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# Modeling life course pathways from adverse childhood experiences to adult mental health



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## ABSTRACT

Although the association between adverse childhood experiences (ACEs) and adult mental health is becoming well established, less is known about the complex and multiple pathways through which ACEs exert their influence. Growing evidence suggests that adversity early in life conveys not only early impacts, but also augments risk of stress-related life course cascades that continue to undermine health. The present study aims to test pathways of stress proliferation and stress embodiment processes linking ACEs to mental health impairment in adulthood. Data are from the 2011 Behavioral Risk Factor Surveillance Survey, a representative sample of Washington State adults ages 18 and over ( $N = 14,001$ ). Structural equation modeling allowed for testing of direct and indirect effects from ACEs through low income status, experiences of adversity in adulthood, and social support. The model demonstrated that adult low income, social support and adult adversity are in fact conduits through which ACEs exert their influence on mental health impairment in adulthood. Significant indirect pathways through these variables supported hypotheses that the effect of ACEs is carried through these variables. This is among the first models that demonstrates multiple stress-related life course pathways through which early life adversity compromises adult mental health. Discussion elaborates multiple service system opportunities for intervention in early and later life to interrupt direct and indirect pathways of ACE effects.

## 1. Introduction

Experiences of adversity in childhood, such as maltreatment and caregiver dysfunction, are well known to have life-long, often severe implications for mental health. Adverse childhood experiences (ACEs) are also relatively common, with estimates of adult population prevalence of having at least one ACE ranging from 52% to 67% (CDC, 2010; Felitti et al., 1998; Wade et al., 2016). Childhood adversity puts individuals at risk for the development of psychological disorders not only in childhood and adolescence but into adulthood (Green et al., 2010; Nurius, Green, Logan-Greene, & Borja, 2015). More specifically, individuals who have experienced ACEs are at increased risk of depression, anxiety, aggression, suicide risk (Anderson, Tiro, Price, Bender, & Kaslow, 2002; Chapman et al., 2004; Turner, Finkelhor, & Ormrod, 2006), behavioral disorders (McLaughlin et al., 2012), and substance abuse (Mersky, Topitzes, & Reynolds, 2013).

Although association between adverse childhood experiences and later mental health outcomes is now established, we are in early stages of testing complex and multiple pathways through which ACEs operate. Interim stress and social support are known to be important to these pathways, yet few studies have formally tested their mediation (Font & Maguire-Jack, 2016; Raposa, Hammen, Brennan, O'Callaghan, & Najman, 2014). Income status is also associated with ACEs, but the role that low income plays in structuring

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these childhood circumstances and pathways into adulthood is less well understood (Font & Maguire-Jack, 2016). Researchers have called for integrative models to investigate relationships between multiple resource and stress pathways (Wickrama, Lee, O'Neal, & Kwon, 2014). To address these gaps, we set out to formally investigate whether adult adversity, social support, and adult income status serve as mediators. The present study integrates theories of stress embodiment and stress proliferation, and tests associated psychosocial mechanisms to create a snapshot of cumulative life course adversity effects that are catalyzed by early life exposures to ACEs (Maschi, Baer, Morrissey, & Moreno, 2013; Moos, Schutte, Brennan, & Moos, 2005).

Stress is a consequence of early adversity as well as a risk factor for subsequent mental health problems, a reciprocal relationship whereby stress begets additional stress (Hazel, Hammen, Brennan, & Najman, 2008; Moos et al., 2005). Continuous exposure to stress degrades physiological resources (Wickrama et al., 2014), and is associated with increased allostatic load. Allostatic load is an index of physiological dysregulation, and prolonged exposure to stress increases stress hormone levels and hypothalamic–pituitary–adrenal (HPA) axis dysregulation (Doan, Dich, & Evans, 2014). Adults who experienced child maltreatment have been shown to have increased levels of inflammation and HPA-axis activation (Danese & McEwen, 2012), and increased inflammation biomarkers distinguish individuals who experienced child maltreatment and were depressed from those who were depressed without maltreatment (Danese et al., 2008). Toxic stress resulting from ACEs can cause physiological dysregulation that can lead to impairments in learning and the ability to adapt to new adversities, and sets the physiological stage for stress-related health and mental health problems (Shonkoff et al., 2012).

Risky social environments mold coping styles, emotion regulation and social cognitions, all of which are involved with shaping stress responses (Hager & Runtz, 2012; McCrory, Dooley, Layte, & Kenny, 2015; Raposa et al., 2014). These impairments limit success in systems such as education and the workforce as well as undermine healthy social development, increasing the risk of subsequent exposure to environmental stressors, the perception of stress, and reactions that escalate stress or complicate adaptive coping. Thus, these social environmental effects likely have biological underpinnings that can be explained through the effect of toxic stress on physiological systems.

### 1.1. ACEs and low income in adulthood

Although an association between childhood adversity and lower income in adulthood has been documented, less has been established about the role of adult economic disadvantage in the association between ACEs and adult mental health outcomes (Font & Maguire-Jack, 2016). While income is a strong determinant of physical and mental health, its effects are largely indirect, operating through differential exposure to adverse conditions (Adler & Newman, 2002). Adverse exposures tend to co-vary, be cumulative in nature, and undermine cognitive, emotional, and behavioral development (Edwards, Holden, Felitti, & Anda, 2003; Hankin, 2005). Households with low income may struggle to provide adequate nutrition and supportive parenting, and structural constraints make health care access and choosing healthy behaviors more difficult, leading to diminished emotional resources (Wickrama, Conger, & Abraham, 2005).

Moreover, the social contexts in which childhood adversity occur are rarely time-limited. Higher levels of early life adversity tend to be associated with relatively persistent structural contexts that elevate risk that children will be exposed to subsequent stressful experiences, resulting in a continuity of stress exposures (Pearlin, Schieman, Fazio, & Meersman, 2005; Raposa et al., 2014). ACEs can also influence social contexts more proximal to traumatic events, so that adversity impacts one's school outcomes, leading to lower educational attainment, and lower socioeconomic achievement in adulthood (Raposa et al., 2014). Economic disadvantage is also a factor in influencing the quality and depth of one's social network, as well as social cohesion in the broader environment (Adler & Newman, 2002). Previous studies have documented the detrimental effects that ACEs have on adult socioeconomic achievement (Font & Maguire-Jack, 2016).

### 1.2. Adult adversity

In order to better understand the effects of early life adversity, more attention to processes of stress accumulation is needed. As noted, the effect of childhood adversity is not limited to childhood, but leads to accumulation of stressors across development, in a chain reaction of adversity (Shrira, 2012; Turner & Butler, 2003). Exposure to adversity may result in a pessimistic explanatory style and cognitive attributional biases which lead to depression vulnerability (Rutter, Kim-Cohen, & Maughan, 2006). Growing up in negative social environments likely influences the selection of similar social environments in adulthood (Raposa et al., 2014). Adults with psychopathology have higher levels of ACEs as well as more high-risk behaviors, substance use disorders, exposure to trauma in adulthood, psychiatric problems, medical service utilization, incarceration and homelessness (Lu, Mueser, Rosenberg, & Jankowski, 2008). In addition, these experiences of stress proliferation are known to operate through biological pathways by increasing HPA-axis reactivity resulting in neurobiological dysregulation (Juster, McEwen, & Lupien, 2010).

### 1.3. Social support

Significant evidence supports the critical role that social support plays in protecting against mental health problems and in buffering stress exposure (Thoits, 2011). Social support may act to buffer stress by providing coping assistance and emotional sustenance (Thoits, 2011). Social contexts in which ACEs occur are often associated with poor systems of social support. This may be because experiences of adversity implicate parents or caregivers, or due to loss of support related to the adversity itself; i.e., parental death or separation, relocation etc. (Turner & Butler, 2003). ACEs are also associated with unhealthy lifestyles characterized by more

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