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# Emotion dysregulation mediates the relationship between lifetime cumulative adversity and depressive symptomatology

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ABSTRACT

Repeated exposure to stressful events across the lifespan, referred to as cumulative adversity, is a potent risk factor for depression. Research indicates that cumulative adversity detrimentally affects emotion regulation processes, which may represent a pathway linking cumulative adversity to vulnerability to depression. However, empirical evidence that emotion dysregulation mediates the relationship between cumulative adversity and depression is limited, particularly in adult populations. We examined the direct and indirect effects of cumulative adversity on depressive symptomatology in a large community sample of adults ($n = 745$) who were further characterized by risk status: never-depressed ($n = 638$) and "at-risk" remitted mood-disordered ($n = 107$). All participants completed the Cumulative Adversity Inventory (CAI), the Difficulties in Emotion Regulation Scale (DERS), and the Center for Epidemiologic Studies Depression Scale (CES-D). Bootstrapped confidence intervals were computed to estimate the indirect effect of emotion dysregulation on the relationship between cumulative adversity and depressive symptomatology and to test whether this indirect effect was moderated by risk status. Emotion dysregulation partially and significantly mediated the relationship between cumulative adversity and depressive symptomatology independent of risk status. Overall, cumulative adversity and emotion dysregulation accounted for 50% of the variance in depressive symptomatology. These findings support the hypothesis that disruption of adaptive emotion regulation processes associated with repeated exposure to stressful life events represents an intrapersonal mechanism linking the experience of adverse events to depression. Our results support the utility of interventions that simultaneously emphasize stress reduction and emotion regulation to treat and prevent depressive vulnerability and pathology.



Lifetime cumulative adversity, defined as the joint effect of repeated exposure to stressful events across the lifespan, has consistently been associated with incrementally worse health outcomes, including increased prevalence of psychiatric symptoms and increased vulnerability to psychiatric disorders (e.g. Benjet et al., 2010; Mabunda and Idemudia, 2012; Turner and Lloyd, 1995, 2004; Shrira, 2012). These findings are consistent with longitudinal investigations that have documented an association between exposure to stressful life events and depressive symptomatology (Caspi et al., 2003; Hammen, 2005; Kessler, 1997; Turner and Lloyd, 1995). Notably, however, lifetime cumulative adversity has been shown to provide a more robust account of variability in depressive symptomatology than more constrained measures of stress exposure, such as those that limit the exposure timeframe (typically to the past year or the past six months) or that are restricted to a single event or event category, such as parental neglect (Turner and Lloyd, 1995; Turner et al., 1995). The identification of mechanisms linking adversity to poor health outcomes is essential for the development of preventive interventions; however, these mechanisms remain inadequately understood.

Emotion regulation has been conceptualized as a multimodal process through which individuals consciously and nonconsciously modulate their emotions (Bargh and Williams, 2007; Rottenberg and Gross, 2003) to respond to environmental demands, including stressful events (Campbell-Sills and Barlow, 2007; Gratz and Roemer, 2004; Gross, 1998; Thompson, 1994). Emotion regulation has been identified as a central component in a wide range of psychiatric symptoms and disorders (Aldao et al., 2010; Bradley et al., 2011) and is increasingly regarded as a key mechanism of

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change in therapeutic contexts (Fairholme et al., 2009; Gratz and Tull, 2010; Mennin and Fresco, 2009). Depression, which has been conceptualized as a "distress disorder," (Watson, 2005) is widely regarded as a consequence of affective dysregulation (Campbell-Sills and Barlow, 2007; Gross and Munoz, 1995; Mennin et al., 2007). The multiple linkages between stress exposure, emotion dysregulation, and depression suggest that alterations to emotion regulation processes may represent a pathway linking cumulative adversity to vulnerability to depression (Repetti et al., 2002).

There is increasing evidence that exposure to acute and chronic stress reduces emotion regulation capacity (Dvir et al., 2014; Kim et al., 2013; McEwen, 2004; Sinha, 2001). Multiple studies have documented the profound neurobiological consequences of repeated exposure to stress on prefrontal and limbic-striatal functioning involved in the processing and regulation of emotions (Ansell et al., 2012; Davidson et al., 2002; Seo et al., 2014). There is evidence that efficient use of adaptive emotion regulation strategies may protect against the adverse consequences of stress (Hopp et al., 2011; Ochsner and Gross, 2005; Shallcross et al., 2010; van der Veek et al., 2009); however, a recent study demonstrating that acute stress provocation markedly impairs the efficacy of cognitive emotion regulation (Raio et al., 2013) suggests that emotion regulation capacity may be actively impaired during stressful experiences, which may, in turn, enhance psychiatric vulnerability.

To date, only a handful of studies have specifically examined emotion dysregulation as a potential mediator of the stress-psychopathology association, but these studies have consistently supported the hypothesis that emotion dysregulation mediates the stress-psychopathology relationship (e.g. Coates and Moore, 2014; Crow et al., 2014; Goldsmith et al., 2013; McLaughlin and Hatzenbuehler, 2009; Moriya and Takahashi, 2013; Stevens et al., 2013). For example, McLaughlin and Hatzenbuehler (2009) reported that emotion dysregulation mediated the relationship between recent stressful life events and changes in internalizing symptomatology over time in a short-term longitudinal study of adolescents. However, the studies that have been conducted to date have been consistently limited by the use of questionnaire-based assessments to measure participants' exposure to and appraisal of stressful events. Research indicates that questionnaire-based assessments are less accurate and more subject to bias than interview-based measures and that individuals' subjective perceptions of distress are likely to be confounded with the symptomatology of depression (Hammen, 2005). In addition, virtually all of the studies that have been conducted to date have been restricted to adolescent populations or have focused exclusively on early life adversity (Grant et al., 2006).

This overriding emphasis in the extant literature on early life adversity may be shortsighted in light of the durable and cumulative effects of traumatic stress (Turner and Lloyd, 1995), stress-related neural plasticity in the mature brain (McEwen, 2012), and evidence that stress accumulation may accelerate biological aging processes leading to increased stress-related vulnerability in geriatric populations (Shrira, 2012). Shrira (2012) reported that lifetime cumulative adversity was related to continuous vulnerability to depressive symptoms, as well as increased risk of mental health deterioration over time, in a longitudinal sample of elderly adults. To our knowledge, no prior studies have tested the hypothesis that emotion dysregulation mediates the relationship between lifetime cumulative adversity and depressive symptomatology in early and middle adulthood.

The present analysis deployed cross-sectional data from a large community sample of never-depressed and "at-risk" remitted mood disordered adults who completed the Cumulative Adversity Inventory (CAI; Turner et al., 1995), the Difficulties in Emotion Regulation Scale (DERS; Gratz and Roemer, 2004), and the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977). Although the sample did not include currently depressed individuals, thereby limiting the generalizability of the proposed mediation model to clinical populations, prior research indicates substantial continuity between subthreshold depressive symptomatology and syndromal depression (Enns et al., 2001; Flett et al., 1997; Solomon et al., 2001). Furthermore, subthreshold depression constitutes a major risk factor for the development of syndromal depression and is associated with significant functional impairment (Cuijpers and Smit, 2004; Cuijpers et al., 2007; Kessler et al., 1997; Lewinsohn et al., 2000; Rodriguez et al., 2012).

We hypothesized first (1) that cumulative adversity and emotion dysregulation would be independently associated with depressive symptomatology in both the never-depressed and at-risk cohorts; second (2) that the indirect effect of emotion dysregulation would significantly mediate the association between cumulative adversity and depressive symptomatology; and third (3) that the strength of the risk pathways would be significantly stronger in the at-risk cohort than in the never-depressed cohort. The latter two hypotheses were tested simultaneously in a moderated mediation model that incorporated both the never-depressed and at-risk cohorts. In addition, secondary mediation analyses and pairwise contrasts were conducted to determine the independent contributions of the six emotion dysregulation subscales.

## 1. Materials and methods

### 1.1. Participants

Six hundred and thirty-eight never-depressed and one hundred and seven "at-risk" remitted mood disordered individuals were recruited from a community sample in and around the New Haven, CT area. Demographics for these samples are reported in Table 1. No significant differences between the never-depressed and at-risk cohorts were detected in racial-ethnic composition, $\chi^2(2, n = 745) = 5.85$, $p = .054$ or marital status, $\chi^2(2, n = 745) = 2.45$ $p = .29$. However, statistically significant differences were detected in gender, $\chi^2(1, n = 745) = 8.14$, $p = .01$; age $t(743) = -2.43$, $p = .02$; and years of education $t(743) = -1.96$, $p = .050$. On average, the at-risk cohort scored significantly higher than the never-depressed

**Table 1**
Sample demographics.

| | Never-depressed ($n = 638$) | At-risk ($n = 107$) |
|---|---|---|
| Age | | |
| Mean (*SD*) | 29.5 (9.24) | 31.3 (9.49) |
| Median | 26 | 29 |
| Range | 18–50 | 18–50 |
| Years of education (*SD*) | 15.18 (2.29) | 15.64 (2.30) |
| Gender, *n* (%) | | |
| Male | 280 (43.9) | 34 (31.8) |
| Female | 358 (56.1) | 73 (68.2) |
| Race-ethnicity, *n* (%) | | |
| Non-Hispanic White | 455 (71.3) | 86 (80.3) |
| Non-Hispanic Black | 135 (21.2) | 16 (15.0) |
| Other | 48 (7.5) | 5 (4.7) |
| Marital status, *n* (%) | | |
| Never married | 452 (70.8) | 70 (65.4) |
| Married | 109 (17.1) | 22 (20.6) |
| Divorced, separated, or widowed | 77 (12.1) | 15 (14.0) |
| Adverse life events (*SD*) | 10.34 (6.23) | 12.73 (6.36) |
| Total emotion dysregulation (*SD*) | 68.69 (19.85) | 78.88 (21.42) |
| Depression symptoms (*SD*) | 10.81 (8.69) | 16.04 (10.2) |