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Adverse childhood experiences to adult adversity trends among parents: Socioeconomic, health, and developmental implications

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Abstract

Exposures to adverse childhood experiences compromise the early developmental foundation of people long before they become parents. These exposures partly take place within the family environment – a context tightly shared by parents and children. Despite considerable evidence regarding effects of adverse childhood experiences (ACEs), differential patterns of childhood and adulthood adversity accumulation among currently parenting adults is relatively less understood. The present study helps address this gap using the 2011 Behavioral Risk Factor Surveillance System Washington State data of respondents ages 18 and older who are currently parenting a minor child. Results demonstrate the proliferative nature of adversities, increasing risk of elevated life course stress, as well as parental socioeconomic, health and functioning outcomes that affect the family environment. Findings also suggest the resilience of some parents who, despite exposures to ACEs, were able to avoid heightened adversities in later life that could pose risk to their children's developmental environments.

Keywords

Adversity; Intergenerational; Parenting; Health; Socioeconomic

1. Introduction

Adverse childhood experiences (ACEs) carry jeopardy across multiple life domains, and now two decades of adversity research reveals that adversities function in a cumulative manner and are tied to worsening outcomes in physical well-being, mental health, and social domains in adulthood (Anda et al., 1999; Dong et al., 2004; Felitti et al., 1998; Hughes et al., 2017; Larkin, Shields, & Anda, 2012; Nurius, Green, Logan-Greene, & Borja, 2015; Solís et al., 2015). Part of these effects could be attributed to an ongoing context of adversity that results in the stacking of stressful experiences across the lifespan-often affecting the family environment – a context tightly shared by parents and children. These exposures can include a combination of toxic and chronic stressors such as exposures to maltreatment, parental incarceration, mental illness, and substance use that could compromise the developmental foundation of people before and during parenting (Chung et al., 2010). The result is a

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number of problems in adulthood like substance use, mental health problems, and incarceration that form the context of those adults' children's experiences. Further, these problems compromise parenting, an important socializing influence on child adjustment outcomes, which in turn, form the foundation for adult outcomes (Steele et al., 2016). Although there is now considerable evidence regarding ACEs and their effects, relatively less understood are the patterns and links of accumulation germane to parent-child dyads. The present study helps address this gap, with implications for efforts to curtail a growing public health concern of adversity accumulation and effects on child and family well-being.

A life course perspective recognizes that adversities in adulthood are often connected to a series of interrelated hardships and stressors and are often the results of conditions that are distally located in the life course (Pearlin, Schieman, Fazio, & Meersman, 2005). A considerable body of research has evidenced these links, primarily focusing on the persistence of poverty and economic hardship, trauma, risky behaviors, crime and delinquency, violence and psychopathology (Font & Maguire-Jack, 2016; Jones, Nurius, Song, & Fleming, 2018; Myers et al., 2015).

2. Conceptual foundations

Useful for understanding differences in intergenerational patterns of adversity is a merged perspective of cumulative adversity (CA) and life course (LC) principle of linked lives. These perspectives guide the current inquiry, and contextualize individuals in history, from early life to their eventual roles as parents, "setting in motion another life cycle" (Elder Jr, 1998). A cumulative adversity perspective is important because the effect of one risk factor alone may be small, but the effects of multiple risk factors together can be considerable (Evans, Li, & Whipple, 2013). Stressors tend to give rise to further stressor exposures as chains of risk and trends of stress proliferation often build over one's life course (Kuh, Ben-Shlomo, Lynch, Hallqvist, & Power, 2003; Pearlin et al., 2005). Stress response process models operationalize stress proliferation as earlier life stressors such as ACEs increase the likelihood of subsequent stressors, which result in cascades of stacking or layering of adversities over time. Stress embodiment specifies biological processes through which exposures to stressors such as chronic maltreatment compromise the development of brain regions and physiological functioning that, in turn, compromise physical and mental health development (Babenco, Kovalchuk, & Metz, 2015; Dannlowski et al., 2012; Kleinhaus et al., 2013; Lester et al., 2009; Tearne et al., 2015; Teicher, Anderson, & Polcari, 2012; Teicher, Samson, Anderson, & Ohashi, 2016).

A cumulative risk index used across multiple studies significantly predicted poorer outcomes across several developmental domains in children better than any single risk factor alone, including less success and competence of children in academic, conduct and peer domains (Ansell, Rando, Tuit, Guarnaccia, & Sinha, 2012; Appleyard, Egeland, van Dulmen, & Sroufe, 2005; 0; Evans et al., 2007; MacKenzie, Kotch, & Lee, 2011). The long arm of early adversity reaches well into adulthood when children become adults, with adverse experiences setting the stage of their adult life and, potentially, their parenting and family environment.

The life course principle of linked lives provides further scaffolding towards examining ACEs among parents in the context of later adult stress exposures and conditions, especially since the family environment, and other related factors such as parental warmth and maternal depression are important predictors of child outcomes in multiple domains (Lengua, Honorado, & Bush, 2007). Parents and their children are interdependent, and their lives are linked through shared events and experiences where the misfortune of one is potentially shared in their relationships and life conditions (Carr, 2018; Elder Jr, Johnson, & Crosnoe, 2003). Children from lower socio-economic family environments often have poorer educational outcomes and greater social disadvantage in adulthood (Duncan, Ziol-Guest, & Kalil, 2010; Hair, Hanson, Wolfe, & Poliak, 2015). Some factors that link economic status to health and other outcomes include the limited opportunity structures that often persist across the life course. Similarly, children exposed to poverty tend to exhibit poorer self-regulatory skills compared than children in non-poor families (Flouri, Midouhas, & Joshi, 2014; Lengua et al., 2007; Roy, McCoy, & Raver, 2014)—disparities that further undermine learning and success.

These ongoing exposure to psychosocial stress including economic hardship often results in psychobiological systems (e.g., nervous, endocrine and immune), fostering toxic stress response that leads to problematic patterns of cortisol release, abnormalities in the stress-sensitive physiological regions, and elevated levels of inflammation (Danese & McEwen, 2012; Kiecolt-Glaser et al., 2003; McEwen, 2007; Shonkoff et al., 2012). The wear and tear of these systems referred to as allostatic overload, conveys the burden on biological systems as the body strives to regain balance through recalibrations in response to stress challenges. Although adaptive in one sense, these processes lead to premature aging, putting individuals at higher risk for conditions such as cardiovascular disease, metabolic disorders, and cognitive decline in later life (Peavy et al., 2009; Rosmond, 2005; Steptoe & Kivimaki, 2013). The embodiment of the wear and tear could very well begin in childhood where exposures to social and economic adversities in tandem with other stressors such as parental substance use, parent mental illness, or victimization could become a potent combination that induces a toxic stress response early in the life course and compromise a healthy developmental foundation.

Various adversities and their long-term effects become the life context of many adults and their children. As some adults transition into parenting, their lifetime accumulation of adversities and their consequences are part of the context within which they fulfill parenting roles. As the early accumulation of ACEs is linked to health risk behaviors and poor social, economic and educational outcomes, many parents have to contend with consequences such as greater substance use and smoking, incarceration and/or partner incarceration, exposures to violence, limited economic resources, mental health challenges, and chronic health conditions (Anda et al., 2002; Bouvette-Turcot et al., 2017) in addition to the challenges and stress brought on by parenting. In turn, children from household environments characterized by the above challenges are likely to confront the demands of multiple stressors related to their parents' adversities as these shape an early developmental environment of elevated stress. Within proliferative stress trajectories, these conditions are also implicated in the early accumulation of adversities of their children, potentially establishing a pattern similar to that of the parents' childhoods.

Evidence shows that parenting-related factors have a role in early childhood development. Maternal parenting has been shown to account for the effects of cumulative risks such as poverty, parental depression, or being an ethnic minority on child outcomes such as effortful control (Lengua et al., 2007). Unfortunately, parenting capacities are susceptible to compromise by the confluence of a life course of adversities, with erosive consequences on adult outcomes across physical, mental, behavioral, social, and economic domains, particularly so in the absence of resilience-fostering protective factors. This is particularly true for parents experiencing depression – an outcome that has been linked to parenting behaviors such as neglect and maltreatment and lower parenting self-efficacy (Kohlhoff & Barnett, 2013; Turney, 2011).

3. The present study

This study builds upon the robust literature on ACEs and contributes a novel examination of patterns of adversity accumulation among adults currently parenting minor children. The aim is to test whether differential patterns of life course stress within an ACEs framework result in poorer later life outcomes, wherein consistent exposure to adversity in both childhood and adulthood is related to poorer statuses and outcomes in adulthood. Within a population representative sample, we distinguish respondents as to their level of ACEs and of adversities experienced in adulthood that directly parallel these forms of childhood adversity. An underlying premise is that greater cumulative parental adversity exposure will be associated with poorer economic and health statuses and that these less favorable outcomes pose jeopardy for their healthy environments needed by children.

4. Methods

4.1. Sample

Data were collected from the 2011 Behavioral Risk Factor Surveillance System (BRFSS) for Washington State ($n = 9375$)—a population-based state survey through the Centers for Disease Control applying disproportionate stratified random sampling to construct a representative sample. Participants are non-institutionalized adults age 18 years or older, living in a household with a working telephone. BRFSS recommended sampling weights were followed to adjust for a variety of sample demographic characteristics to be representative of Washington State.

To investigate patterns of childhood and adulthood adversities among parents, two steps were required to construct the study sample. Data from all respondents with minor children in the home were extracted from BRFSS 2011, and then weighted based on the number of minor children reported by each respondent. This yields a dataset ($n = 3552$) that simulates children as the unit of analysis and portrays the association of parental adversity histories on all youth currently in the home.

This sample of parents was 59.1% female and reported the following racial/ethnic composition: 74.8% Caucasian, 14.8% Latino/a, 3.2% Asian, 2.0% African American, 1.6% Native American, 0.5% Hawaiian/Pacific Islander, and 3.1% mixed or other race/ethnicity. Average sample age was 41.6 years ($SD = 10.2$), with approximately 37% age 45 or older

and 63% age 44 or younger. The sample majority was identified as predominantly heterosexual, with approximately 1% gay/lesbian and 1% bisexual. In addition, 26.7% of the sample reported a household income below \$35,000, and 42.3% was \$75,000 or more. Almost 10.9% of the sample never received a high school diploma, 18.9% had a high school education only, 27.6% attended some college, and 42.7% had a college degree and/or advanced training.

4.2. Measures

4.2.1. Forms of assessed adversity—Adverse Childhood Experiences (ACEs)

score is the sum of dichotomized “yes” responses across 6 of the CDC categories (CDC, 2010) of adverse experiences before the age of 18: serious mental illness, substance abuse (alcohol or illegal drugs) and incarceration of a household member during childhood; parental divorce or separation; and experiences of physical abuse and sexual abuse (range: 0–6, $M = 1.24$, $SD = 1.40$). For these ACE items, we had available item equivalents of respondents’ adulthood adversities for those experiences.

Adult Adverse Experiences (AAEs) score is the sum of dichotomized “yes” responses across 6 adverse experiences in respondents’ adulthood, paralleling six of the original ACE inventory: serious mental illness (feeling nervous/hopeless/restless/depressed/that everything was an effort/worthless), substance abuse (binge drinking/drug abuse/marijuana use), incarceration, divorce/separation/widowhood, physical victimization (slap, hit, kick, punch, beat by intimate partner), or sexual victimization (attempted or completed forced sex by adult/person) (range: 0–6, $M = 0.69$, $SD = 0.97$). These adult adversities then constitute ACEs for their minor children living in the home.

4.2.2. Household socioeconomic resources—Socioeconomic Status included (1) *annual household income* (8-level ordinal scale: \$0 to \$10,000; \$10,000 to \$15,000; \$15,000 to \$20,000; \$20,000 to \$25,000; \$25,000 to \$35,000; \$35,000 to \$50,000; \$50,000 to \$75,000; and \$75,000 or more; $M = 6.45$, $SD = 1.80$), (2) *highest education completed* (4-level ordinal scale: not high school graduate, high school graduate, some college, college graduate; $M = 3.02$, $SD = 1.02$), and (3) *out of work* (0 = employed, self-employed, homemaker, student, and retiree, 1 = reported as being out of work or unable to work; $M = 0.12$, $SD = 0.32$).

Food and Housing Insecurity included (1) *overpast 12 months, how often could not afford nourishing food* (0 = never to 4 = always; $M = 0.70$, $SD = 1.05$), (2) *food insufficiency index* (sum of affirmative replies that over the past 12 months the following occurred: food purchased did not last, could not afford balanced meals, had to cut meal size or skip meals, ate less than needed, and was hungry but could not afford more food; $M = 0.55$, $SD = 1.27$), (3) *residential stress* (lack of ability to pay rent/mortgage: 0 = never to 4 = always; $M = 1.08$, $SD = 1.19$), and (4) *homelessness* (sum of yes replies to residing in: a transitional housing, a shelter (domestic violence/emergency), or voucher lodging and/or a vehicle, abandoned building, or public outdoors (range: 0–2, $M = 0.02$, $SD = 0.15$).

Health Care Access included (1) *healthcare coverage* (0 = none, 1 = private or public insurance; $M = 0.81$, $SD = 0.39$), (2) *medical cost barrier* (0 = none, 1 = needed see a doctor

but not able because of cost; $M = 0.17$, $SD = 0.37$), (3) *routine checkup* ($M = 3.15$, $SD = 1.12$), and (4) *dental checkup* ($M = 3.38$, $SD = 1.03$) (the latter two assessed as: 0 = never, 1 = 5 or more years, 2 = within the past 5 years, 3 = within the past 2 years, and 4 = within the past year).

4.2.3. Health and functioning—Health risk behaviors included (1) *tobacco use* (0 = never, 0 = past or current; $M = 0.33$, $SD = 0.47$), (2) *Body Mass Index* (0 = neither overweight or obese, 1 = overweight, 2 = obese, using CDC calculated BMIs; $M = 0.89$, $SD = 0.79$), and (3) *use of seatbelts* (0 = never, 1 = seldom, 2 = sometimes, 3 = nearly always, and 4 = always; $M = 3.91$, $SD = 0.41$).

Disability included (1) *serious injury* in workplace ($M = 0.04$, $SD = 0.19$), (2) *limited activity* due to physical, mental, or emotional problems ($M = 0.22$, $SD = 0.41$), and (3) *health problems with special equipment* ($M = 0.03$, $SD = 0.17$) (three indicators above measured as “yes/no”), and (4) *number of falls* in past three months (range: 0–12, $M = 0.26$, $SD = 0.90$).

Physical Health was comprised of: (1) *chronic illness index* (sum of “yes” responses to diagnoses (diabetes, high blood pressure, high cholesterol, heart attack, angina, stroke, asthma, cancer, and arthritis) (range: 0–9, $M = 1.44$, $SD = 0.87$), (2) *self-assessed wellness* (Likert-type scale: poor = 0 to excellent = 4; $M = 2.71$, $SD = 0.99$), (3) number of *days per month physically unhealthy* ($M = 2.83$, $SD = 6.57$), and (4) number of *days per month that poor health disrupts usual activities* ($M = 3.39$, $SD = 7.09$) (the latter two ranged: 0–30).

Mental Health impact included (1) number of *days per month mentally unhealthy* ($M = 3.08$, $SD = 6.86$), (2) number of *days per month that emotional problem disrupts doing work* ($M = 0.66$, $SD = 3.43$) (the above two ranged: 0–30), and (3) *receiving medicine (Rx) or treatment (Tx) for mental disorder* (0 = no, 1 = yes; $M = 0.13$, $SD = 0.33$).

4.3. Parent adversity groups and analysis plan

Variables representing adverse experiences in parent respondents’ childhoods and adulthoods were: 1) ACEs and 2) AAEs. To derive groups of parental adversity, scores were dichotomized as low (0–1) and high (2+) to create a four-group typology of adversity trajectories from parents to children labeled as: 1) Consistently low (Low/Low) ($n = 2138$), 2) Lessening (High ACEs/Low AAEs) ($n = 805$), 3) Increasing (Low ACEs/High AAEs) ($N = 220$), and 4) Sustained High (High/High) ($n = 389$). The sample mean levels for ACEs = 1.24 and AAEs = 0.69, thus the cut-off points of 2 are the sample means. Groups constructed with higher cut-off points resulted in the same pattern of results with more marked mean group differences. We reasoned that a cut-off point of 2 provides a relatively conservative test of elevated adversity with direction of trends increasingly marked for those with levels above 2. Group differences on these variables assessed via ANOVA reported in Tables 1 and 2.

A series of Multivariate ANCOVAs were undertaken within the seven domains constituting household socioeconomic resources, and health and functioning, with gender and race as covariates. This analytic approach first tests the effect of the four parental adversity groups

on multiple measures that are distinct yet conceptually related under a common underlying domain (e.g., tobacco use, BMI, and use of seatbelts for health risk behaviors) simultaneously by using Wilks' lambda statistic (λ) in SPSS. Use of MANCOVAs limits the risk of an inflated Type I error and thereby provide more conservative omnibus test than ANOVAs alone (Hair, Black, Babin, Anderson, & Tatham, 2006). Individual ANOVAs for measures in each domain were also conducted with listwise deletion to capture mean differences across groups, followed by post-hoc tests for identifying significant pairs of groups. The number of included cases in each of the individual ANOVAs (Tables 2, 3, and 4) indicate a low percentage of missing values for most of the variables. We used a conservative approach to address missing data through listwise deletion given that the sample size provided enough statistical power to detect differences. Detailed MANCOVA and ANOVA results are presented in Tables 3 and 4.

5. Results

5.1. Sample characteristics

Table 1 indicates that demographics did vary across the groups. Younger (18–34) respondents were overrepresented in the High/Low group and those 65 or older more so in the Low/High and High/High groups. Females were overrepresented in the Low/High and High/High groups and sexual minority respondents in the High/High group. Race was less distinctly patterned with White and Native American respondents more so in the High/High group, and mixed-race respondents in both the Low/High and High/High groups relative to their sample representation.

Table 2 shows respondent ACE and AAE item distributions as well as averages across groups, each of which achieved significance. Those in the High/High group accumulated a greater number of adversity in childhood and adulthood and reported strikingly higher prevalence on the majority of items. The prevalence of AAEs in the High/High group is significantly higher compared to the group with lesser exposure to ACEs. Elevated childhood adversity was associated with greater exposure to substance abuse in the household with only a small but statistically significant difference between the High/Low and the High/High group. Higher ACE score was associated with greater prevalence of adversities in adulthood for all AAE items. The extent of most AAE items is also greater for the High/High group compared to the other group with similarly elevated AAE, except for substance use and divorce/separation/widowhood where there was a greater percentage among the Low/High group. Disproportionality was particularly high for adulthood mental illness with double the prevalence of High/High relative to Low/High respondents (35.73% vs. 17.27%) and nearly double parental incarceration rate relative to High/Low respondents (27.51% vs. 14.29%). Although the Low/Low and Low/High groups both had no or one ACE, the Low/High group reported double to triple levels of parental mental illness and violence witnessed in the home in childhood.

5.2. MANCOVAs and ANOVAs

Tables 3 and 4 present the results of MANCOVAs and the associated ANOVAs and post-hoc tests. All MANCOVA results achieved significance, indicating that the mean value of each

of the domains is meaningfully different for the four parental adversity groups. Thus, the central study premise was supported: that differences in the trajectories of adversity among parents represented by the four groups is associated with broad band differences in household socioeconomics and parental health—factors that constitute their children’s family environments.

Following significant MANCOVA results, individual ANOVAs and post-hoc tests provide additional specificity regarding the nature of intergroup differences (Tables 3 and 4). For instance, within the SES domain, the group test for income ($F = 52.14, p < .001$) achieved significance and each paired group contrast achieved significance. Between-group differences across food/housing insecurity measures were significant for most contrasts. Groups with greater proximal adversity as indicated by high AAEs regardless of ACE levels reported greater difficulties with health care access compared to the groups with current low AAEs. The presence of elevated adversity, whether childhood or proximal, was associated with greater health challenges across most of the health measures. Those in the High/High groups have distinctly compromised physical and mental health, and worse health behaviors compared to the other groups, except for differences in disabilities compared to the other higher AAEs group (Low/High).

6. Discussion

Although ACEs are widely recognized as predictive of adverse health outcomes across the life course, the patterns of parents’ adversity accumulation are considerably less investigated. This study extends our current understanding of early adversity by assessing parents’ ACEs in conjunction with their adult adversities to identify trajectories of adversity accumulation that represent parent-child dyads. Findings provide a snapshot of the shared family environment contexts of parents and their minor children as a function of parental adversity histories; specifically through variations in socioeconomic and parental health factors that constitute their children’s context of development. Parents with higher levels of adversity demonstrated markedly poorer health and economic circumstances, strikingly so for those with the greatest stress proliferation as manifest in both their childhood and adult adversities. This study also provides evidence suggesting the potential for resilience despite early adverse experiences among parents with lessening patterns of exposures (High/Low)—decreasing the risk of adversity in the family environments of their children.

In terms of demographics, the sustained high adversity group has an overrepresentation of women and sexual minorities (here assessed as gay/lesbian or bisexual). Parents under age 35 were overrepresented in the High/Low group. This trend may sustain, yet this group can experience further toxic adversities during their subsequent parenting years. Moreover, the sustained high adversity parents are close in average age to High/Low parents. These findings underscore the value of supporting parents early on to reduce their risk of stress proliferation and of adversity histories seriously undermining their capacity to provide secure, healthy, and nurturing household environments. Race and ethnicity were less distinctively patterned, yet some parents of color are overrepresented among higher adversity histories.

Adversity defines the early foundation of those that accumulated the most adversity in adulthood where the early exposures could weaken the base on which succeeding life experiences are built. Those in the Low/High group experienced significantly greater number of ACEs than Low/Low parents, particularly evident in childhood sexual abuse, which may be partly accountable for experiencing greater adult adversities and less favorable adult outcomes. Those in the High/High group reported significantly higher exposure to all adult adversities High/Low parents and poorer outcomes, reinforcing the value of interrupting continued patterns of adversity accumulation for those transitioning to adulthood and adulthood roles with higher ACEs.

Although the majority of general population parents reported lower levels of adversity exposure in both their childhood and adulthood, the evidence also points towards disadvantage and unfavorable circumstances that define the life experiences of many. Families in this sample who experienced sustained higher adversity carry the greatest burden of socio-economic disadvantage. Parents with early life adversity exposures and adult socioeconomic disadvantage report greater parenting stress and problems (Steele et al., 2016). Children in these families are raised in contexts of limited resources that could shape opportunities and risks that in turn, could dictate educational and economic prospects that further exacerbate. The stress proliferation framework explains this as the domino effect of one or more stressors that may foster secondary stressors within a life course and, thus, across generations.

Results provide further evidence of the proliferative nature of adversities as they are likely to diffuse across domains, evidenced by the worse physical and mental health and greater health risk behaviors of those experiencing sustained high adversity trajectory. The elevated AAEs of adults in this group suggest that elevated chronic stress already characterizes the early developmental context of their children from the onset. Early life experiences of adversity can be either growth-promoting or seriously damaging. However, it is the unrelenting nature of some adversities that is often responsible for the damaging effects on early brain architecture that could have a lasting impact on the social, emotional, and behavioral outcomes of young children. The stacking of adversities such as the chronic lack of economic resources, exposure to health risk behaviors, physical and mental health challenges of parents and other related difficulties such as parental divorce or incarceration indicate a potentially ongoing exposure of children to stressful situations in their families.

These adversities can disrupt brain architecture and thereby profoundly delay early learning and socio-emotional development (Fox, Levitt, & Nelson III, 2010; Hair et al., 2015). Exposures to early life adversities have been linked to smaller hippocampal grey matter and lower frontal and temporal lobe volume which are both potential pathways of poverty towards poorer academic outcomes and disrupted cognitive and behavioral development (Teicher et al., 2012). Language and cognitive deficits can already be observed among children living in poverty as early as seven months of age (Hamadani et al., 2014). These detrimental effects underscore a critical need to address AAEs and lessen their intergenerational impact on children. Early childhood is a sensitive period for development and presents a crucial window of opportunity for early childhood and parent interventions to

make a lasting impact towards the promotion and strengthening of a healthy foundation for future development.

Results further show that the exposure to chronic adversities could last a lifetime for many families. This is particularly true for parents in the High/High groups who, thus far, have sustained accumulation of adversities. The results not only point to intergenerational implications through parenting and the family environment, but also demonstrate a cascade that is observed across multiple life domains. Research on adversity has generated robust evidence regarding the harmful impact of ACEs on later health and dysregulated stress response (Anda et al., 2002; Felitti et al., 1998). When individuals are exposed to stressful experiences such as ACEs, physiological responses are observed through increased heart rate, blood pressure, and heightened secretion of stress hormones, which are generally adaptive. However, in the context of stacked or unrelenting adversity, these physiological states may fail to return to baseline, and the body's ability to cope with stress becomes dysregulated (McEwen, 2007).

A compromised stress regulation combined with chronic health issues, mental health-related challenges, and substance use may pose a more significant challenge, particularly for parents in the sustained high adversity group as they contend with the demands of their parenting role and ongoing burden of adversities. Even as children, these parents may have had compromised capacities to self-regulate and tolerate stress which carried implications towards their capacities to fulfill expected parenting roles as adults. Difficulties in emotional regulation are linked to coping difficulties and poor parenting that manifest in harsh parenting practices (Borelli, West, Decoste, & Suchman, 2012). Coping behaviors such as substance use may also be implicated in trends such as parental lack of monitoring, lesser parental involvement and harsher discipline. Further, maternal psychopathology and experiences of cumulative trauma have been linked to poorer parenting practices, increased propensity for child abuse and neglect, greater verbal and physical hostility. As such, children exposed to greater psychosocial stressors at home have worse socio-emotional and physical health outcomes, especially those with highly stressed parents.

Maternal stress has been linked to increased risk for heavier weight and obesity in childhood (Kozyrskyj et al., 2011; Tate, Wood, Liao, & Dunton, 2015). Parental stress is also implicated in less ideal health-related behaviors during childhood, such as poor oral health, physical hygiene, and safety practices (e.g., wearing a seatbelt) (Park & Walton-Moss, 2012). The increased tobacco use among parents in the High-High trajectories further underscores concern for perinatal exposure of their children to environmental toxins such as tobacco which has been linked to compromised fetal brain development, increased risk for multiple morbidities in childhood such as asthma, allergies, and metabolic disorders as well as long-term risk for cardiovascular diseases into adulthood (Cheraghi & Salvi, 2009; Ekblad, Korkeila, & Lehtonen, 2015; Ino, 2010)

Despite this concerning picture for parents and children in highly stressed home environments, the evidence presented in this study also demonstrates that for some parents with higher ACE exposures, continued adversity into adulthood does not hold true. There are windows of opportunity to interrupt what might become negative trajectories and prevent

intergenerational cascades of adversity. The present study demonstrates that parents in the High-Low lessening trajectory have better socio-economic and health indicators than those in the increasing and more pronounced sustained high exposures. Parents in the sustained low adversity group (Low/Low) and lessening group (High/Low) exhibit more positive health-related behaviors and contend with fewer health-related impediments to workforce participation. This enables greater access to health and other resources and, in turn, experience fewer chronic stressors, which, in total, have been found associated with greater engagement in positive parenting practices and less harsh parenting (Ettinger, Riley, Colantuoni, & Mendelson, 2018). Greater access to resources among those with increasing and sustained high adversity patterns could contribute to better outcomes for their children as some resources are known to have a protective effect against earlier disadvantage. This finding suggests the resilience of some parents who, despite their exposure to ACEs, can prevent the heightened exposure of their children to adversities.

6.1. Use of surveillance data: strengths and limitations

These results are based on analysis of a population-representative survey collected annually in the U.S. for public health purposes. These data reflect a broad array of demographic and health-related factors across multiple life domains. Its broad scope sets it apart from other surveys, especially those conducted among indicated or systems-based populations, as it allows a practical strategy for broader assessments of adversities and their relation to various outcomes in childhood and later in life. Embedding early and later life 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 such as the self-report nature of the data and retrospection needed for items such as the ACEs. Considerable testing of recalled ACE accounts has demonstrated stable linear trends, limited association with participants' state of mind during data collection, and recall bias skewed in the direction of underreporting ACE occurrences (Corso, Edwards, Fang, & Mercy, 2008; Yancura & Aldwin, 2009), including milder forms of childhood adversity and comparison of retrospective and prospective results (Hardt, Vellaisamy, & Schoon, 2010). However, the presence of ACEs is nonetheless based on a limited number of items recalled by many well into adulthood, suggesting caution in interpretation.

Also, results reported here are based on secondary analysis of an existing dataset. The researchers' assessments of ACEs and AAEs were constrained to the existing variables in the dataset. As such, other important adverse experiences were not assessed, such as exposures to community violence, bullying, racism, and immigration challenges, experiences that may influence adult outcomes and shape the context of early childhood development. As these are cross-sectional data, causality cannot be fully established. We are distinguishing the groups on the basis of their parallel childhood/adulthood adversity exposures, but cannot definitively establish temporality within the adulthood items.

Additionally, this sample is based on a predominantly white sample and may suggest the underestimation of adverse experiences as some people of color not reached by this survey often disproportionately more adversities than their white counterpart (Cronholm et al.,

2015; Wade Jr. et al., 2016). Although 39% of respondents with higher adult adversities report divorce or separation, these findings characterize one parent's conditions and, thus, do not capture information about other parents or caregivers who may be important to the family environment.

Notwithstanding the above limitations, this study presents a novel approach to understanding adversity using an intergenerational lens and contributes a more nuanced empirical understanding of the context of early childhood development.

7. Conclusion

These results have significant implications towards a linked lives perspective in research and intervention development wherein life course adversities of parents are considered as shaping their children's adverse experiences (Szilagyi & Halfon, 2015). While screening for early life adversity is important, research on interrupting worsening trajectories of accumulating adversities among parents is of high importance. So are the reduction of parenting stress and the promotion of parent resilience as these become the foundation of childhood development. Researchers, health and allied health professionals, educators and community providers are important resources for cultivating education and services to better support parents, especially those that are disproportionately impacted by adversities.

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References

- Anda RF, Croft JB, Felitti VJ, Nordenberg D, Giles WH, Williamson DF, & Giovino GA (1999). Adverse childhood experiences and smoking during adolescence and adulthood. *Jama*, 282(17), 1652–1658. [PubMed: 10553792]
- Anda RF, Whitfield CL, Felitti VJ, Chapman D, Edwards VJ, Dube SR, & Williamson DF (2002). Adverse childhood experiences, alcoholic parents, and later risk of alcoholism and depression. *Psychiatric Services*, 53(8), 1001–1009. [PubMed: 12161676]
- Ansell EB, Rando K, Tuit K, Guarnaccia J, & Sinha R (2012). Cumulative adversity and smaller gray matter volume in medial prefrontal, anterior cingulate, and insula regions. *Biological Psychiatry*, 72(1), 57–64. [PubMed: 22218286]
- Appleyard K, Egeland B, van Dulmen MHM, & Sroufe LA (2005). When more is not better: The role of cumulative risk in child behavior outcomes. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 46(3), 235–245.
- Babenko O, Kovalchuk I, & Metz GA (2015). Stress-induced perinatal and transgenerational epigenetic programming of brain development and mental health. *Neuroscience & Biobehavioral Reviews*, 48, 70–91. [PubMed: 25464029]
- Borelli JL, West JL, Decoste C, & Suchman NE (2012). Emotionally avoidant language in the parenting interviews of substance dependent mothers: Associations with reflective functioning, recent substance use, and parenting behavior. *Infant Mental Health Journal* 33(5), 506–519. [PubMed: 23049148]
- Bouvette-Turcot A-A, Unternaehrer E, Gaudreau H, Lydon JE, Steiner M, Meaney MJ, et al. (2017). The joint contribution of maternal history of early adversity and adulthood depression to

socioeconomic status and potential relevance for offspring development. *Journal of Affective Disorders*, 207, 26–31. [PubMed: 27685851]

- Carr D (2018). The linked lives principle in life course studies: Classic approaches and contemporary advances Social networks and the life course (pp. 41–63). Cham: Springer.
- Cheraghi M, & Salvi S (2009). Environmental tobacco smoke (ETS) and respiratory health in children. *European Journal of Pediatrics*, 168(8), 897–905. [PubMed: 19301035]
- Chung EK, Nurmohamed L, Mathew L, Elo IT, Coyne JC, & Culhane JF (2010). Risky health behaviors among mothers-to-be: The impact of adverse childhood experiences. *Academic Pediatrics*, 10(4), 245–251. [PubMed: 20599179]
- Corso PS, Edwards VJ, Fang X, & Mercy JA (2008). Health-related quality of life among adults who experienced maltreatment during childhood. *American Journal of Public Health*, 98(6), 1094–1100. [PubMed: 18445797]
- Cronholm PF, Forke CM, Wade R, Bair-Merritt MH, Davis M, Harkins-Schwarz M, ... Fein JA. (2015). Adverse childhood experiences: Expanding the concept of adversity. *American Journal of Preventive Medicine*, 49(3), 354–361. [PubMed: 26296440]
- Danese A, & McEwen BS (2012). Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiology & Behavior*, 106(1), 29–39. [PubMed: 21888923]
- Dannlowski U, Stuhrmann A, Beutelmann V, Zwanzger P, Lenzen T, Grotegerd D, ... Bauer J. (2012). Limbic scars: Long-term consequences of childhood maltreatment revealed by functional and structural magnetic resonance imaging. *Biological Psychiatry*, 71(4), 286–293. [PubMed: 22112927]
- Dong M, Giles WH, Felitti VJ, Dube SR, Williams JE, Chapman DP, & Anda RF (2004). Insights into causal pathways for ischemic heart disease adverse childhood experiences study. *Circulation*, 220(13), 1761–1766.
- Duncan GJ, Ziol-Guest KM, & Kalil A (2010). Early-childhood poverty and adult attainment, behavior, and health. *Child Development*, 82(1), 306–325.
- Ekblad M, Korkeila J, & Lehtonen L (2015). Smoking during pregnancy affects foetal brain development. *Acta Paediatrica*, 104(1), 12–18. [PubMed: 25169748]
- Elder GH Jr. (1998). The life course as developmental theory. *Child Development*, 69(1), 1–12. [PubMed: 9499552]
- Elder GH Jr., Johnson MK, & Crosnoe R (2003). The emergence and development of life course theory *Handbook of the life course* (pp. 3–19). Boston, MA: Springer.
- Ettinger AK, Riley AW, Colantuoni E, & Mendelson T (2018). Trajectories of early parenting practices among low-income ethnically diverse women. *Journal of Child and Family Studies*, 27(2), 615–628.
- Evans GW, Kim P, Ting AH, Teshler HB, & Shannis D (2007). Cumulative risk, maternal responsiveness, and allostatic load among young adolescents. *Developmental Psychology*, 43(2), 341–351. [PubMed: 17352543]
- Evans GW, Li D, & Whipple SS (2013). Cumulative risk and child development. *Psychological Bulletin*, 139(6), 1342. [PubMed: 23566018]
- Felitti VJ, Anda RF, Nordenberg D, Williamson DF, Spitz AM, Edwards V, ... Marks JS. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The adverse childhood experiences (ACE) study. *American Journal of Preventive Medicine*, 14(4), 245–258. [PubMed: 9635069]
- Flouri E, Midouhas E, & Joshi H (2014). Family poverty and trajectories of children's emotional and behavioural problems: The moderating roles of self-regulation and verbal cognitive ability. *Journal of Abnormal Child Psychology*, 42(6), 1043–1056. [PubMed: 24473936]
- Font SA, & Maguire-Jack K (2016). Pathways from childhood abuse and other adversities to adult health risks: The role of adult socioeconomic conditions. *Child Abuse & Neglect*, 51, 390–399. [PubMed: 26059537]
- Fox SE, Levitt P, & Nelson CA III (2010). How the timing and quality of early experiences influence the development of brain architecture. *Child Development*, 81(1), 28–40. [PubMed: 20331653]
- Hair JF, Black WC, Babin BJ, Anderson RE, & Tatham RL (2006). *Multivariate data analysis*. Upper Saddle River, NJ: Pearson Prentice Hall.

- Hair NL, Hanson JL, Wolfe BL, & Poliak SD (2015). Association of child poverty, brain development, and academic achievement. *JAMA Pediatrics*, 169(9), 822–829. [PubMed: 26192216]
- Hamadani JD, Tofail F, Huda SN, Alam DS, Ridout DA, Attanasio O, & Grantham-McGregor SM (2014). Cognitive deficit and poverty in the first 5 years of childhood in Bangladesh. *Pediatrics*, 134(4), e1001–e1008. [PubMed: 25266433]
- Hardt J, Vellaisamy P, & Schoon I (2010). Sequelae of prospective versus retrospective reports of adverse childhood experiences. *Psychological Reports*, 107(2), 425–440. [PubMed: 21117468]
- Hughes K, Beilis MA, Hardcastle KA, Sethi D, Butchart A, Mikton C, ... Dunne MP. (2017). The effect of multiple adverse childhood experiences on health: a systematic review and meta-analysis. *The Lancet Public Health*, 2(8), e356–e366. [PubMed: 29253477]
- Ino T (2010). Maternal smoking during pregnancy and offspring obesity: Meta-analysis. *Pediatrics International*, 52(1), 94–99. [PubMed: 19400912]
- Jones TM, Nurius P, Song C, & Fleming CM (2018). Modeling life course pathways from adverse childhood experiences to adult mental health. *Child Abuse & Neglect*, 80, 32–40. [PubMed: 29567455]
- Kiecolt-Glaser JK, Preacher KJ, MacCallum RC, Atkinson C, Malarkey WB, & Glaser R (2003). Chronic stress and age-related increases in the proinflammatory cytokine IL-6. *Proceedings of the National Academy of Sciences*, 100(15), 9090–9095.
- Kleinhaus K, Harlap S, Perrin M, Manor O, Margalit-Calderon R, Opler M, ... Malaspina D. (2013). Prenatal stress and affective disorders in a population birth cohort. *Bipolar Disorders*, 15(1), 92–99. [PubMed: 23339677]
- Kohlhoff J, & Barnett B (2013). Parenting self-efficacy: Links with maternal depression, infant behaviour and adult attachment. *Early Human Development*, 89(4), 249–256. [PubMed: 23398731]
- Kozyrskyj AL, Zeng Y, Colman I, HayGlass KT, Sellers EAC, Becker AB, & MacNeil BJ (2011). Maternal distress in early life predicts the waist-to-hip ratio in schoolchildren. *Journal of Developmental Origins of Health, and Disease*, 2(2), 72–80. [PubMed: 25140921]
- Kuh D, Ben-Shlomo Y, Lynch J, Hallqvist J, & Power C (2003). Life course epidemiology. *Journal of Epidemiology and Community Health*, 57(10), 778. [PubMed: 14573579]
- Larkin H, Shields JJ, & Anda RF (2012). The health and social consequences of adverse childhood experiences (ACE) across the lifespan: An introduction to prevention and intervention in the community. *Journal of Prevention & Intervention in the Community*, 40(4), 263–270. [PubMed: 22970779]
- Lengua LJ, Honorado E, & Bush NR (2007). Contextual risk and parenting as predictors of effortful control and social competence in preschool children. *Journal of Applied Developmental Psychology*, 28(1), 40–55. [PubMed: 21687825]
- Lester BM, Bagner DM, Liu J, LaGasse LL, Seifer R, Bauer CR, ... Das A. (2009). Infant neurobehavioral dysregulation related to behavior problems in children with prenatal substance exposure. *Pediatrics*, 124(5), 1355–1362. [PubMed: 19822596]
- MacKenzie MJ, Kotch JB, & Lee L-C (2011). Toward a cumulative ecological risk model for the etiology of child maltreatment. *Children and Youth Services Review*, 33(9), 1638–1647. [PubMed: 24817777]
- McEwen BS (2007). Physiology and neurobiology of stress and adaptation: Central role of the brain. *Physiological Reviews*, 87(3), 873–904. [PubMed: 17615391]
- Myers HF, Wyatt GE, Ullman JB, Loeb TB, Chin D, Prause N, ... Liu H. (2015). Cumulative burden of lifetime adversities: Trauma and mental health in low-SES African Americans and Latino/as. *Psychological Trauma: Theory, Research, Practice, and Policy*, 7(3), 243–251.
- Nurius PS, Green S, Logan-Greene P, & Borja S (2015). Life course pathways of adverse childhood experiences toward adult psychological well-being: A stress process analysis. *Child Abuse & Neglect*, 45, 143–153. [PubMed: 25846195]
- Park H, & Walton-Moss B (2012). Parenting style, parenting stress, and children's health-related behaviors. *Journal of Developmental & Behavioral Pediatrics*, 33(6), 495–503. [PubMed: 22772823]

- Pearlin LI, Schieman S, Fazio EM, & Meersman SC (2005). Stress, health, and the life course: Some conceptual perspectives. *Journal of Health and Social Behavior*, 46(2), 205–219. [PubMed: 16028458]
- Peavy GM, Salmon DP, Jacobson MW, Hervey A, Gamst AC, Wolfson T, ... Khandrika S. (2009). Effects of chronic stress on memory decline in cognitively normal and mildly impaired older adults. *American Journal of Psychiatry*, 166(12), 1384–1391. [PubMed: 19755573]
- Rosmond R (2005). Role of stress in the pathogenesis of the metabolic syndrome. *Psychoneuroendocrinology*, 30(1), 1–10. [PubMed: 15358437]
- Roy AL, McCoy DC, & Raver CC (2014). Instability versus quality: Residential mobility, neighborhood poverty, and children's self-regulation. *Developmental Psychology*, 50(7), 1891–1896. [PubMed: 24842459]
- Shonkoff JP, Garner AS, Siegel BS, Dobbins MI, Earls MF, McGuinn L, et al. (2012). The lifelong effects of early childhood adversity and toxic stress. *Pediatrics*, 129(1), e232–e246. [PubMed: 22201156]
- Solís CB., Kelly-Irving M., Fantin R, Darnaudéry M, Torrisani J, Lang T, & Delpierre C. (2015). Adverse childhood experiences and physiological wear-and-tear in midlife: Findings from the 1958 British birth cohort. *Proceedings of the National Academy of Sciences*, 112(7), E738–E746.
- Steele H, Bate J, Steele M, Dube SR, Danskin K, Knafo H, ... Murphy A. (2016). Adverse childhood experiences, poverty, and parenting stress. *Canadian Journal of Behavioural Science/Revue Canadienne des Sciences du Comportement*, 48(1), 32–38.
- Steptoe A, & Kivimäki M (2013). Stress and cardiovascular disease: An update on current knowledge. *Annual Review of Public Health*, 34(1), 337–354.
- Szilagyi M, & Halfon N (2015). Pediatric adverse childhood experiences: Implications for life course health trajectories. *Academic Pediatrics*, 15(5), 467–468. [PubMed: 26344714]
- Tate EB, Wood W, Liao Y, & Dunton GF (2015). Do stressed mothers have heavier children? A meta-analysis on the relationship between maternal stress and child body mass index. *Obesity Reviews*, 26(5), 351–361.
- Tearne JE, Allen KL, Herbison CE, Lawrence D, Whitehouse AJ, Sawyer MG, & Robinson M (2015). The association between prenatal environment and children's mental health trajectories from 2 to 14 years. *European Child & Adolescent Psychiatry*, 24(9), 1015–1024. [PubMed: 25431038]
- Teicher MH, Anderson CM, & Polcari A (2012). Childhood maltreatment is associated with reduced volume in the hippocampal subfields CA3, dentate gyrus, and subiculum. *Proceedings of the National Academy of Sciences*, 109(9), E563–E572.
- Teicher MH, Samson JA, Anderson CM, & Ohashi K (2016). The effects of childhood maltreatment on brain structure, function and connectivity. *Nature Reviews Neuroscience*, 27(10), 652–666.
- Turney K (2011). Labored love: Examining the link between maternal depression and parenting behaviors. *Social Science Research*, 40(1), 399–415.
- Wade R Jr., Cronholm PF, Fein JA, Forke CM, Davis MB, Harkins-Schwarz M, ... Bair-Merritt MH. (2016). Household and community-level adverse childhood experiences and adult health outcomes in a diverse urban population. *Child Abuse & Neglect*, 52, 135–145. [PubMed: 26726759]
- Yancura LA, & Aldwin CM (2009). Stability and change in retrospective reports of childhood experiences over a 5-year period: Findings from the Davis longitudinal study. *Psychology and Aging*, 24(3), 715–721. [PubMed: 19739927]

Table 1

Demographic characteristics across four parental adversity groups.

	Sample	Parental adversity groups ⁺				F or χ^2	
		Low/low (N = 2138)	High/low (N = 805)	Low/high (N = 220)	High/high (N = 389)		
		Avg. (sd.)	Avg. (sd.)	Avg. (sd.)	Avg. (sd.)		
Age						F(3, 3538) = 18.78 ***	
		41.60 (10.20)	42.36 (10.17)	39.61 (9.51) *	40.42 (10.53)		
	Col %						
Age			$\chi^2(9, N = 3542) = 96.28 ***$				
	18–34	23.35	18.83	33.13	29.56		
	35–44	39.84	41.27	36.36	40.10		
	45–64	34.58	37.70	29.51	26.99		
	65 or older	2.23	2.21	1.00	3.34		
Gender						$\chi^2(3, N = 3552) = 71.53 ***$	
	Male	40.93	45.84	38.01	25.19		
	Female	59.07	54.16	61.99	74.81		
Race						$\chi^2(18, N = 3524) = 132.68 ***$	
	White	74.83	73.16	76.78	78.55		
	Latino	14.84	16.54	14.48	10.08		
	Black	2.01	2.32	1.50	1.55		
	Asian	3.15	4.54	1.00	0.00		
	Native American	1.62	0.57	3.25	3.88		
	Hawaiian/PI	0.45	0.61	0.37	0.00		
	Mixed race	3.09	2.27	2.62	5.94		
Sexual orientation						$\chi^2(6, N = 3477) = 80.00 ***$	
	Heterosexual	98.36	98.89	98.62	94.03		
	Gay/Lesbian	0.66	0.82	0.25	1.04		
	Bisexual	0.98	0.29	1.13	4.94		

⁺ Group labels refer to ACEs/AAEs.

$p < .001$

 $p < .01$
**
 $p < .05$
*

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Table 2
Mean differences of childhood (ACE) and adulthood adversity experience (AAE) items and totals across parental adversity groups.

	Sample	Parental adversity groups				F or χ^2	
		Low/low	High/low	Low/high	High/high		
		Avg. (sd.)	Avg. (sd.)	Avg. (sd.)	Avg. (sd.)		
ACE	Sum Ranges 0–6 <i>a.b.c.d.e.f</i>	1.24 (1.40)	0.37 (0.48)	2.69 (0.90)	0.57 (0.50) *	F(3, 3548) = 3645.35 ***	
AAE	0–6 <i>a.b.c.d.e.f</i>						3.41 (1.13) **
	Items						
	0.69 (0.97)	0.27 (0.45)	0.43 (0.50)	2.31 (0.62)			2.60 (0.75)
	%	%	%	%			%
Parental ACEs in Childhood	Serious mental illness <i>a.c.d.e.f</i>					$\chi^2(3, N = 3552) = 1032.99$ ***	
	22.61	6.17	49.69	12.73			62.47
	Substance abuse <i>a.c.d.e.f</i>					$\chi^2(3, N = 3552) = 1393.55$ ***	
	32.49	11.37	73.91	14.09			73.26
	Incarceration <i>a.c.d.e.f</i>					$\chi^2(3, N = 3552) = 461.67$ ***	
	6.84	0.89	14.29	0.91			27.51
	Parental divorce/separation <i>a.c.d.e.f</i>					$\chi^2(3, N = 3552) = 1077.94$ ***	
	29.59	11.46	64.35	14.09			66.07
	Physical abuse <i>a.c.d.e.f</i>					$\chi^2(3, N = 3552) = 978.56$ ***	
	18.41	4.21	40.62	5.45			57.84
	Sexual abuse <i>a.c.d.e.f</i>					$\chi^2(3, N = 3552) = 799.90$ ***	
	14.30	3.18	26.21	9.55			53.47
Parental AAEs in Adulthood	Serious mental illness <i>b.c.d.e.f</i>					$\chi^2(3, N = 3552) = 516.02$ ***	
	7.97	2.95	5.34	17.27			35.73
	Substance abuse <i>b.c.d.e.f</i>					$\chi^2(3, N = 3552) = 475.57$ ***	
	18.33	11.97	11.93	55.91			45.24
	Incarceration <i>a.b.c.d.e</i>					$\chi^2(3, N = 3552) = 261.51$ ***	
	3.86	0.89	3.23	13.64			15.94

	Sample	Parental adversity groups				F or χ^2
		Low/low	High/low	Low/high	High/high	
Divorce/separation/widowhood ^{b,c,d,e,f}	10.70	4.40	4.35	45.91	38.56	$\chi^2(3, N = 3552) = 724.43^{***}$
Physical victimization ^{a,b,c,d,e,f}	13.68	3.79	7.08	50.00	61.18	$\chi^2(3, N = 3552) = 1195.75^{***}$
Sexual victimization ^{a,b,c,d,e,f}	14.50	3.32	11.06	48.64	63.75	$\chi^2(3, N = 3552) = 1191.26^{***}$

Post-hoc test designations: Tukey HSD test.

^aSignificant difference at 0.05 level between Low/Low and High/Low.

^bSignificant difference at 0.05 level between Low/Low and Low/High.

^cSignificant difference at 0.05 level between Low/Low and High/High.

^dSignificant difference at 0.05 level between High/Low and Low/High.

^eSignificant difference at 0.05 level between High/Low and High/High.

^fSignificant difference at 0.05 level between Low/High and High/High.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

Table 3

MANCOVA/ANOVA tests of group differences: Household socioeconomic resource domains with gender and ethnicity as covariates.

	Parental adversity groups					λ/F statistic
	Low/low	High/low	Low/high	High/high		
SES					MANOVA: $\lambda = 26.55$ ***	
Income ^{a,b,c,d,e,f}	6.67	6.52	5.91	5.48 *	F(3, 3217) = 52.14 ***	
Education ^{a,c,e,f}	3.09	2.99	2.98	2.72 **	F(3, 3512) = 15.08 ***	
Out of work ^{b,c,d,e,f}	0.08	0.10	0.17	0.31	F(3, 3514) = 60.23 ***	
Food/Housing insecurity					MANOVA: $\lambda = 20.43$ ***	
Could not afford nourishing food ^{a,b,c,e,f}	0.52	0.81	0.91	1.47	F(3, 1700) = 35.67 ***	
Food insufficiency index ^{a,b,c,e,f}	0.40	0.63	0.62	1.24	F(3, 3507) = 38.41 ***	
Residential stress: lack of ability to pay rent/mortgage ^{a,b,c,e,f}	0.85	1.17	1.42	2.21	F(3, 1659) = 44.39 ***	
Homelessness ^{a,b,c,e,f}	0.003	0.028	0.042	0.128	F(3, 3444) = 44.67 ***	
Health care access					MANOVA: $\lambda = 13.78$ ***	
Healthcare coverage	0.82	0.82	0.84	0.79	F(3, 3540) = 0.74	
Medical cost barrier ^{b,c,e,f}	0.13	0.16	0.22	0.31	F(3, 3534) = 25.13 ***	
Routine checkup ^{c,e}	3.18	3.19	3.07	2.95	F(3, 3484) = 3.04 ***	
Dental checkup ^{b,c,d,e,f}	3.47	3.40	3.25	2.95	F(3, 3488) = 29.25 ***	

Note: Manova F-statistic and the associated p -value are based on Wilks' lambda (λ).

Group differences based on Tukey HSD post-hoc test.

^a Significant difference at 0.05 level between Low/Low and High/Low.

^b Significant difference at 0.05 level between Low/Low and Low/High.

^c Significant difference at 0.05 level between Low/Low and High/High.

^d Significant difference at 0.05 level between High/Low and Low/High.

^e Significant difference at 0.05 level between High/Low and High/High.

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Significant difference at 0.05 level between Low/High and High/High.

$p < .05$.
*
 $p < .01$.
**
 $p < .001$.

Table 4

MANCOVA/ANOVA tests of group differences: Health and functioning domains with gender and ethnicity as covariates.

	Intergenerational groups of adversity transmission				
	Low/Low	High/Low	Low/High	High/High	λ /F statistic
Health risk behaviors					MANOVA: $\lambda = 29.30^{***}$
Tobacco use <i>a.b.c.d.e.f</i>	0.23	0.38	0.49 [*]	0.68 ^{**}	F(3, 3503) = 131.27 ^{***}
BMI <i>a.c.e.f</i>	0.84	0.92	0.83	1.10	F(3, 3257) = 9.21 ^{***}
Use of seatbelts in car <i>a.b.c.e.f</i>	3.95	3.92	3.87	3.76	F(3, 3518) = 26.51 ^{***}
Disability					MANOVA: $\lambda = 7.08^{***}$
Serious injury <i>a.b.c.d.e</i>	0.02	0.04	0.09	0.09	F(3, 2538) = 14.84 ^{***}
Limited activity <i>a.b.c.d.e.f</i>	0.16	0.23	0.35	0.43	F(3, 3500) = 57.29 ^{***}
Health problems with special equipment <i>b.c.d.e</i>	0.02	0.03	0.06	0.08	F(3, 3547) = 15.19 ^{***}
No. times of falls <i>b.c.e</i>	0.20	0.25	0.46	0.57	F(3, 1304) = 6.97 ^{***}
Physical health					MANOVA: $\lambda = 9.25^{***}$
Chronic illness index <i>a.c.e.f</i>	1.36	1.48	1.47	1.72	F(3, 3518) = 19.72 ^{***}
Self-assessed wellness <i>a.b.c.e.f</i>	2.81	2.69	2.65	2.26	F(3, 3510) = 35.43 ^{***}
No. days/month physically unhealthy <i>a.b.c.e.f</i>	2.11	2.97	3.19	6.18	F(3, 3477) = 42.80 ^{***}
No. days/month poor health disrupts usual activities <i>c.e.f</i>	2.65	2.99	3.29	6.30	F(3, 1947) = 22.28 ^{***}
Mental health					MANOVA: $\lambda = 55.15^{***}$
No. days/month mentally unhealthy <i>a.b.c.d.e.f</i>	1.87	3.36	4.45	8.55	F(3, 3488) = 115.90 ^{***}
No. days/month to emotional problem disrupts doing work <i>b.c.d.e.f</i>	0.26	0.30	0.98	3.31	F(3, 3494) = 99.66 ^{***}
Receiving Rx/Tx for mental disorder <i>a.b.c.e.f</i>	0.08	0.17	0.17	0.28	F(3, 3510) = 50.40 ^{***}

Note: MANCOVA F-statistic and the associated p-value are based on Wilks' lambda (λ).

Group differences based on Tukey HSD post-hoc test

^aSignificant difference at 0.05 level between Low/Low and High/Low.

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b Significant difference at 0.05 level between Low/Low and Low/High.
c Significant difference at 0.05 level between Low/Low and High/High.
d Significant difference at 0.05 level between High/Low and Low/High.
e Significant difference at 0.05 level between High/Low and High/High.
f Significant difference at 0.05 level between Low/High and High/High.

* $p < .05$.
** $p < .01$.
*** $p < .001$.