disadvantage. For example, it makes it difficult to ensure this dataset captures the vast experiences of rural and urban deprivation that vary depending on region throughout the United States (Messer et al., 2006). Additionally, since the NDI was merged with BRFSS data, the assigned NDI value acts as a geographic proxy for the individual’s current living
situation and is only an estimate. Because the NDI is reliant on census tracts, converting these to corresponding BRFSS zip code may not fully match a person’s defined neighborhood context and resulting stressors (Messer et al., 2006). Since the NDI was created using the ACS from 2013-2017, there is a slight gap in timing between when BRFSS data was collected in 2010 and the NDI’s assessment of zip codes throughout the United States, which may or may not affect the approximation of deprivation. For example, if a participant’s zip code experienced a dramatic shift in SES over the course of those seven years, such as through disinvestment, the assigned NDI value may be inaccurate, altering findings. Thus, hypotheses may need to be reanalyzed with a more current dataset to check the stability of these results. Additionally, the Perceived Social Support variable is measured using only one indicator, whereas a more ideal global measure would contain multiple indicators to gain the most accurate sense of someone’s social support system. Because this dataset only contained a small percentage of BIPOC individuals, future research is recommended to better parse out the impact of race/ethnicity on resulting health risk behaviors among those with multiple types of child trauma.

Finally, because measurements of health risk behaviors are current (i.e. most questions only ask about engagement in HRBs in the last 30 days) instead of taken over the course of an individual’s growth and development, this data likely underestimates HRBs. For example, individuals who have previously engaged in HRBs but no longer do would not be captured in this data. Taken together, the aforementioned limitations likely resulted in low model description ($r^2 = 4.5\% - 6.2\%$). Thus, longitudinal investigations
that are able to track how ACEs result in health risk behaviors across the life course may improve upon this aspect.
Chapter 6: Conclusion

The findings in this study point to the necessity of researching and framing ACEs within “an integrated “stress and resource” framework” in order to better understand, prevent, and treat disparities in health resulting from multiple exposures to child trauma (Wickrama et al., 2015, p. 1119). While ACEs research typically emphasizes the likelihood of experiencing health issues, combining this with potential avenues for resources helps clarify what supports are helpful, as well as what specific pathways they buffer, engendering more tailored interventions. As this research indicates, multi-faceted, intersectional efforts are needed for those with multiple exposures, as a variety of individual and community factors contribute to differential outcomes.

This study also reveals that part of creating a more integrated ACEs research model that better attends to multiple ACE exposures requires being able to account for how “place” and its embedded oppressive components comingle with childhood trauma, affecting these stress pathways, especially in poor communities (Bruner, 2017). This is integral to having a more accurate understanding of the impact of trauma and multiple traumas. Thus, these findings create a more nuanced understanding of individual-level explanations of ACE-related outcomes and encourage movement toward a multi-level biologic and socio-structural model as implicated in ecosocial theory (Krieger, 2001). A multi-level approach has importance in defining new strategies to handling the prevention and intervention of childhood trauma and its resulting ill health. Instead of limiting approaches to individual behavioral interventions – which tend to be less effective because they are absent of “contextual influences of life in low-resourced communities” –
targeting the multifaceted aspects of deprivation creates multiple potential points of
change within neighborhoods (Laraia et al., 2012, p. 10).

Further, the impact of neighborhood deprivation highlights the necessity of
coordinating care within the context of one’s community (Ellis, 2017). Researchers have
found that one of the most significant barriers to “engaging and empowering parents and
community members” in efforts to buffer ACEs is the perceived disregard of the
pervasiveness of neighborhood stressors in shaping low-income families’ lives (Ellis,
2017, p. S91). Addressing deprivation with community-wide initiatives shifts the focus to
prevention or upstream approaches focused on social determinants of health, while also
externalizing blame and validating the experiences of those in the community (Ellis,
2017). Not only is this approach more efficient in comparison to intervention efforts that
occur after behaviors are already initiated, it also addresses the intergenerational
transmission of ACEs that are further complicated and facilitated by the toxic stress of
poverty (Ellis, 2017). Many neighborhood social inequities that pose stress across one’s
life and contribute to disease development can be improved through policy changes,
including resource redistribution, employment opportunities, and housing improvements
(Roux & Mair, 2010, p. 126). By specifically identifying that the components measured
within the NDI compound ACE-related health risk for those with multiple ACEs, this
helps clarify which factors to target first in reducing resulting behavioral risks (Roux &
Mair, 2010).

Addressing multidimensional poverty-related stressors while simultaneously
locating resources within the community that protect against multiple ACEs further
broadens this coordination of care and enables community resilience, a requirement for
ACE prevention (Ellis, 2017). This study’s findings indicate the substantial benefit of high perceived social support regardless of the number of ACE exposures, which offers opportunity to bolster existing community social supports/resilience in creative ways. These social supports may include community centers, cultural connection, engagement with a religious community, friendship networks, or mentorship programs (Bellis et al., 2018). Because social support seems particularly beneficial during critical developmental periods in youth when health risk behaviors are more likely to be initiated, finding ways to connect schools with social support avenues may also prove effective in reaching a wider swath of individuals (Bellis et al., 2018).

While this study demonstrates that the impact of exposure to multiple ACEs on health risk behaviors remained steady regardless of race/ethnicity, future research must explore the stability of this influence when other physical health correlates are included. Further, it will be important for researchers to use a more direct assessment of the moderating influence of racial inequity, such as experiences of daily and/or major discrimination, in order to fully understand its relationship with ACE-related health. In doing so, researchers can help conceptualize health differences in terms of socio-structural contributors rather than maintaining the idea that ACE-related health is solely the “property of individuals and the result of individual behaviors” (Bowleg, 2017, p. 678).

Lastly, in addition to ecosocial theory, the findings of this study encourage a life course, cumulative stress approach to better understanding multiple ACEs and overall health (Ellis, 2017). Life course perspective has the objective of interpreting health by “understanding how the past influences the present” to identify critical periods of
developmental impact, as well as how a person’s exposure to different risks and resources accumulate and shift health trajectories (Burton-Jeangros et al., 2015, p. 2). Applying this perspective to multiple ACEs reveals the complexity of ACE-related health: that assessing the cumulative effect of numerous stressors over one’s life in addition to multiple childhood traumas, as well as the presence or lack of buffers, such as a supportive, consistent relationship, can shift ACE outcomes (Bruner, 2017; Burton-Jeangros et al., 2015). Chronic, cumulative stress, which is common for individuals with multiples exposures to ACEs and for those in highly deprived environments, appears to be more damaging than isolated stress occurrences, as it acts as a “chain of risk” for multiple health issues, including health risk behaviors (Lampert et al., 2015, p. 269; Burton-Jeangros et al., 2015). The earlier stressful experiences occur, such as for those with child trauma, there are “increasingly diverging patterns in health trajectories” (Burton-Jeangros et al., 2015, p. 7). Thus, life course perspective helps us understand that without examining the dynamic interplay and accrual of trauma, environmental circumstances, and available resources, we risk misinterpreting trauma-related health behavior among those with multiple ACEs.
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