Published in final edited form as:
J Psychiatr Res. 2015 February ; 61: 89–96. doi:10.1016/j.jpsychires.2014.11.012.

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.

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Conflicts of interest
We wish to confirm that there are no known conflicts of interest associated with this Publication.

Contributors
Protocol design and implementation was performed by Rajita Sinha. Data analysis was performed by Benjamin Abravanel. Benjamin Abravanel prepared the first draft of the manuscript. All authors have contributed to and approved the submission of the manuscript. We affirm that the manuscript has been read and approved by all named authors and that there are no other persons who satisfied the criteria for authorship but are not listed. We further affirm that the order of authors listed in the manuscript has been approved by all of us.

Keywords

Depression; emotion regulation; cumulative stress; adversity; mediation

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 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, 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 relative 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 Epidemologic 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.

Materials and Methods

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 cohort on measures of cumulative adversity, $t(743) = -4.689$, $p < .001$; emotion dysregulation, $t(743) = -5.89$, $p < .001$; and depressive symptomatology, $t(135.32) = -5.90$, $p < .001$. Notably, the mean CES-D score in the at-risk cohort was approximately 16, which is the clinical cut-off for depression risk (Radloff, 1977; Weissmann et al., 1977), whereas the mean CES-D score in the never-depressed cohort fell below this threshold.

All participants were between the ages of 18 and 50 years, able to read and write in English at least at a sixth grade level as assessed by self-report, and met stringent health requirements as assessed by a specialist research nurse. Exclusion criteria included DSM-IV criteria for current mood or anxiety disorders, dependence on any drug other than nicotine, head injury, and current use of prescription medications for any psychiatric or medical disorders. All participants gave both written and verbal informed consent, and the study was approved by the Human Investigation Committee of the Yale University School of Medicine, which is compliant with the Code of Ethics of the World Medical Association and consistent with the Declaration of Helsinki. In total, eleven hundred individuals were consented into the study, of whom eight hundred and eight-one met the initial eligibility screening and signed informed consent, and, of those, seven hundred and sixty-one completed the study.

Procedures

Potential subjects completed a telephone screening to determine eligibility based on inclusion and exclusion criteria. Following initial eligibility screening, participants were scheduled for assessment sessions as part of a larger protocol on stress and emotion regulation, during which time they met with research assistants to complete informed consent; medical, substance abuse, and psychiatric health assessments, including the Structured Clinical Interview for DSM-IV (First et al., 1996); the CAI (Turner et al., 1995); the DERS (Gratz and Roemer, 2004); the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977); and a baseline demographic questionnaire. Participants also

received a physical examination with a research nurse assessing cardiovascular, renal, hepatic, pancreatic, hematopoietic, and thyroid function to ensure that participants were not acutely ill or receiving active treatment for a medical illness. Breathalyzer and urine toxicology screens were conducted at each appointment to ensure drug-free status among participants. In total, participants completed three to four appointments over the course of four to six weeks, and all participants received monetary compensation for their research participation.

Measures

The Cumulative Adversity Index (CAI; Turner et al., 1995) is a 140-item interview that measures lifetime exposure to an array of traumatic and nontraumatic stressful life events, thereby facilitating analysis of the joint effect of repeated exposure to stressful life events, referred to as cumulative adversity. The CAI encompasses four stress domains comprising four subscales: major life events, life trauma, recent life events, and chronic stress. Trained interviewers asked participants about the occurrence, timing, and frequency of specific stressful events during their lifetime (Supplemental Table 1 presents the frequency with which specific life events were endorsed on the CAI in the never-depressed and at-risk cohorts). Prior research supports the use of interview-based assessments to efficiently improve the reliability and validity of retrospective reports of stressful life events (Dohrenwend, 2006; Hammen, 2005). In order to further eliminate potential sources of bias, the present study excluded the chronic stress subscale of the CAI, which elicits individuals' subjective perception of distress. A composite cumulative adversity score was computed as the sum of the three adverse life event subscales: major life events, life trauma, and recent life events. The CAI has previously demonstrated validity in both cross-sectional and prospective investigations of the onset of psychiatric and physical health conditions (e.g. Ansell et al., 2012; Gayman et al., 2008; Turner and Lloyd, 2004; Scott et al., 2008), including good internal consistency and test-retest reliability, and demonstrated good reliability in the current sample (α = .83).

The Difficulties in Emotion Regulation Scale (DERS; Gratz and Roemer, 2004) was used to assess emotion dysregulation. The DERS is a multidimensional instrument that incorporates six separate, but related subscales: (a) lack of emotional awareness (Awareness); (b) lack of emotional clarity (Clarity); (c) non-acceptance of emotional responses (Nonacceptance); (d) limited access to emotion regulation strategies (Strategies); (e) impulse control difficulties (Impulse); and (f) difficulties engaging in goal-oriented behavior (Goals). Respondents indicate the frequency with which each item applies to them using a scale ranging from 1 (almost never, 0%–10%) to 5 (almost always, 91%–100%). Items are summed to calculate a total score, and higher total scores indicate increased emotion dysregulation. Previous research indicates that the DERS has sound psychometric properties including good internal consistency, acceptable test-retest reliability, and discriminant validity in nonclinical samples (Gratz and Roemer, 2004), and the DERS demonstrated good reliability in this sample (α = .84). Additionally, all six of the DERS subscales have previously been found to be positively correlated with depression in adults (Ortega, 2009).

Depressive symptomatology was assessed using the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977). The CES-D is a brief 20-item self-report scale designed to measure depressive symptomatology in the general population. The CES-D incorporates affective (thirteen items) and somatic (seven items) symptoms of depression. The CES-D has been validated for use with a variety of populations, including both adolescents and adults and healthy and nonhealthy individuals (Devins et al., 1988; Garrison et al., 1991). Participants rate each item for the past week from 0 (rarely) to 3 (most or all of the time). The CES-D has sound psychometric properties, including excellent internal consistency, adequate test-retest reliability, and discriminant validity (Radloff, 1977), and demonstrated excellent reliability in this sample ($\alpha = .90$).

Data Analysis

Pearson correlations were computed to assess the zero-order relationships among cumulative adversity, emotion dysregulation, and depressive symptomatology in the never-depressed and at-risk cohorts. Next, we collapsed the never-depressed and at-risk cohorts in order to test a moderated mediation model in ordinary least squares regression, following procedures described by Preacher and Hayes (2008). This model tested emotion dysregulation total score as a mediator of the relationship between cumulative adversity and depressive symptomatology. The model simultaneously tested risk status (never-depressed vs. at-risk) as a moderator of each of the three direct and indirect pathways in order to determine whether the strength of the hypothesized pathways differed significantly between the two cohorts.

The sampling distribution of indirect effects was bootstrapped 10,000 times to provide nonparametric estimates of sampling distributions of the indirect effects. This approach has generally outperformed the more traditional model described by Baron and Kenny (1986) and has been shown to maximize statistical power (Preacher and Hayes, 2008). Effects were considered significant if zero was not included in the 95% confidence interval. Kappa-squared mediation effect size was computed using procedures outlined by Preacher and Kelley (2011). Kappa-squared is the ratio of the obtained indirect effect to the maximum possible size the indirect effect could have been, given the variances. The overall effect of the moderator, risk status, on the indirect effect of emotion dysregulation was considered significant if the 95% confidence interval for the index of moderated mediation (Hayes, 2013, in press) did not include zero.

Finally, secondary mediation analyses were conducted to assess the parallel contributions of the six DERS subscales, and pairwise contrasts were used to test whether these indirect paths were significantly different from one another (Preacher and Hayes, 2008). All of the regression and mediation analyses were controlled for age, gender, and marital status, which are the most consistent sociodemographic correlates of depression (Bromet et al., 2011). All analyses were conducted using SPSS version 20.0 and the PROCESS macro developed by Andrew Hayes (2013).

Results

Descriptive statistics

Table 2 displays the zero-order correlations between key study variables. In both the never-depressed and at-risk cohorts, cumulative adversity and emotion dysregulation were independently associated with depression symptoms, and cumulative adversity was significantly associated with emotion dysregulation.

Primary mediation analyses

Figure 1 displays the path coefficients from the primary bootstrapped regression and moderated mediation analyses, which treated emotion dysregulation as a mediator of the association between cumulative adversity and depression symptoms and risk status as a moderator of each of the three direct and indirect pathways. The regression coefficients are unstandardized and are adjusted for age, gender, and marital status.

The overall regression model was significant, $F(8, 736) = 75.05$, $p < .001$, $R^2 = .50$, although no statistically significant effects of age ($B = -.02$, $SE = .03$), gender ($B = -.52$, $SE = .52$), or marital status ($B = -.71$, $SE = .43$) were detected. Cumulative adversity was significantly predictive of the hypothesized mediating variable, emotion dysregulation ($B = .49$, $SE = .14$). The conditional total direct effect of cumulative adversity on depressive symptomatology was significant across both the never-depressed ($B = .45$, $SE = .05$) and at-risk ($B = 49$, $SE = .10$) cohorts. After accounting for the indirect effect of emotion dysregulation, the relationship between cumulative adversity and depressive symptomatology decreased slightly, but remained significant, indicating partial mediation. The 95% confidence interval for the conditional indirect effect of emotion dysregulation did not include zero in either the never-depressed [.05, .20] or at-risk [.05, .20] cohorts, indicating that the observed partial mediation effects were significant. The kappa-squared effect size indicated the presence of a medium mediation effect in the full sample, $\kappa^2 = .10$, 95% CI [.04, .17].

No significant interactions were detected between cumulative adversity and risk status on emotion dysregulation, $B = .24$, $SE = .33$, 95% CI [−.41, .88]; between emotion dysregulation and risk status on depressive symptomatology, $B = .06$, $SE = .03$, 95% CI [−.002, .13]; or between cumulative adversity and risk status on depressive symptomatology, $B = -.06$, $SE = .11$, 95% CI [−.28, .15]. Furthermore, the 95% confidence interval for the index of moderated mediation [−.12, .37] included zero. Collectively, these results indicate that neither the direct effect of cumulative adversity nor the indirect effect of emotion dysregulation was significantly conditional on the effect of risk status. A three-dimensional graphical representation of the linear relationships between cumulative adversity, emotion dysregulation, and depression symptoms in the full sample is presented in Figure 2.

Secondary mediation analyses

In order to determine which of the emotion dysregulation dimensions were responsible for the observed indirect effect, we conducted a multiple mediator analysis, with the six emotion dysregulation subscales treated as parallel mediators. Because the results of the primary

moderated mediation model did not indicate the presence of significant group differences, this multiple mediator analysis was tested in the full sample. Consistent with the primary mediation analyses, the results indicated that the emotion dysregulation subscales partially mediated the effect of cumulative adversity on depression symptoms. Table 4 summarizes the indirect effects of the emotion dysregulation subscales on the relationship between cumulative adversity and depression symptoms. Specifically, the indirect effects of Goals and Strategy on the relationship between cumulative adversity and depression were significant; however, the indirect effects of the other four subscales were not significant.

Finally, we conducted pairwise contrasts of the indirect effects using bootstrapped 95% confidence intervals (Preacher and Hayes, 2008). Based on this analysis, confidence intervals that do not include zero indicate the presence of a significantly stronger indirect effect. Table 5 summarizes these results. Pairwise contrasts revealed that the indirect effect of Strategy was significantly stronger than that of any of the other subscales, including the indirect effects of Goals. The indirect effect of Goals, although significant, was not significantly stronger than that of the other subscales, with the exception of Nonacceptance.

Discussion

Prior research indicates that repeated exposure to stressful life events across the lifespan, referred to as lifetime cumulative adversity, is a potent determinant of poor physical and mental well-being and a major risk factor for depression. The conceptualization of emotion dysregulation as a potential pathway linking cumulative adversity to psychiatric vulnerability is consistent with research indicating that emotion dysregulation is itself a major risk factor for the development of diverse psychopathologies, including major depressive disorder, and that exposure to acute and chronic stress adversely affects adaptive emotion regulation processes.

The extant literature contains a number of investigations that have reported a significant mediating effect of emotion dysregulation on the stress-psychopathology association; however, the research that has been conducted to date has tended to focus exclusively on early life adversity and/or adolescent psychopathology. Indeed, to our knowledge, the results of the current study are the first empirical evidence that emotion dysregulation mediates the association between lifetime cumulative adversity and depressive symptoms throughout early and middle adulthood. That the observed mediation effect was partial emphasizes the multiple mechanisms by which stress “gets under the skin,” as well as the multiple linkages between the pathophysiological sequelae of adversity and allostatic load and the syndrome of depression (McEwen, 2004, 2012; Tafet and Bernardini, 2003).

Crucially, the regression models explained fully half of the variance in depressive symptomatology, suggesting that cumulative adversity and emotion dysregulation are jointly associated with tremendous vulnerability. Moreover, contrary to our expectations, the proposed mediation model was equally successful in describing the relationships between lifetime cumulative adversity, emotion dysregulation, and depressive symptomatology in both the never-depressed and at-risk cohorts. This finding suggests that vulnerability to depression associated with cumulative adversity and emotion dysregulation is not contingent

on a unique pathophysiology of depression; instead, cumulative adversity and emotion dysregulation are incrementally associated with increased depressive symptomatology across the spectrum of vulnerability.

There are several plausible interpretations for the observed indirect effect of the Strategies subscale. According to Gratz and Roemer (2004), the Strategies subscale represents “the belief that there is little that can be done to regulate emotions effectively once an individual is upset.” The indirect effect of Strategies may reflect a structural knowledge deficit, suggesting that the individuals who are most likely to be exposed to high lifetime adversity are also the least likely to possess the requisite knowledge and skills to effectively regulate their emotions, thereby producing enhanced vulnerability to stress-related psychopathology. Alternatively, the indirect effect of Strategies may reflect a kind of learned helplessness (Seligman and Cook, 1978), whereby individuals who are repeatedly exposed to stressful life events develop a generalized sense of powerlessness in the face of uncontrollable adversity. The indirect effect of Strategy also emphasizes the importance of self-efficacy within the domain of emotion regulation, consistent with prior research on the etiology of depression (Bandura et al., 2003; Caprara et al., 2010; Maddux and Meier, 1995).

These results have important implications for the development of interventions that seek to treat stress-related pathology and vulnerability to depression. In particular, our results underscore the importance of emotion regulation as a specific target for intervention, consistent with recent perspectives in psychotherapy (Berking et al., 2008; Fairholme et al., 2009; Mennin and Fresco, 2009). Given the apparent primacy of the domain of emotion regulation captured by the Strategies subscale, skills-based emotion regulation training, such as that included in Dialectical Behavior Therapy, may be particularly effective. Our results highlight the utility of such interventions across the adult lifespan and suggest that individuals who have been exposed to high lifetime adversity might particularly benefit. At the same time, the fact that the observed mediation effect was partial suggests that interventions that target emotion regulation may be most successful when they are paired with stress reduction interventions.

Several existing evidence-based interventions have been shown to facilitate adaptive emotion regulation, including behavioral activation therapy (Syzdek et al., 2009) and mindfulness-based cognitive therapy (Corcoran et al., 2009), both of which were developed specifically for the treatment of depression symptoms. In addition, several novel interventions that target emotion regulation capacity are in the process of being developed, including emotion regulation therapy (Mennin and Fresco, 2009), affect regulation training (Berking et al., 2013), and contextual emotion regulation therapy (Kovacs and Lopez-Duran, 2012), the latter of which was designed for the treatment of pediatric depression. In a randomized clinical trial, Berking et al. (2013) found that an integrated therapeutic approach that combined cognitive behavioral therapy and an abbreviated variation of affect regulation training significantly outperformed cognitive behavioral therapy.

There are several limitations to the current findings. First, because the data were cross-sectional, it is impossible to determine the precise nature of the relationship between cumulative adversity, emotion dysregulation, and depression. However, our data were

consistent with longitudinal investigations of the stress-depression and stress-emotion dysregulation associations. Second, our results were based on self-reports, including retrospective reports of stressful life events, which may have been susceptible to reporting bias and over-reliance on memory; however, all of the measurement instruments, including the CAI, are well established and well validated. Third, the sample was predominantly composed of non-Hispanic White/Caucasian and Black/African-American participants, meaning that cross-cultural generalizability may be limited. A final limitation in considering the generalizability of these findings is that the sample was comprised of individuals who did not meet criteria for current mood or anxiety disorders. Although prior research suggests that there is substantial continuity between subthreshold and clinical depression, the proposed models may not adequately represent the effects in clinically depressed or anxious samples. These limitations notwithstanding, the current study was unique in that it deployed data from a large community sample of adults, including a medium-sized cohort of “at-risk” remitted mood disordered adults, and utilized an interview-based measure of lifetime adversity, paired with established measures of emotion dysregulation and depressive symptomatology, to demonstrate that emotion dysregulation partially mediates the relationship between the lifetime cumulative adversity and depressive symptomatology.

Conclusion

Overall, the current findings suggest that future longitudinal studies to assess the direct and indirect effects of cumulative adversity and emotion dysregulation on psychiatric vulnerability and pathology are warranted. In order to extend the model across the full spectrum of vulnerability, future studies ought to incorporate clinical populations. Crucially, our results emphasize the importance of stress reduction and emotion regulation skills as simultaneous targets for interventions that seek to treat or reduce the prevalence of depressive vulnerability and pathology.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Role of funding source

The current study was supported in part by National Institutes of Health grant DK099039 (Rajita Sinha PI). Study sponsors had no role in study design, data collection, analysis and interpretation; the writing of the report; or in the decision to submit the paper for publication.

We would like to thank all of the members of research staff who assisted with data collection and entry. Additionally, we would like to express our gratitude to Adam Hong for providing conceptual guidance pertaining to the initial design of data analyses and to Dr. Emily Ansell for offering her expertise on moderated mediation. This study was supported by National Institutes of Health grant DK099039.

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Highlights

- We examined direct and indirect effects of life event stress on depression
- We modeled emotion dysregulation as a mediator of the stress-depression association
- Both stress and emotion dyregulation were significantly associated with depressive symptoms
- Emotion dysregulation significantly mediated the stress-depression association
- The strength of the proposed mediation pathways was not conditional on risk status

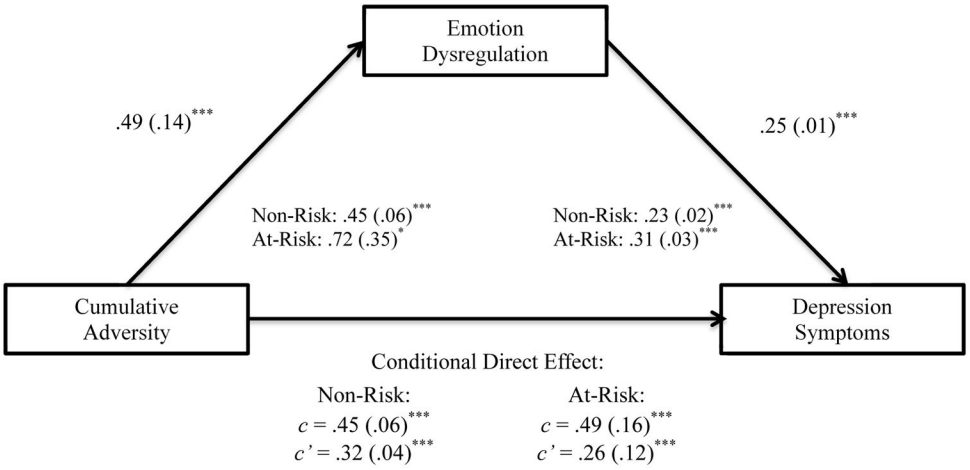

Figure 1.

Path Diagram Representing the Relationships between Cumulative Adversity, Emotion Dysregulation, and Depression Symptoms. * $p < .05$, ** $p < .01$, *** $p < .001$. $R^2 = .50$. Regression coefficients are unstandardized. c = Total direct effect. c' = Direct effect after accounting for indirect (mediated) effect of emotion dysregulation.

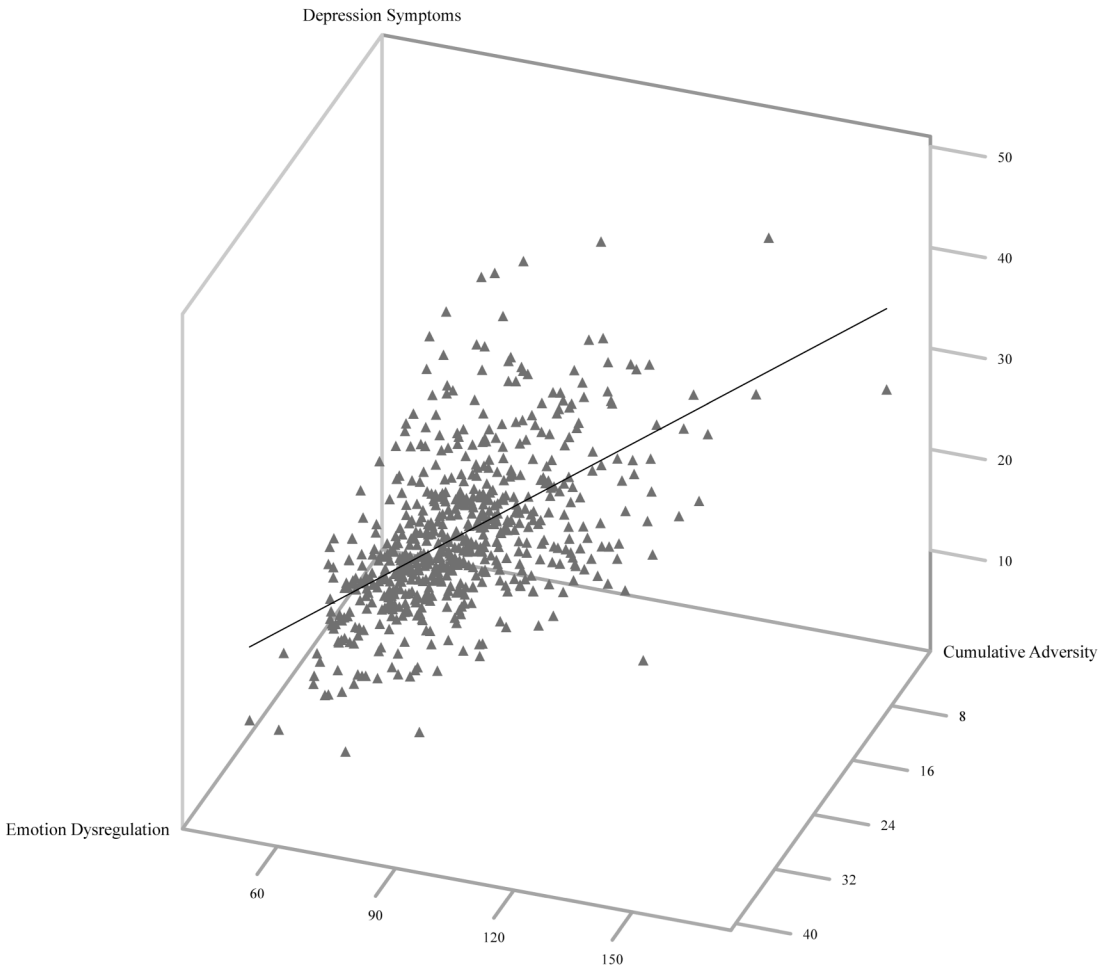

Figure 2.

Three-Dimensional Scatterplot of the Relationships between Cumulative Adversity, Emotion Dysregulation, and Depression Symptoms.

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)

Table 2

Zero-Order Correlations between Study Variables

	Never-Depressed (*n* = 638)			At-Risk (*n* = 107)			Total (*n* = *745*)		
	(1)	**(2)**	**(3)**	**(1)**	**(2)**	**(3)**	**(1)**	**(2)**	**(3)**
(1) Cumulative Adversity	—	.13**	.30***	—	.20**	.28**	—	.17***	.32***
(2) Emotion Dysregulation		—	.62***		—	.69***		—	.66***
(3) Depression Symptoms			—			—			—

Note.

* $p < .05$,

** $p < .01$,

*** $p < .001$

Table 3

Indirect Effects of Emotion Dysregulation Subscales.

	B	*SE*	95% CI
Awareness	.01	.01	−.00, .03
Clarity	.01	.01	.00, .04
Goals	.02*	.01	.01, .04
Impulse	.02	.02	−.01, .07
Nonacceptance	.00	.01	−.01, .02
Strategy	.13*	.03	.07, .21
Total	.20	.04	.11, .29

Note.

*Significant indirect effect as determined by bootstrapped 95% confidence intervals.

Table 4

Pairwise Contrasts of Indirect Effects

	B	*SE*	95% CI
Strategy			
versus Awareness	.12*	.03	.07, .20
versus Clarity	.12*	.03	.06, .20
versus Goals	.11*	.03	.06, .19
versus Impulse	.10*	.04	.03, .20
versus Nonacceptance	.13*	.03	.07, .21
Goals			
versus Awareness	.01	.01	−.01, .04
versus Clarity	.00	.01	−.02, .03
versus Impulse	−.01	.02	−.05, .04
versus Nonacceptance	.02*	.01	.01, .05

Note.

*Significantly greater indirect effect as determined by boostrapped 95% confidence intervals.