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Research article

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.

Introduction

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; Schilling, Aseltine, & Gore, 2008).

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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 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 and outcome disparities (Anda, Butchart, Felitti, & Brown, 2010; Nurius, Green, Logan-Greene, Longhi, & Song, in press).

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 and Shippee, 2009; 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, 2010; 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).

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