Extended Abstract

Adverse Childhood Experience and Chronic Health Conditions in Adulthood: Examining the Intersections of Race/ethnicity, Gender, and Adult Socioeconomic Status

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Introduction

A growing body of studies highlight that many adult diseases and health disparities in adulthood are rooted in childhood adversities (Hayward and Gorman 2004; Turner, Thomas and Brown 2016). For instance, although cardiovascular diseases (CVDs) are usually manifested in adulthood, studies demonstrate that CVDs are, in fact, causally linked to childhood adversity and disadvantage (Hamil-Luker and Angela 2007). A growing body of studies has shown that stressful and traumatic life events experienced during sensitive periods of developments can increase the risk for chronic conditions in adulthood (Miller et al. 2011). Studies report that childhood adversities are associated with premature mortality and an array of chronic conditions in adulthood (Brown et al. 2009; Danese and McEwen 2012; Felitti et al. 1998). These studies find a dose-response relationship such that the likelihood of early mortality and chronic conditions in adulthood increases exponentially with the number of adverse childhood experiences.

The prevalence of ACEs is pervasive in the United States. The 2016 national survey of children's health found that about 46 percent of children under the age of 18 experienced at least one form of ACEs and 22 percent children experienced 2 or more ACEs in the United States (Bethell et al. 2017). The most common forms of ACEs experienced by children were parental divorce/separation (25%), followed by living with anyone with alcohol or drug problem (9%), and parent served time in jail (8%). According to CDC, an estimated 3.4 million children in the United States experienced any form of parental neglect and physical abuse in 2012 (CDC 2014). The lifetime cost of such child maltreatment is estimated to be $124 billion each year (CDC 2014). Evidence suggests that the adult victims of child abuse are at higher risk of developing chronic medical conditions including cardiac events, hypertension, diabetes, and asthma (Anda et al. 2008; Felitti and Anda 2010; Friedman et al. 2015; Gilbert et al. 2015).

The incidence of ACEs is not randomly distributed. Studies have shown that the burden of some ACEs varies starkly by race/ethnicity and gender. Concerning gender, studies find that women report more chronic stress, recent life events, and daily stressors than men (Matud 2004; Turner and Avison 2003). Globally, self-reported child sexual abuse is more common among female than male respondents (Stoltenborgh et al. 2011). Past research examining race/ethnicity has found that black adults report the higher prevalence and a higher number of ACEs than white adults (Reinert et al. 2015; Sternthal, Slopen and Williams 2011). A recent analysis of the 2011-12 National Survey of Children's Health has shown that Hispanic and black children of U.S. born parents were exposed to a higher number of adversities than white children. Children from racial/ethnic minorities were disproportionately exposed to certain adversities such as financial hardships, having an incarcerated parent, being exposed to parental domestic violence, being exposed to neighborhood violence, and experiencing unfair treatment due to race/ethnicity (Slopen et al. 2016). The analysis has also shown that racial/ethnic disparities in childhood

adversity persist at every level of income. The racial differences in stressful and traumatic life events are also implicated in how they lead to health disparities in adulthood (Jackson, Knight and Rafferty 2010).

Although a growing body of studies documents the association between childhood adversities and adult health outcomes, previous studies are limited in many ways. First, although the earlier studies pioneered the extraordinary effects of ACE on health, those studies offer limited utility for identifying a specific population at increased risk for psychosocial adversities, as they do not represent a diverse population (Felitti et al. 1998; Gilbert et al. 2015). This makes effective population-level health policy planning especially difficult.

Second, existing studies on inequalities in chronic conditions employ a unidimensional approach which implicitly assumes that race/ethnicity and gender identities are separate and additive axes of social inequality. Most studies traditionally use additive multivariate analysis privileging one gender over another and one race over another with articulating the view that health inequalities grow as a result of consequences of additive effects of social stratification categories. Such additive approach fails to unpack how social inequality indicators may constitute simultaneous, mutually reinforcing, interlocking, and multiplicative lines of inequality in health outcomes (Brown et al. 2016a; Richardson and Brown 2016). Understanding these interlocking and multiplicative aspects of inequality in chronic conditions holds strong potential to translate the insights of social inequality to health services research and population health interventions tailored to group differences and needs of diverse population groups.

Third, although most studies control for gender and race/ethnicity, these studies have inadequate or no theoretical framework explaining the gender and racial/ethnic differences in ACE and health. Most studies tend to overlook the theoretical justifications as to how the structural position of racial and ethnic minorities in the U.S. may disproportionately expose them to ACE. Further, few studies have theoretically elucidated how psychosocial stress and behavioral pathways that impact adult health may operate differently for intersecting categories of gender and race/ethnicity. Except for a few recent studies, most studies do not frame the research question linking ACE to chronic conditions from an intersectionality perspective (Brown et al. 2016b; Keith and Brown 2018; Richardson and Brown 2016; Veenstra 2011). In particular, while intersectionality has emerged as a dominant theoretical approach in the studies of gender and identity, it is less commonly used in the areas of longitudinal effects of stress.

Fourth, although researchers generally recognize the importance of early life adversities, there is a considerable level of disagreement concerning the relative importance of different types of stressors. For instance, it is relatively unclear what the most salient stressors are and how stress exposure varies depending on the type of stressors. Most importantly, there are very few studies that demonstrate how stressors are structured by broader structural factors such as race/ethnicity and gender (McEwen and McEwen 2017; Sternthal, Slopen and Williams 2011). In particular, much prior work on early life adversities and health focuses on childhood SES. Except for a few notable past works (Anda et al. 1999; Felitti 2009; Felitti and Anda 2010), less attention has been devoted to examining the role of various types and counts of psychosocial stressors encountered in childhood on adult chronic conditions. More importantly, most studies use a limited range of stressors and overlook how different types of stressors, both individually and collectively, relate to social structure and shape the stress-health associations (Sternthal, Slopen and Williams 2011).

Life course stress process perspective

One of the critical contributions of the stress process model is its conceptualization of the stress proliferation processes over the life course. The concept of 'stress proliferation' emphasizes that serious stressors, whether in the form of acute life events or chronic strains, beget additional stressors (Pearlin et al. 2005). The process of stress proliferation may unfold in many ways. For instance, the stressors may proliferate from one generation to the next and one life domain to another domain (e.g. from loss of parents to child's financial strains, from parental neglect to child's substance use, from parental incarceration to child's financial strains, substance abuse, and social stigma) (Thoits 2010; Umberson et al. 2005). Again, the stressors can entail multiplicative and accumulative processes. For example, childhood trauma and stressors can lead to stressful life experiences in adolescent and young adulthood, which in turn lead to further stressors in later adulthood (Thoits 2010). Studies report that childhood adversities negatively affect adult mental health through direct, indirect, and accumulative processes (Turner and Avison 2003; Turner, Thomas and Brown 2016).

Life course stress process is a theoretical framework that links early life stressful conditions with later health (Elder, Johnson and Crosnoe 2003). The life course perspective is based on the central premise that early life conditions are temporally linked with and have a long-lasting impact on later health. The stressors and stress proliferation are the two primary concepts in the life course stress process model. Stressors are defined as the "circumstances and experiences to which it is difficult to adjust and, therefore, that can impose deleterious effects on emotions, cognition, behavior, physiological functioning, and well-being" (Pearlin and Bierman 2013:326). Regarding the linkages between early life conditions and later life health trajectories, the life course stress process model offers two distinct but complementary explanations: the critical period model and the accumulation of risk model.

The critical period model theorizes that adversities experienced during critical periods including utero environment, early postnatal, infancy, and childhood have a lasting signature on the future trajectories of health. The critical period implies exposure to adversities during these critical periods increases the susceptibility to certain diseases through a process called 'biological imprinting' or 'scarring.' Studies have shown that toxic stress in young children has the potential to disrupt the developing brain architecture and lead to functional differences in memory, cognition, and linguistic and socio-emotional skills development (McEwen 2005; McEwen 2006).

The accumulation of risk model assumes that individuals accumulate the social and biological risks across the life course. In other words, adversities and insults gradually accumulate and work as significant risk factors for future health trajectories. Sociological research highlights that early life events have the potential to trigger these chains of risk. Specifically, childhood socioeconomic status is thought to carry a lasting signature on the pathways to adult health trajectories through influencing exposure to stressors, psychosocial resources, and adaptation capacities (Umberson et al. 2014). The exposure to ACE may result in toxic stress that has the potential to lead to poor health even a decade later. Research has shown that the stressful and traumatic events experienced in early life become biologically embedded that these events can set children on a long-term trajectory of increased risk for an array of chronic conditions including cardiac events, diabetes, and asthma (Shonkoff et al. 2012).

Using the life course principle of 'accumulative risk,' many scholars argue that traumatic and stressful life events experienced in childhood may work as 'a cumulative risk' and contribute

to the cumulative damage on biological system and gradually increase the risk of morbidity and mortality later in life (Ben-Shlomo and Kuh 2002). In other words, the 'chains of risk model' posits that one adverse exposure can lead to another adverse outcome. Consistent with this theoretical perspective, a large body of studies document why individuals who grew up in poverty, lived in stressful childhood circumstances and are exposed to significant forms of family dysfunctions are more likely to engage in risky health behaviors and have elevated levels of anxiety and depressive disorders (Chapman et al. 2004).

Stressful life events in early life can inflict an enduring biological imprint on health trajectories across the life course. The 'biological embedding process' implies that cardiovascular system and stress-response physiological systems are profoundly vulnerable to chronic activation of childhood adversities in such a way that chronic stress results in higher levels of allostatic load, secretion of stress hormones, and a host of cardiometabolic profiles that have been shown as the strong predictors of chronic conditions such as heart disease (Danese et al. 2009; Gilbert et al. 2015).

Racial disparities in health: Cumulative disadvantage

Racial differences in health in the United States are well-established. Black Americans fare worse on health compared to whites in the United States. Regarding the racial differences in health over the life course, life course scholars offer two overarching hypotheses, often complementary to each other: cumulative disadvantage and weathering hypothesis. The cumulative disadvantage hypothesis argues that socioeconomic and racial differences in health grow over time as a result of the differential exposure to risk factors associated with poorer SES and access to social support and protective resources (Dannefer 2003; DiPrete and Eirich 2006).

The cumulative disadvantage hypothesis implies that childhood stressors and adverse exposures can accumulate and build over the life course. The cumulative disadvantage is manifested in how racial minorities have an unfavorable position in the status hierarchy, and they face a persistent material disadvantage compared to whites (Ferraro, Schafer and Wilkinson 2016). One crucial example of material disadvantage is the racial residential segregation. Segregation confines children of racial minorities to worse environments characterized by chronic poverty, low property values, environmental hazards, and neighborhood stressors that set children on a lifelong trajectory of social inequality. These pathways of psychosocial stress result in racial disparities in physical and mental health that widen over time (Massey and Denton 1993; Shuey and Willson 2008; Williams and Collins 2001).

Consistent with the cumulative disadvantage hypothesis, Geronimus, Hicken, Keene & Bound (2006) proposed a "weathering hypothesis" which posits that racial minorities are more likely to experience early health deterioration as a consequence of their higher burden of cumulative social, economic, and political marginalization. Geronimus and her colleagues argue that "on a physiological level, persistent, high-effort coping with acute and chronic stressors can have a profound effect on health" (Geronimus et al. 2006:826). They continue to argue, "…the stress inherent in living in a race-conscious society that stigmatizes and disadvantages blacks may cause disproportionate physiological deterioration, such that a black individual may show the morbidity and mortality typical of a white individual who is significantly older" (Geronimus et al. 2006:826).

Gender and health

Although women tend to live longer than men, women have more disabilities and morbidities. Women tend to suffer more from acute and non-fatal chronic conditions (e.g. arthritis) than men and women's longer life is characterized by more functional limitations and poor health conditions (Read and Gorman 2006; Rieker, Bird, and Lang 2008). On the other hand, men have more life-threatening conditions (e.g., heart disease) than women and are at higher risk of accidental death or injury. These differences in men's and women's physical health are considered as a gender-health paradox. Central to the gender-health paradox is the argument that although women live longer than men, i.e., they have lower mortality, they also report higher morbidity in the form of poorer self-rated health and greater use of health services (Case and Paxson 2005; Read and Gorman 2010).

Women's excess morbidity is attributed to acute (e.g., lower respiratory diseases, headache, etc.) and non-fatal chronic conditions (e.g., arthritis and disability). Studies have shown that women's excess morbidity is partly caused by their longer length of life (Read and Gorman 2010). Compared to women, men at relatively younger ages engage in risky health lifestyles (e.g., heavy drinking, illicit drug use, smoking) that diminish their life chances (Read and Gorman 2010). Men's health-damaging lifestyles have a cumulative negative impact on their health conditions, mainly as those health lifestyles cause them to die prematurely from life-threatening conditions such as heart diseases and diabetes (Case and Paxson 2005; Read and Gorman 2010; Rieker, Bird, and Lang 2008).

Existing research on gender differences in health offers a combination of biological, socio-structural, behavioral, and psychosocial explanations. Biological explanations suggest that physiologically women are more robust than men. For example, the female sex hormone 'estrogen', contrary to 'testosterone,' influences patterns of aging as it helps maintain muscle strength and lower the risk of cardiovascular diseases (CVD) by reducing the circulation of harmful cholesterol. Therefore, postmenopausal women tend to have an increased risk of CVD, equal to men of the same age (Read and Gorman 2010; Regan and Partridge 2013). However, these biological explanations have little insights to offer in explicating why sex differences in health significantly vary by social conditions and over the time and place as illustrated in the previous sections.

Sociological explanations focus on men's and women's relative social position and their differential exposure to health behaviors, risk factors, and access to protective material and non-material resources (e.g., income, education, health insurance, social networks). The most common explanation offered in the medical sociology literature is that socio-economic status (SES) profoundly shapes women's and men's health and well-being through various socio-psychological pathways. In general, SES-based explanations posit that individuals of higher SES are more likely to have better health as they have greater access to an array of resources needed to prevent or manage disease conditions when they occur (Link and Phelan 1995; Phelan and Link 2013; Marmot 2008).

A large body of research also documented how health behaviors and coping mechanisms produce gender differences in physical and mental health. Men and women differ in health-damaging or health-protecting practices that make a difference in health/mortality outcomes. For instance, studies have shown that men cope with stress by engaging in heavy drinking, substance abuse, and smoking, while women tend to internalize stress by expressing elevated levels of anxiety and depression. The health-damaging practices among men and higher levels of depression among women are associated with gender differences in the prevalence and types of

chronic conditions (Read and Gorman 2010). Research suggests that clinically diagnosed major depression is associated with increased risk of adult mortality and especially mortality that relates to CVD (Mirowski and Ross 2003; Rieker and Bird 2005). Depression is also associated with decreased immune function and increased disease severity as depression leads to increased pain and inflammation through chemical mechanisms (Rieker and Bird 2005).

The associations between gender and health are mixed once one accounts for gendered patterns in diet, exercise, and violence. Men tend to do more exercise and engage in exercise more regularly than women. Therefore, the health benefits of exercise are found more frequently among men than women. However, men tend to have worse diets compared to women (NCHS 2009; Read and Gorman 2010). Regarding violence, women are more likely to be subjected to sexual and intimate partner violence, most of the time at the hands of men. However, men are more likely to experience injuries and fatalities from drunk driving. Men are also more likely to commit suicide and homicide (Read and Gorman 2010).

The Current Study

Using a nationally representative survey – Behavioral Risk Factor and Surveillance System (BRFSS) and drawing on the integrated framework of the life course stress process and intersectionality perspective, this study examines how gender and race/ethnicity structure the exposure to ACE and vulnerability to chronic conditions. In particular, I investigate two factors: 1. whether the clustering of and exposure to ACE is structured by race/ethnicity and gender and; 2. whether the association between ACE and chronic conditions varies by the intersecting categories of race/ethnicity, gender, and adult SES.

Conceptual Framework

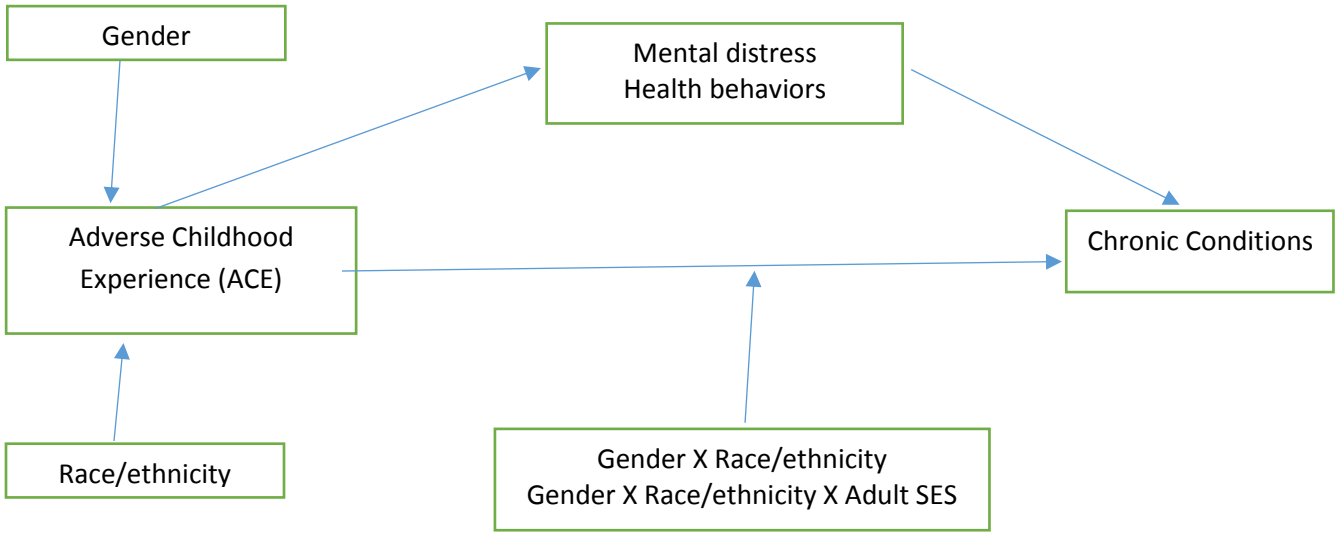

Figure-1: Conceptual framework of the study

Drawing on the integrated framework of life course stress process and intersectionality perspective, I outline that gender and race/ethnicity are the critical structural variables in the model. I hypothesize that the exposure to ACEs would be structured/clustered by these structural

variables. I expect to see some ACEs are disproportionately higher among women compared to men (e.g., sexual abuse) and among minorities compared to whites (e.g., living with household members being incarcerated, witnessing domestic violence between parents, living with substance-abusing and mentally ill household members). ACEs have both a direct and indirect relationship with chronic conditions. These associations partly reflect the life course pathways in which ACEs as early life stressors has a long-lasting impact on later life conditions. Two of the pathways include the process of psychosocial distress and health behaviors which are the mediators in the link between ACE and chronic conditions. Finally, in line with the intersectionality approach, I am hypothesizing that the relationships between ACE and chronic conditions would vary by the intersecting categories of race/ethnicity, gender, and adult SES. I expect that low SES minorities (e.g., blacks and Hispanics) are more likely than low SES whites to have the stronger impact of ACEs on chronic conditions. Again, I expect that the effect of ACEs is stronger among low SES black women relative to low SES white women.

Data and sample

This study uses data from four rounds of the Behavioral Risk Factor Surveillance System (BRFSS) of 2009-2012. The BRFSS is a cross-sectional random-digit-dialed telephone survey annually conducted in the United States. This survey is administered to a probability sample of non-institutionalized adults aged 18 or above in almost all states and its territories in the United States. The core component of the survey consists of basic socio-demographic, health conditions, and health-related risk behavior questions. In addition to a core component, there are several optional modules on different topics. Depending on the state program purposes, different states choose to add some of these optional modules to the core component in their respective states. For examples, two states in 2009 (AR, LA), five states each in 2010 (DC, HI, NV, VT, WI), and 2011 (MN, MT, VT, DC, WI), and four states in 2012 (IA, NC, TN, WI) included the optional module on ACE. This study pooled data from these four years and 16 states in which the ACE module was included in the BRFSS.

Chronic conditions

This study includes a total of 7 indicators of self-reported chronic conditions: three indicators of cardiovascular disease (e.g., heart disease, heart attack, & stroke), diabetes, asthma, obesity, and functional limitation. The count of all conditions is dichotomized and ranged from 0-7. This is count measure.

Adverse childhood experience

The BRFSS ACE module consists of a total of 11 items, representing different types of child abuse (physical, sexual, and emotional) and household dysfunction (e.g., substance abuse, mental illness, domestic violence, incarceration/jail, and divorce/separation). All respondents were asked to retrospectively report whether in their childhood (before age 18) they had ever exposed to these experiences. A count variable of overall ACE is constructed by summing the number of ACE events a respondent had ever experienced.

Race/ethnicity

BRFSS calculated variable has the following 8 race/ethnicity categories: 1) White only, non-Hispanic, 2) Black only, non-Hispanic, 3) Asian only, non-Hispanic, 4) Native Hawaiian or other Pacific Islander only, non-Hispanic 5) American Indian or Alaskan Native only, non-Hispanic, 6) Other race only, non-Hispanic, 7) Multiracial, non-Hispanic, and 8) Hispanic.

Adult SES

Adult SES is measured using three variables: current employment status, educational attainment, and yearly household income. All of these variables are categorical.

Health Behaviors

The analysis uses three health behavior measures: smoking, drinking, and exercise. Following previous work (Jackson, Knight and Rafferty 2010), I used a count variable of negative health behaviors representing whether a respondent is currently smoking, drinking, and not exercising. The score in negative health behaviors ranges from 0-3.

Mental Distress

Mental distress is measured using the following two questions: "Thinking about your mental health, which includes stress, depression, and problems with emotions, for how many days during the past 30 days was your mental health not good?" and "How many days did you not get enough sleep in past 30 days".

Demographic controls

Demographic controls included current age and marital status.

Analytical Strategy

The initial analysis examines the distribution of sample characteristics by running descriptive statistics such as means and percentages of the variables used in analyses. At the bivariate level, the distribution of ACEs by race/ethnicity and gender is examined. Using confirmatory factor analysis, three sub-scales of ACE - emotional/physical abuse, sexual abuse, and household dysfunction are constructed. Along with the overall ACE counts, these three sub-scales are used for testing the hypothesis that racial/ethnic and gender patterns of ACE exposure vary depending on the specific type and count of stressors. Finally, Poisson regression models controlling for the control variables were fitted to test the relationships outlined in the conceptual framework.

Results

This section will follow ASAP.

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