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*Child Abuse Negl*. Author manuscript; available in PMC 2016 July 01.

Published in final edited form as:

*Child Abuse Negl*. 2015 July ; 45: 143–153. doi:10.1016/j.chiabu.2015.03.008.

# Life Course Pathways of Adverse Childhood Experiences Toward Adult Psychological Well-Being: A Stress Process Analysis

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## Abstract

Growing evidence suggests that toxic stressors early in life not only convey developmental impacts but also augment risk of proliferating chains of additional stressors that can overwhelm individual coping and undermine recovery and health. Examining trauma within a life course stress process perspective, we posit that early childhood adversity carries a unique capacity to impair adult psychological well-being both independent of and cumulative with other contributors, including social disadvantage and stressful adult experiences. This study uses data from a representative population-based health survey ($N$ = 13,593) to provide one of the first multivariate assessments of unique, cumulative, and moderated effects of adverse childhood experiences (ACEs) toward explaining 3 related yet distinct measures of adult mental health: perceived well-being, psychological distress, and impaired daily activities. Results demonstrate support for each set of hypothesized associations, including exacerbation and amelioration of ACEs effects by adult stress and resilience resources, respectively. Implications for services and future research are discussed.

### Keywords

trauma; adverse childhood experiences; stress; victimization; mental health

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## Life Course Pathways of Adverse Childhood Experiences Toward Adult Psychological Well-Being: A Stress Process Analysis

Exposure to significant childhood adversity affects a daunting proportion of young people (Anda et al., 2006; Dube et al., 2001), constituting one of the most detrimental impacts on youth development (Kilpatrick, Saunders, Smith, 2003; Widom, 2000). Early life adversities include experiences such as maltreatment, neglect, witnessed violence, and household dysfunctions such as parental mental illness or substance abuse, and incarceration of one or more family members. Evidence increasingly indicates that diverse childhood adversities often co-occur (Edwards, Holden, Felitti, & Anda, 2003; Finkelhor, Ormrod, & Turner, 2007), shifting attention from single forms to cumulative exposures (Arata, Langhinrichsen-Rolin, Bowers, & O'Brien., 2007; Dube et al., 2003). Exposure to these events generates step-dose patterns wherein greater exposure to multiple forms of stressful experiences are associated with a wider range of impaired health outcomes, including psychiatric (Dube, Williamson, Thompson, Felitti, & Anda, 2004; Schillilng, Aseltine, & Gore, 2007).

Findings from population-based studies indicate that childhood adversity is common and associated with development of psychological disorders not only in childhood but in adolescence and adulthood (Green et al., 2010; Kessler, Davis, & Kendler, 1997; McLaughlin et al., 2012). More specifically, adverse childhood experiences (ACEs) have demonstrated increased risk of depression, anxiety, aggression, suicide risk (Anderson, Tiro, Price, Bender & Kaslow, 2002; Chapman et al., 2004; Turner, Finkelhor, & Ormrod, 2006), personality disorders (Afifi et al., 2010), behavior disorders (McLaughlin et al., 2012), and substance abuse (Mersky, Topitzes, & Reynolds, 2013; Turner & Butler, 2003). Children exposed to adverse psychosocial experiences demonstrate elevated risk for depression in addition to high inflammation levels and clustering of metabolic risk markers reflecting multiple biological system dysregulations, with the effects of these adversities being nonredundant, cumulative, and independent of developmental contributors (Danese et al., 2009). Childhood adversities tend to be interrelated, creating layered stress, exert damage to various aspects of the developing brain, foster maladaptive health and behavioral habits, and be associated with limited protective relationships (Shonkoff et al, 2012).

Assaults to psychological health are important in their own right, but their tendrils of effects also extend through a panoply of psychosocial and functioning domains; the influence of these underlying effects are often underway before clinical symptoms are manifested (Ferraro & Shippee, 2009). Efforts to understand the longer-term implications of early life adversity are turning to life course approaches that offer perspectives on cumulative adversity. In this regard, useful theoretical tools include the stress-process model, which stipulates points of connection at which ACEs evoke stress responses at the time of the assault, threaten the individual's adaptive capacities, catalyze secondary stressors, and increase likelihood of future stress exposures and resource paucity (Pearlin, 2010; Pearlin, Schieman, Fazio, & Meersman, 2005).

This study builds on prior work through a life course approach to the effects of early life adversity on adult psychological health (Kuh, Ben-Shlomo, Lynch, Hallqvist, & Power, 2003). We draw upon the stress-process model in theorizing ways through which early life

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factors work with later life factors to cumulatively and interactively influence psychological well-being. We posit that socially patterned adversities in childhood are part of life course cascades that influence socioeconomic (SES) position as well as later life stress exposure and stress ameliorating resources. Those patterns are examined through hierarchical regressions that allow differentiation of unique, interactive, and cumulative relationships with three complementary indicators of psychological well-being.

## Embedding ACEs Within Multiple Pathways to Psychological Health

Although resilience is a deeply rooted human strength (Bonanno & Mancini, 2008), the imprint of toxic stressors early in life sets in motion social and biological chains of exposures that, as stressors accumulate, can overwhelm individual coping and available resources to support recovery and health (Min, Minnes, Kim, & Singer, 2013; Taylor & Stanton, 2007). Early adversities such as those assessed through the CDC ACEs measure of household dysfunction and child maltreatment indicate a range of conditions characterized by multiple forms of chronic and acute stress. In addition to negatively affecting stress responding and development (Shonkoff, Boyce, & McEwen, 2009), these exposures may also act as catalysts for subsequent interlocked chains of stress exposures (Anda, Butchart, Felitti & Brown, 2010).

The construct of stress proliferation provides a foundation for operationalizing life course processes of adversity accumulation taking place over time. Individuals with early life circumstances and characteristics that render them vulnerable are more likely to encounter and generate stressful events throughout their youth and into adulthood (Turner & Butler, 2003; Turner & Schieman, 2008), to accumulate stress burden, to experience social and behavioral difficulties, and increase their odds of negative mental health (Aneshensel, 2009; Springer, 2009; Umberson, Liu, & Reczek, 2008). As Min et al. (2013) note, attention to intermediary variables that link ACEs to later adult health is, thus far, relatively limited yet important to identifying critical domains of intervention toward fostering resilience and prevention of later pathology.

Early life adversities thus serve as primary stressors that set the stage for and interact with secondary stressors in the form of further adversities. These proliferative processes flow through multiple life domains (education, work, relationships), linking chains of risk and creating interrelated hardships that connect ACEs and later life outcomes (Ferraro & Shippee, 2008; Pearlin et al., 2005). For example, early life adversity undermines learning and academic achievement, compromising success in adulthood across educational, workforce, and socioeconomic domains (Evans & Kim, 2007; Sansone, Leung, & Wiederman; 2012; Zielinski, 2009). In turn, this undermined achievement creates contexts biased toward exposure to additional social stressors, a paucity of social and personal resources, and adult mental disorders (Turner, 2013; Wickrama, Conger, Surjadi, & Lorenz, 2010). Increased exposure to later adverse life events may take a range of forms such as relationship problems, residential instability, disability, and involvement with the criminal justice system (Larkin & Park, 2012; Lu, Mueser, Rosenberg, & Jankowski, 2008; Nurius, Logan-Greene, & Green, 2012; Schussler-Fiorenza, Rose, Xie, & Stineman, 2014).

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Early exposure to adversity predicts later susceptibility to impaired health and functioning in part through processes that affect the growth and functioning of brain structures and alter neuroendocrinological operations involved in stress response functions, such as the hypothalamic-pituitary-adrenal axis (Nemeroff, 2004). This physiological dysregulation constitutes a biological embedding of stress that carries forward through development (Danese et al., 2009; Hertzman & Boyce, 2010; Jaffee & Christian, 2014). Altered neurobiological processes stemming from childhood adversity appear to increase stress sensitization, suggesting an additional pathway through which ACEs may have developmental origins to adult mental health. That is, growing evidence supports theorizing that early adversity sensitizes individuals to later psychopathology, such as depression, by reducing their tolerance to subsequent, even minor stressors (Hammen, Henry, & Daley, 2000; Harkness et al, 2006). This dysregulation, in turn, may lead to lower levels of stress triggering stress reactivity (McLaughlin, Conron, Koenen, & Gilman, 2010), with increased intensity of response and resistance to treatment (Heim, Plotsky, & Nemeroff, 2004), fostering proliferation of stress and psychological burden in the pathway to adulthood (Hazel, Hammen, Brennan, & Najman, 2008).

Processes of stress proliferation and stress sensitization underscore the importance of resilience-fostering resources, factors that would be expected to buffer stress and encourage healthy behaviors. Social integration and support have been identified among the most important intervention targets to reduce both the biological effects of stress overload and to stimulate more positive cognitive and emotional processing (McEwen & Gianaros, 2010; Thoits, 2011) Yet these protective factors tend to be unevenly distributed. For example, early stressors such as trauma and disordered family life constrain access to healthy social ties and lifestyles needed to build satisfactory social support (Hill, Kaplan, French, & Johnson, 2010; Vranceanu, Hobfoll, & Johnson, 2007). As a result, those exposed to early stressors often lack interpersonal buffers of social stress in later life and experience poorer mental health (Murphy, Shevlin, Armour, Elklilt, & Cristoffersen, 2014; Sperry & Widom, 2013).

Whereas the relationship of elevated stress exposures has been established with health-threatening behaviors such as substance use, risky sex, and overeating, sleep and physical activity are increasing targets of attention for offsetting damage of neurobiological dysregulation. Early life adversity is associated, for example, with difficulty in calming to achieve sleep and with poor sleep quality (Chapman et al., 2011; Strine & Chapman, 2005), remaining significant even after controlling for a range of adult contributing factors (Koskenvuo et al., 2010). These patterns lessen the availability of sleep as a stress buffer and exacerbate effects of current stress (Greenfield, Lee, Friedman, & Springer, 2011). Similarly, physical activity, particularly when social in nature, demonstrates biological, psychological, and social mechanisms though which activity reduces perceived stress, stress effects, and stimulates other adaptive health behaviors (Fleshner, 2005; Rimmele et al., 2009), preventing oxidative stress and anxiety-related behaviors associated with sleep deprivation (Vollert et al., 2011). Insufficiency of both sleep and activity can be pivotal markers of deficits in stress-ameliorative behavior (Wijndaele et al., 2007). We argue the value of sleep and physical activity as mutable, broadly accessible resilience resources,

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bolstering individual defenses against stressors and the reduction of dysregulations such as stress sensitization (Chatburn, Coussens, & Kohler, 2014).

### The Present Study

These proposed life-long cascades of proliferated stressors and muting of resources serve to join with early adversity in a range of forms that influence health independently, interactively, and cumulatively. In this study, we theorize that elevated ACEs reflect multiform stress exposure that jeopardizes psychological health and well-being through multiple life course conduits. Within a representative population-based survey, we formulated three hypotheses. We anticipate that ACEs will demonstrate direct effects on adult mental health regardless of adult conditions wherein unique effects persist in multivariate models. Second, support for stress proliferation pathways will emerge wherein early life adversity is significantly associated with adult conditions; specifically, adult SES characteristics, significant adult adverse experiences, and paucity of resilience resources that influence psychological well-being. Third, in addition to supporting a cumulative model of childhood and adult experiences creating an additive influence, we anticipate subsequent stress and resilience resources will moderate the relationship of early adversity to mental health by affecting respondents with higher levels of early life stress to a greater extent than other respondents.

## Method

### Sample

Data were drawn from the 2010 Behavioral Risk Factor Surveillance System (BRFSS) for Washington State. BRFSS is a cross-sectional, random-digit-dialed telephone survey conducted in collaboration with the Centers for Disease Control and Prevention (CDC) by health departments in all 50 states and U.S. protectorates (CDC, 2011). Participants are noninstitutionalized English- and Spanish-speaking adults 18 years or older who live in a household with a working landline telephone. Washington uses a disproportionate stratified sampling method with one adult per household randomly selected to participate in the survey, with a refusal rate (eligible contacts declined to participate) of 18.31%.

The study sample ($n$ =13,593) consisted of 60.7% females (average age 57.1 years; $SD$ = 16.0), of whom 33.8% were 65 years or older and 23.8% were 44 years or younger. The racial/ethnic composition of sample was 86.9% Caucasian, 5.0% Hispanic, 2.2% Asian, 1.3% African American, 1.2% Native American, 0.2% Hawaiian/Pacific Islander, and 3.2% mixed or other race/ethnicity. Approximately a third (36.4%) of the sample's household income was $35,000 or less, and 28.1% was $75,000 or more. 5.8% of the sample never completed high school, 23.4% had a high school education only, 32.2% attended some college, and 38.7% had a college degree. BRFSS-recommended sampling weights based on population estimates from the U.S. Census Bureau were used to correct for younger age and male gender underrepresentation.

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## Measures

**Demographics** consisted of age, gender (0 = *male*, 1 = *female*), race/ethnicity (seven categories reported above; Caucasian was the referent), and sexual orientation (1 = *heterosexual* [97.7%], 2 = *gay/ lesbian* [1.3%], 3 = *bisexua*l [0.9%]; *heterosexual* was the referent).

**Socioeconomic factors** included highest education level completed (6-level categorical scale: *no formal schooling, grades 1–8, not high school graduate, high school graduate or GED, some college, college graduate*) and annual household income (8-level categorical scale: *$0 to less than $10,000; $10,000 to less than $15,000; $15,000 to less than $20,000; $20,000 to less than $25,000; $25,000 to less than $35,000; $35,000 to less than $50,000; $50,000 to less than $75,000; and $75,000 or more*). Medical cost barrier was assessed on foregoing needed medical care because of cost: (reported this cost barrier; yes=1, no= 0).

**Adverse childhood experiences—**An ACE score was calculated by summing dichotomized *yes* responses across eight ACE categories in the childhood household before 18 years old: mental illness, substance abuse (alcoholic or illicit drugs), incarcerated family member, parental divorce, witnessing domestic violence, victim of physical abuse (parent/ adult hit, beat, kick, physically hurt), sexual abuse by adult/person 5 or more years older), and verbal abuse (CDC, 2010). The study sample scores ranged from 0 to 8 points ($M$ = 1.54, $SD$ =1.82) with the following percentile sample distributions: zero ACEs= 40.10%; 1=21.99%, 2=13.44%, 3=8.78%, 4=6.35%, 5=4.81% 6=2.67%, 7=1.45%, 8=.41%.

**Significant adulthood stressors** is a sum of dichotomized *yes* responses across five significant adult adverse events: incarceration, one or more periods of homelessness, relationship loss (divorced, separated, widowed), involuntary employment loss, and disability status (uses special equipment) Scores ranged from 0 to 5 ($M$ = 0.57, $SD$ = 0.80).

**Resilience resources** included *sense of community* (range 0–7; $M$ = 5.63, $SD$ =1.28), operationalized as the sum of two items: how often respondents received the emotional and social support they needed (rated on a 5-point Likert scale, never-always), and their level of satisfaction with their neighborhood (responses used a 4-point Likert scale). These items are consistent with the feeling-oriented sense of community construct (McMillan & Chavis, 1986), capturing perceptions and interactions related to membership and needs being met.

An ordinal *stress ameliorating health habits* index was based on sleep (reporting three or fewer versus 4–30 days of not enough sleep in the last month based on a median split of the item distribution) and physical activity (meeting CDC recommendations for moderate or vigorous physical activity). Responses were coded as follows: four or more days of poor sleep in the last month *and* low physical activity=0 (10.4%); poor sleep *or* low physical activity=1 (47.4%); and getting enough sleep with recommended physical activity=2 (42.3%).

**Mental health** was assessed in three forms. *Perceived well-being is* a mean of five items scored with a 5-point Likert scale (*strongly disagree* to *strongly agree; M = 4*.11, $SD$ =.78; $\alpha$ = 0.90). This inventory captures cognitive dimensions reflective of positive attitudes,

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appraisals, and beliefs (e.g., life satisfaction, life close to ideal, gotten what is important). *Psychological distress* is a mean of six symptoms of mental health problems (feeling nervous, hopeless, restless, depressed, worthless, everything an effort,) assessed on a 5-point Likert scale (*none* to *all of the time; M* =1.46, *SD* =.56; $\alpha$ = 0.82), capturing clinical dimensions of mental disorder (Kessler et al., 2002). *Missed days of work/activities* is an ordinal variable (0=0 days, 1=1 day, 2=2 days, 3= 3–7 days, 4=8–14 days, 5=15–21 days, and 6=22–30 days) measuring the number days in past 30 that a mental health condition or emotional problem interfered with usual activities such as work, studies, self-care, or caregiving. This variable was originally continuous and recoded as ordinal in this study to control for skew (range for continuous variable: 0–30; *M* = 88, *SD*=4.05).

### Analysis Plan

We first examined the bivariate correlations among study variables (see Table 1). We then undertook theory-guided hierarchical regressions, sequentially regressing the three measures of mental health onto four sets of explanatory variables, controlling for demographics: ACE score; SES characteristics; significant adult stressors; and resilience resources such as social support and health habits. This procedure tests multiple pathway and cumulative effects as well as the explanatory utility of each variable set and the individual predictor. Differential effects of adult stressors (exacerbating) and resilience resources (ameliorative) was tested through interaction effects of each on ACEs in relationship to each dependent variable. As the three outcome variables all demonstrated some degree of skew, all analyses were repeated with transformed variables. These results did not differ substantially, thus, to ease interpretation, nontransformed results were retained.

## Results

### Bivariate Correlations

All bivariate correlations were significant and in the expected directions (Table 1). ACE scores had robust associations with all psychological health indicators, especially psychological distress. No bivariate correlation between explanatory variables was sufficiently high to suggest problems with multicollinearity (Tabachnick, & Fidell, 2012).

### Hierarchical Regressions

The full regression model for each of the three mental health outcomes achieved significance (see Table 2). In addition, each of the predictor sets contributed significant additional explanation of each of the mental health indictors (change in $R^2$) consistent with a cumulative contribution model. The attenuation of the $\beta$ coefficient of ACEs with the addition of subsequent predictor set suggests shared variance, wherein variables in that step (e.g., adult stressors) overlap with ACEs effects on the health outcome (Lindenberger & Pötter, 1998; Mirowsky, 2013). Finally, as hypothesized, higher ACE scores were significantly associated with all mental health outcomes controlling for all other predictors, indicating a unique contribution consistent with a critical period life course effect.

Similarly, relationships of individual predictors to outcomes were consistent with expectations. Income and inability to pay were independently contributive for days of

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missed activities (although less robustly). A similar health-erosive pattern of association was evident through significant associations of adult stressors with all outcome variables. Sense of community and health habits were both positively associated with all mental health outcomes, net of other contributors, sense of community more robustly so. Among demographics, age was a fairly consistent contributor, with older age associated with more positive outcomes; being female was associated with greater perceived well-being; bisexuality trended toward less favorable outcomes; and race/ethnicity findings were weakly patterned, with limited significant group-specific effects.

### Testing Interaction Effects

Each of the hypothesized moderator variables (*adult stressors, sense of community, health habits*) and its multiplicative term with their ACE score were entered in separate regression models (see Table 3), controlling for demographic and SES variables parallel to Step 2 in Table 2. Separate testing of multiple moderators is recommended to avoid potentially confounding co-linearity effects (Fairchild & MacKinnon, 2009). In all cases, the regression models achieved significance. Two-thirds of the tested moderator terms achieved significance, controlling for covariates. In testing resilience resources (sense of community and health habits), the β coefficients for ACE scores significantly increased after inclusion of the moderation terms, particularly in testing sense of community. For example, the ACE path coefficient for psychological distress after controlling for demographics and SES was . 27 (Table 2), and .77 in the moderation test of sense of community (Table 3).

Figure 1 illustrates the exacerbating (adult stressors on ACEs) and ameliorating (sense of community on ACEs) moderation effects. The steepest slopes (strongest impact) are evident for individuals who reported greater adult stressors and lower sense of community in the context of higher ACEs. Individuals with high ACEs but lower adult stressors and high adult support reported mental health outcomes comparable to respondents with low ACE histories.

## Discussion

This study extends assessment of early life adversity effects on adult mental health in several ways. Guided by models of life course stress and trauma, our multivariate framework allowed for differentiating between the cumulative and distinct contributions of ACEs, adult SES and stressors, demographics, and resilience-fostering resources. Results provided support for multiple pathways underlying the impact of ACEs, including direct and moderated effects evident across three complementary indicators of psychological health: mental health symptoms, perceived well-being, and impairment of daily activities. This study is among the first to directly test moderating effects of adult stress in exacerbating the relationship of ACEs to mental health and, conversely, of resilience resources in ameliorating the risk of ACEs leading to later mental health problems. To understand life-long processes and consequences of stress and adversity it is essential to examine trauma and psychopathology from a cumulative adversity perspective. Moreover, this perspective encourages attention toward mutable preventive and resilience-building approaches to health promotion.

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### Unique, Proliferative, and Cumulative Pathways

Central to the current study's aims, our results support the premise that ACEs carry unique associations to compromised psychological health well into adulthood. The life course critical period concept recognizes potential for damage that begins in developmentally sensitive periods during childhood (Ben-Shlomo & Kuh, 2002). Specifically, early life trauma captured through total ACEs maintained unique significant association with the three indicators of impaired mental health.

The inclusion of three related yet distinct features of adult mental health permits examination of the stability of findings and their interpretations across models. Given that the final model controlled for associations with all other study variables, we speculate that these β coefficients are conveying, at least in part, that damage stemming from ACEs constitutes the childhood roots of adult health outcome inequalities (Shonkoff et al., 2009). Such damage is transmitted through interwoven social and biological mechanisms across the life course (Jaffee & Christian, 2014; Miller, Chen, & Cole, 2009). Although human systems strive to adapt to ACE traumas, these adaptations often tax a child's developing biological and psychosocial systems, resulting in dysregulations (e.g., stress sensitization) that dilute psychological and physical well-being (Danese & McEwen, 2012; Hostinar & Gunnar, 2013).

Findings also support our hypotheses that early stressors exert prolonged influence into later years through stress proliferation—a cascade of processes that accumulate stressful events in adulthood, increasing susceptibility to compromised psychosocial functioning (Aneshensel, 2009; Thoits, 2010; Umberson et al., 2008). Higher ACE scores were associated with poorer adult conditions, including low SES, high adult adversity, and diminished resilience resources, each of which influenced psychological well-being. These life conditions illustrate chains of risk in which one set of adversities tends to lead to another. For example, multiple ACEs generate consequences such as less educational achievement, which leads to financial insecurity that then increases risk of adult adversities such as homelessness, marital conflict, injuries, and unemployment. Consequently, this cascade of adversities over the life course weakens opportunities for stable social supports, ability to obtain professional help, and maintenance of healthy habits; all of which collectively and progressively chip away at psychological well-being. These pathways parallel observed patterns in allostatic load wherein the damaged caused by stacking or chronicity of stressors grows and spreads through biological systems (McEwen, 2008).

Although the buffering effects of protective factors relative to ACEs have only recently been assessed for adulthood consequences, conceptualizations of resilience processes and initial evidence point to protective resources as critical to advancing understanding (Rutter, 2006). Our findings illustrate the essential role of social resources and health-fostering behaviors in softening the blow of adversities and other stressful experiences on mental health and psychological well-being. Protective resources play an important complementary role as indicators of health and affiliated relationships (Taylor & Stanton, 2007). Even in cases of immediate challenges, strong social networks can bolster individual cognitive responding, support positive appraisals (optimism, sense of efficacy), and provide instrumental support (Thoits, 2010). Stressful conditions in early and later life have been shown to impede sleep

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quality and physical activity, which, in turn, are linked to greater risk for mental health problems (Callaghan, 2004; Greenfield et al., 2011). However, sleep and activity are essential resources for stress amelioration that help to regulate metabolic (Warburton, Nicol, & Bredin, 2006) and hormonal systems (Camacho et al., 1991; Edenfield & Blumenthal, 2011), offset disturbance aggravated by neurobiological alternations and hyperarousal (Koskenvuo, Hublin, Partinen, Paunio, & Koskenvuo, 2010), and foster psychological equilibrium and resilience to life stressors.

### Interactive Effects Shaping the Impact of ACEs

Collectively, the study findings support a cumulative model of life course adversity affecting functional and symptomatic dimensions of psychological well-being. Our findings also illustrate that the effects of later-life modifiers might be in play even if the processes are not immediately evident. The significant direct effect of adult adversities on mental health demonstrates population-relevant risk potential. However, the interactive relationships of adult adversities with ACEs illustrate that those with the highest levels of childhood trauma who also encounter greater adult adversities are most vulnerable to suffering mental health problems. That is, in addition to evidence of childhood stress being additive to lifetime stress burden and its prediction of psychopathology (Turner & Lloyd, 2004), our findings indicate that later stress can amplify the effect of early adversities, particularly for those with higher levels of exposures.

By contrast, resilience resources demonstrated ameliorative effects. On average, respondents who had a high sense of community were similar in psychological wellness across lower to higher levels of early adversity. However, the absence of this social resource among those with high ACEs was starkly associated with poorer mental health. Notably, sleep and activity showed the strongest moderating effects of ACEs in functioning. Those with high ACEs but healthy patterns of sleep and activity missed almost no work or other activities due to mental health problems, whereas those with high ACEs and poor habits missed many. Individuals with higher childhood adversity report poor sleep quality or disturbances such as pain at night, feeling hot/cold, or bad dreams, illustrating psychobiological mechanisms through which adversities are carried, potentially jeopardizing behavioral health as well as functional engagement in work and activities important to successful adulthood and psychological health (Charuvastra & Cloitre, 2009; Greenfield et al., 2011).

Similarly, although regular exercise demonstrates robust inverse associations with psychological distress, such activities are less available to and practiced by stressed populations (Hamer, Stamatakis, & Steptoe, 2009). These tandem trends buttress our hypothesis that personal and social resources offer cumulative protection (Masten, Cutuli, Herbers, & Reed, 2009) and, although beneficial to all, are likely to be particularly important to higher risk people by ameliorating the impact of stress. Moreover, once moderation was accounted for, the direct negative effect of the ACE coefficients increased substantially, suggesting the risk of ACEs for mental health among the general population might be underestimated in analyses that do not account for moderator effects.

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## Implications and Future Directions

Although the life course has many points for health prevention and intervention, this study joins others in urging that we think systematically about early life assessment during sensitive developmental windows, transitions, and milestone completions (e.g., 0 to 3 years stage, educational transitions, entry into parenthood). Findings point to the "double-jeopardy" of both high-stress and low-resource life circumstances and the resulting erosion of health and well-being that these combinations create. The compounding effect draws attention toward not only individual-level interventions but also to upstream factors that generate and sustain inequalities, exacerbating the accrual of adversities of those that have lesser resources and resilience capacity (Thoits, 2010).

In practical terms, these findings support the need for low-barrier integrated health care approaches that are attentive to stress embodiment--grounded in contextual approaches to wellness (Jaffee & Christian, 2014)--and that leverage resilience resources toward altering the course of ACE-affected trajectories. Conservatively, these findings point to pathways for lifelong psychological health among those who experience considerable adversity in early and later life, as well as the need for integrated team approaches to address needs across domains of individuals' and families' lives.

Under the Affordable Care Act, service models will increasingly integrate primary care with mental and behavioral health care, encouraging person-centered care with broader consideration of patients' social ecologies. Multilayered approaches to lessening life course exposure to adversity and fostering resilience will be key to minimizing proliferation of stress, to curbing psychiatric distress related to inevitable life stressors, and to fortifying supports and resources (Maschi, Baer, Morrisey, & Moreno, 2013). Notably, attention should be paid to the ways in which stress arises out of the contexts of people's lives, with levels and types of exposures differentially distributed across circumstances defined by social statuses, creating health disparities that set some individuals on a "resource edge" necessitating greater intervention and supports (Hobfoll, Johnson, Ennis, & Jackson, 2003).

Perceived well-being, which is based on multiple cognitive indicators of current life appraisals, provides a window into appraisal components of respondents' stress processing. Results indicating significant sustained significance of ACEs, with elevated coefficients after controlling for moderation effects, are consistent with stress-process mechanisms through which stressful life events become internalized as cognitive products (e.g., negative beliefs or attitudes) and cognitive habits (e.g., less optimistic interpretation of life events and expectancies; Matthews, Gallo, & Taylor, 2010). Although causal ordering of the psychological health outcomes cannot be established here, these findings support the value of interventions such as cognitive-based therapies to revise maladaptive appraisal patterns that affect individuals' emotional, behavioral, social, and biological well-being.

To more fully assess ACE effects (direct and indirect) and identify factors that demonstrate power to curb ACE effects, future research needs to include a spectrum of protective factors at the individual and community levels in data collection, theorized multivariate analysis, as well as biological data that can illuminate stress embodiment alongside psychosocial processes. Research focusing on early childhood adversity sets the stage for

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reconceptualizing adult health problems as developmental disorders. It also links models of social disadvantage with child maltreatment prevention and intervention efforts advocating for the alleviation of toxic childhood stress as a means of reducing persistent health disparities associated with trauma and chronic stress (Shonkoff & Garner, 2012). The life course cumulative adversity model used in this study is consistent with this capacity and calls for integrative approaches that locate health etiology and intervention responses within ecobiodevelopmental frameworks that grasp progressive, dynamic relationships between cumulative adversities and psychological well-being.

### Use of Administrative Data and Limitations

The current analysis draws on public health administrative data collected annually across all U.S. states. As part of a nationally integrated system, data from population-based surveys such as the BRFSS offer a practical strategy for developing a broadly based and multi-pathway understanding of life course adversities within a risk and protective factor framework. This framework yields valuable insights as to etiology and intervention targets related to psychological and physical health and their interplay. Data from sources such as child protective services tend to lack broader etiological antecedents, respondent characteristics, or later life outcomes important to life course examination, and limit attention to only those individuals with formally reported maltreatment or other serious adversities (Putnam-Hornstein, Needell, & Rhodes, 2013). Embedding child maltreatment and other toxic stressors into routine health surveys opens access to community-based samples that not only offer data complementary to other sources but are more representative of the general population.

Nevertheless, these benefits reside alongside limitations. These findings are based on self-reported, cross-sectional data, including a retrospective survey assessment of early adverse experiences that could introduce recall bias into the results. Because the current study is based on dichotomized yes/no responses of ever experiencing items before age 18 versus more precise prevalence or duration estimates, bias is likely reduced. Substantial testing of retrospective ACE accounts has demonstrated stable linear trends, minimal association with participants' psychological state at the time of data collection, and recall bias largely in the direction of underreporting ACE occurrences (Corso, Edwards, Fang, & Mercy, 2008; Yancura & Aldwin, 2009), including milder forms of childhood adversity associated with later mental health problems (Taylor et al., 2006) and comparison of retrospective and prospective results (Hardt, Vellaisamy, & Schoon, 2010). However, the presence of early adversity is nonetheless based on a limited number of items with multiple intervening decades, suggesting caution in interpretation.

As with most secondary analysis, measurement is constrained to the data routinely collected, which often includes single item measurement, can lack theoretically important variables, and limits the questions that can be posed. In examinations of early life adversity, exposure to poverty in childhood is a highly salient item, noting links between childhood SES and later life SES. While our study does make use of available measures of adult SES, information about early life poverty and economic status are not currently part of the BRFSS dataset. Potential for systematic patterns of nonparticipation, such as those who are not

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home-based, speak neither English nor Spanish, or not accessible by landline phone for interviewing, may contribute to biased estimate of population parameters. Despite these limitations, this study contributes to the empirical base regarding early life adversities and adult psychological health within a stress process framework, examining complex pathways into and through adulthood.

## Conclusion

Prevention remains a top priority in the realm of child and family welfare and is the foremost implication of early adversity research. Furthermore, early detection of and intervention in cases of child abuse, family dysfunction, and unmet psychosocial and functional needs can help to shift life course trajectories into more promising paths. At the same time, proliferating stress processes reinforce the ameliorative value of adversity assessment deeply into adulthood. Factors such as poverty, low educational attainment, inability to obtain professional care, adverse adult exposures, and limited supports strengthen the relationship of child trauma to health as well as bear their own imprint. This chaining pattern incrementally results in the stacking of risks and adversities that take environmental, psychosocial, and neurophysiological forms. Childhood trauma need not set the stage for poor mental health in adulthood, and indeed findings from this study demonstrate that it is, perhaps, never too late to scaffold wellness and high quality of life.

## Acknowledgments

This research was supported in part by a grant from the National Institute on Mental Health grant 5 T32 MH20010 “Mental Health Prevention Research Training Program”, the National Center For Advancing Translational Sciences of the National Institutes of Health under Award Number TL1TR000422, and a Eunice Kennedy Shriver National Institute of Child Health and Human Development research infrastructure grant, R24 HD042828, to the Center for Studies in Demography & Ecology at the University of Washington.

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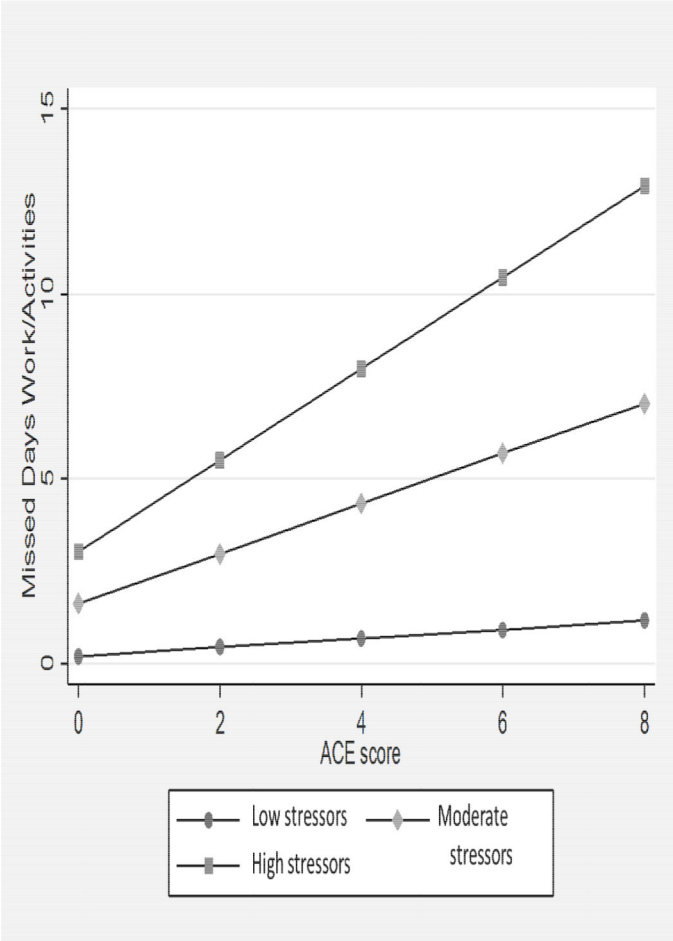


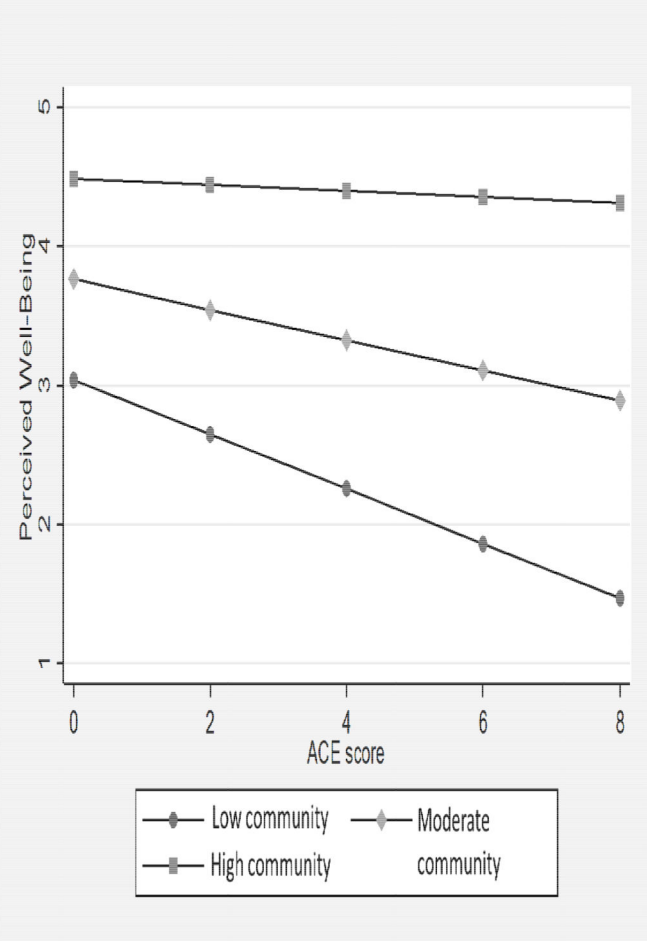


**Figure 1.**

Illustrating moderation relationships: adult stress (amplifying) and sense of community (buffering) effects of ACEs on missed days activities and perceived well-being.

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**Table 1**

Bivariate Correlations Among Study Variables

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
|---|---|---|---|---|---|---|---|---|---|
| 1. ACE score | | | | | | | | | |
| 2. Income | −0.110 | | | | | | | | |
| 3. Education | −0.077 | 0.399 | | | | | | | |
| 4. Medical cost barrier | 0.192 | −0.246 | −0.128 | | | | | | |
| 5. Adult stress | 0.159 | −0.489 | −0.174 | 0.174 | | | | | |
| 6. Sense of community | −0.172 | 0.229 | 0.122 | −0.175 | −0.189 | | | | |
| 7. Health habits | −0.144 | 0.131 | 0.107 | −0.137 | −0.161 | 0.171 | | | |
| 8. Perceived well-being | −0.269 | 0.302 | −0.088 | −0.236 | −0.341 | 0.460 | 0.258 | | |
| 9. Psychological distress | 0.345 | −0.260 | −0.106 | 0.257 | 0.313 | −0.365 | −0.301 | −0.578 | |
| 10. Missed work/activities | 0.228 | −0.022 | −0.068 | 0.184 | 0.309 | −0.243 | −0.185 | −0.417 | 0.579 |

*Note*. All correlations are significant at $p \leq .001$.

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**Table 2**

Hierarchical Regressions Testing Incremental, Unique, and Cumulative Pathways of Study Variables to Mental Health (standardized βs).

| | Perceived Well-Being | | | | Psychological Distress | | | | Missed Days Work/Activities | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **Step 1** | **Step 2** | **Step 3** | **Step 4** | **Step 1** | **Step 2** | **Step 3** | **Step 4** | **Step 1** | **Step 2** | **Step 3** | **Step 4** |
| *F* | **24.57** | **41.07** | **47.11** | **102.09** | **33.35** | **35.20** | **38.94** | **54.08** | **13.58** | **16.50** | **19.54** | **19.30** |
| $R^2$ | 0.10 | 0.19 | 0.23 | 0.37 | 0.15 | 0.22 | 0.25 | 0.34 | 0.08 | 0.12 | 0.16 | 0.18 |
| Age | **0.04** | 0.03 | **0.07** | **0.04** | **−0.11** | **−0.10** | **−0.14** | **−0.11** | −0.01 | −0.01 | **−0.05** | **−0.04** |
| Female | **0.06** | **0.08** | **0.07** | **0.06** | 0.00 | −0.01 | −0.01 | −0.00 | 0.01 | 0.00 | 0.00 | 0.01 |
| African Amer. | **−0.05** | −0.03 | −0.03 | −0.02 | 0.01 | −0.01 | −0.01 | −0.02 | 0.03 | 0.01 | 0.02 | 0.01 |
| Asian Amer. | −0.02 | **−0.03** | **−0.03** | −0.02 | **−0.03** | −0.03 | −0.02 | **−0.04** | −0.01 | −0.01 | −0.00 | −0.01 |
| HPI | 0.00 | 0.00 | 0.00 | −0.01 | −0.01 | −0.01 | −0.01 | −0.01 | 0.02 | 0.01 | 0.02 | 0.02 |
| Native Amer. | −0.03 | −0.01 | −0.01 | −0.01 | −0.02 | −0.03 | −0.03 | −0.03 | **−0.03** | **−0.04** | **−0.04** | **−0.04** |
| Hispanic | 0.01 | **0.07** | **0.05** | **0.06** | −0.01 | **−0.06** | **−0.04** | **−0.05** | 0.00 | **−0.04** | −0.02 | −0.03 |
| Other/Mixed | −0.03 | −0.02 | −0.00 | 0.00 | 0.02 | 0.01 | −0.00 | −0.00 | 0.03 | 0.02 | 0.01 | 0.01 |
| Gay/Lesbian | 0.00 | 0.00 | −0.00 | −0.01 | −0.02 | −0.02 | −0.02 | −0.02 | −0.00 | −0.00 | −0.00 | −0.00 |
| Bisexual | **−0.08** | −0.07 | −0.06 | **−0.06** | **0.10** | 0.08 | 0.08 | **0.08** | **0.12** | 0.11 | 0.11 | **0.11** |
| ACE Score | **−0.27** | **−0.21** | **−0.17** | **−0.11** | **0.33** | **0.27** | **0.24** | **0.19** | **0.24** | **0.19** | **0.16** | **0.14** |
| Income | | **0.24** | **0.16** | **0.11** | | **−0.17** | **−0.10** | **−0.06** | | **−0.15** | **−0.08** | **−0.06** |
| Education | | −0.01 | −0.01 | −0.01 | | 0.01 | 0.01 | 0.01 | | −0.00 | 0.01 | 0.01 |
| Medical cost Barrier | | **−0.16** | **−0.14** | **−0.09** | | **0.18** | **0.16** | **0.12** | | **0.11** | **0.08** | **0.06** |
| Adult stressors | | | **−0.23** | **−0.18** | | | **0.20** | **0.16** | | | **0.22** | **0.20** |
| Sense of community | | | | **0.37** | | | | **−0.26** | | | | **−0.15** |
| Health habits | | | | **0.10** | | | | **−0.14** | | | | **−0.07** |

*Note*. All model statistics and $R^2$ Δ values are significant at $p \leq .001$. Coefficients in bold are significant at $p \leq .05$.

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## Table 3

Regression-Based Tests of Moderation

| | Perceived Well-Being | Psychological Distress | Missed Days Work/Activities |
|---|---|---|---|
| ACE Score | −0.18*** | 0.21*** | 0.10*** |
| Adult Stressors | −0.22*** | 0.14*** | 0.09** |
| Adult Stressors * ACE Score | −0.01 | 0.08* | 0.19*** |
| ACE Score | −0.48*** | 0.77*** | 0.71*** |
| Sense of Community | 0.32*** | −0.16*** | −0.05* |
| Sense of Community* ACE Score | 0.33*** | −0.54*** | −0.54*** |
| ACE Score | −0.24*** | 0.33*** | 0.38*** |
| Health Habits | 0.14*** | −0.15*** | −0.01 |
| Health Habits * ACE Score | 0.05 | −0.08 | −0.23*** |

*Note*. ACE = adverse childhood experiences. Standardized coefficients controlling for demographic and SES variables. Models were run separately for each moderator and dependent variable.

* $p \leq .05$,

** $p \leq .01$,

*** $p \leq .001$.