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Stressful Life Events and Depression Symptoms: The Effect of Childhood Emotional Abuse on Stress Reactivity

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Abstract

Objective—Stressful life events are associated with an increase in depressive symptoms and the onset of major depression. Importantly, research has shown that the role of stress changes over the course of depression. The present study extends the current literature by examining the effects of early life stress on emotional reactivity to current stressors.

Method—In a multiwave study (N = 281, mean age = 18.76; 68% female), we investigated the proximal changes that occur in depressive symptoms when individuals are faced with life stress and whether a history of childhood emotional abuse moderates this relationship.

Results—Results support the stress sensitivity hypothesis for early emotional abuse history. Individuals with greater childhood emotional abuse severity experienced greater increases in depressive symptoms when confronted with current dependent stressors, controlling for childhood physical and sexual abuse.

Conclusions—This study highlights the importance of emotional abuse as an indicator for reactivity to stressful life events.

Keywords

stress sensitization; emotional abuse; depression; stressful life events

Stressful life events have been consistently associated with an increase in depressive symptoms (see Mazure, 1998 for a review) and the onset of major depression in both adults (Hammen, 2005; Stroud, Davila, & Moyer, 2008) and adolescents (e.g., Abela & Skitch, 2006). Elucidating the nature of this association has been an important focus of research as some individuals react more negatively than others to such stressful life events. The present study extends the current literature by examining the effects of early life stress, specifically childhood emotional abuse, on individuals' reactions to current life stressors in developing depressive symptoms.

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[a]Indicates a variable is person mean centered;

[b]Indicates a variable is grand mean centered; EA = emotional abuse; SA = sexual abuse; PA = physical abuse; MDD Hist = history of a depressive episode prior to study inclusion; MDD Prosp = a prospective episode of depression.

Importantly, research has shown that the role of stress changes over the course of depression (for reviews, see Monroe & Harkness, 2005; Stroud et al., 2008). In formulating the stress sensitization or "kindling" hypothesis, Post (1992) described a process whereby the first episode of depression sensitizes an individual to life events, and thus subsequent episodes require less stress to elicit a depressive recurrence. This theory has been instrumental in explaining the high rates of recurrence and shortened intervals between recurrent episodes of depression (Solomon et al., 2000). Stress sensitization research suggests that an individual's reaction to stress changes or that an individual is "scarred" after an episode of depression (Lewinsohn, Allen, Seeley, & Gotlib, 1999). In recent years, research has extended this hypothesis to examine other variables that may confer a similar sensitization to life stress. Importantly, this heightened sensitivity may be analogous to diathesis-stress models, whereby an underlying vulnerability may put an individual at risk based on their maladaptive response to stress (Monroe & Simons, 1991).

Extensions of the kindling hypothesis have examined the effects of exposure to early childhood adverse experiences on later adult stress sensitivity and reactivity. Early negative life events and adverse environments have been shown to predict increases in depressive symptoms (Nolen-Hoeksema, Girgus, & Seligman, 1992) and the onset of major depression (e.g., Klein et al., 2009). Hammen, Henry, and Daley (2000), examining a number of childhood adversities, found a stress-sensitization effect in which women with higher levels of adversity showed more depressive reactions to low levels of stress compared with those with fewer adversities. Results provided an important step in extending Post's (1992) hypothesis to a role for early stressful life events in lowering an individual's threshold for the severity of stress needed to trigger depressive responses. Building on this study, research has examined the prospective relationship between early adversities, more proximal stressors, and the onset of major depression and shown that stress experienced in adolescence mediated the relationship between early adversity and the onset of major depression (Hazel, Hammen, Brennan, & Najman, 2008). These studies employed regression analyses using a composite of early stressful life events and provided an important direction for future studies that use more rigorous designs to test proximal changes in reactivity to stress.

Studies focusing specifically on childhood abuse and neglect have also reported an association with risk for depression (Alloy, Abramson, Smith, Gibb, & Neeren, 2006a). Individuals who experience childhood abuse are three to four times more likely to develop major depression in their lifetime (MacMillan et al., 2001). Bernet and Stein (1999) found that adults with major depression were significantly more likely to have a history of emotional abuse and neglect and physical abuse compared with healthy adults. In addition, those who experienced early childhood abuse had earlier onset, longer duration, more severe symptoms, greater impairment, and more episodes of depression (Klein et al., 2009; Bernet & Stein, 1999).

Although many studies have examined the relationship between childhood abuse and depression, the majority of research focuses on physical and sexual abuse, with fewer studies examining the effects of emotional abuse (Alloy et al., 2006a; Liu, Alloy, Abramson, Iacoviello, & Whitehouse, 2009). Emotional abuse typically includes experiences of being rejected, degraded, terrorized, isolated, or teased (Hart, Germain, & Brassard, 1987). An early review of the emotional and physical abuse literature concluded that emotional abuse was more strongly related to internalizing symptoms and suicide than was physical abuse (Kaplan, Pelcovitz, & Labruna, 1999). Although there are fewer studies of the role of childhood emotional abuse, the evidence for an association between emotional abuse and depressive symptoms and diagnoses is more consistent than for physical and sexual abuse (Alloy et al., 2006a).

In addition, few studies take into consideration the high levels of co-occurrence of different forms of abuse. Indeed, exposure to multiple forms of victimization is common (e.g., Finkelhor, Ormrod, & Turner, 2007), making it important to systematically examine the effects of different types of abuse as they may confer varied outcomes. The studies that have considered the overlap between emotional abuse and at least one other form of abuse suggest that emotional abuse was a stronger predictor of depressive symptoms or diagnoses than was childhood physical or sexual abuse (e.g., Gibb et al., 2001).

Stress sensitization may serve as an important theoretical basis for the various links between childhood maltreatment and later depression. To date, few studies have examined this possibility. Kendler, Kuhn, and Prescott (2004) found that women with sexual abuse histories were significantly more reactive to later stressful life events. Furthermore, women who were exposed to more severe forms of abuse showed a dose-response relationship in their increased sensitivity to developing depression (Kendler et al., 2004). Similarly, Harkness, Bruce, and Lumley (2006) found that individuals who had a history of childhood abuse and neglect were more sensitive to life events and reported lower levels of stressful life events prior to an episode of depression (Harkness et al., 2006). Although these studies either included only women (Kendler et al., 2004) or almost entirely Caucasian participants (98%; Harkness et al., 2006), both provide some evidence of differing reactivity to stress among individuals sensitized by abuse histories. Finally, in a clinical population using a similar longitudinal design to the current study, Dougherty, Klein, and Davila (2004) found that those who experienced poor parenting relationships, including emotional abuse, during childhood exhibited more depressive symptoms when confronted with subsequent chronic stress (Dougherty et al., 2004).

Monroe and Harkness (2005) highlighted the importance of clarity in the interpretations of evidence that support the original kindling hypothesis conception that life stress plays a differential role for the first onset of depression compared with recurrences. They clarify that the stress sensitization model suggests that major stress is essential for initial and early episodes, but successive recurrences of depressive episodes require progressively less stressful events for episode initiation. This implies that life events that initially were incapable of triggering a first depressive episode later acquire the capacity to trigger a recurrence. Consistent with the stress sensitization framework, exposure to childhood adversity may affect the relationship between stress and depression. As a person becomes more sensitized to stress through repeated exposure to childhood abuse, the probability of a depressive reaction to lower level stressful events rises. With repeated recurrence of stress, an accumulation of more minor events become ever more capable of precipitating onset of a depressive episode.

Further, in recent years, research highlights the importance of consideration that stressful life events are heterogeneous in nature. Harkness and colleagues (2006) were the first to distinguish differences between the effects of independent and dependent stressful life events in their relationship to stress sensitization for child abuse survivors in a cross-sectional study with adolescents. Dependent events are events that are in some way influenced by the individual (e.g., fight with a friend, failing a test), whereas independent, or fateful, events are viewed as those that may not be due to an individual's actions or characteristics (e.g., death of a relative, house flooding). Prior research highlights the relationship between dependent, but not independent, life events and depressive outcomes (Harkness, Monroe, Simons, & Thase, 1999; Kendler, Gardner, & Prescott 2006).

The present study aims to add to the stress sensitization literature by examining the independent contribution of childhood emotional abuse on current sensitivity to stressful life events. Consistent with prior studies (Hammen et al., 2000), we speculate that the link

between early emotional abuse and later depressive symptoms may be accounted for by a heightened stress reactivity that will lead to increased depressive reactions. Monroe and Harkness (2005) suggest that longitudinal studies are essential in testing within-person changes in the frequency and effect of stress over time.

Importantly, more advanced methodological tools have been developed to simultaneously examine within- and between-subject differences in longitudinal analysis (Raudenbush & Bryk, 2002). Hierarchical linear modeling (HLM; Raudenbush & Bryk, 2002) enables researchers to better model changes within individuals over time as well as differences between individuals. A multiwave prospective design over relatively brief intervals allows for a sensitive analysis of the relation between life stress and self-reported depressive symptoms and builds on prior cross-sectional or two time-point studies. Therefore, we investigated the proximal changes that occur in self-reported depressive symptoms when individuals are faced with life stress, and whether a history of childhood emotional abuse moderates the development of depressive symptoms in response to recent stressors.

Importantly, this study builds on prior research on emotional abuse by controlling for the overlap of other types of abuse, specifically physical and sexual abuse, to examine the unique contribution of emotional abuse. In addition, different types of stressful events may vary in their potential for eliciting depressive responses (i.e., independent and dependent events). We hypothesized that childhood emotional abuse would be associated with a stronger relation between recent stressful life events and prospective depressive symptoms and that this effect would be stronger for dependent stressful life events as compared with independent events.

Method

The current study takes advantage of a high-risk design, studying individuals hypothesized to be at increased risk for developing depression (Alloy et al., 2000, 2006b). One of the most consistent risk factors for developing depression is the presence of cognitive biases (Alloy et al., 2000, 2006b). Controlling for initial depressive symptom levels, cognitively high-risk participants were significantly more likely than cognitively low-risk participants to develop episodes of major depression (Alloy et al., 2006b). The sample in the present study comprised individuals who varied in vulnerability for depression based on cognitive-risk status. Importantly, when testing interactions (McClelland & Judd, 1993), the high-risk design provides greater variability in the constructs of interest (i.e., stressful life events and depression) and reduces problems related to a restricted range. Sampling across a wide range of observations and including more extreme scores can assist the detection of statistically reliable interactions by reducing standard errors without biasing parameter estimates (McClelland & Judd, 1993), thus increasing power to detect moderation.

Participants

Participants were recruited as a part of the Cognitive Vulnerability to Depression Project (CVD; Alloy et al., 2000, 2006b), a behavioral high-risk, two-site, multiwave, prospective longitudinal study designed to follow individuals at high and low cognitive risk for depression to predict the onset and course of depressive symptoms and disorders.

At phase 1 of recruitment, 5,378 university freshmen completed measures of cognitive vulnerability to depression. Participants who scored in the highest quartile were recruited for the high-risk group and those scoring in the lowest quartile were recruited for the low-risk group. Of those screened, 313 high-risk and 236 low-risk participants were invited to participate in phase 2 of the study.

At phase 2, an expanded version of the Schedule for Affective Disorders and Schizophrenia-Lifetime diagnostic interview (SADS-L; Endicott & Spitzer, 1978) was administered to assess and exclude individuals who currently met criteria for any Diagnostic and Statistical Manual of Mental Disorders Third Edition Revised or Research Diagnostic Criteria (RDC) diagnosis of mood disorder, or any current symptoms of psychosis. Participants were also excluded if they had a history of mood disorders with the exception of depression that had been in remission for at least 2 months (the average period of remission since past depressive episode was 2.31 years; standard deviation [*SD*] = 2.44 years). For further recruitment and demographic information, please see Alloy et al. (2000, 2006b).

The current sample comprised individuals who completed at least 2 years of prospective follow-up in the CVD Project. Of those participants, 42 were missing abuse history and were excluded from the study analyses, leaving 281 individuals in the final study sample (134 high risk and 147 low risk). Information on maltreatment was collected in the study's second year using the Life Experiences Questionnaire (LEQ; Gibb et al., 2001).

Participants ranged from 16 to 24 years of age (mean [*M*] = 18.76; SD = 1.64) at the time of study enrollment and were 68% female. Individuals in the sample were 80% Caucasian, 14% African American, 2% Hispanic, 4% Asian, and 2% other. The high- and low-risk groups did not differ significantly on sex, t (279) = .15, *ns*, age, t (279) = 1.64, *ns*, and ethnicity, t (273) = .41, *ns*, but did differ on Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery, 1979) scores, t (279) = −6.49, $p < .001$, overall stressful life events, t (279) = −2.25, $p < .05$, and independent events, t (279) = −2.16, $p < .05$, at baseline. See Table 1 for demographic and cognitive style characteristics of this sample. This subsample did not significantly differ from the total CVD Project sample on any demographic or cognitive style features. The final sample included 122 individuals with a depressive history. Throughout the course of the study, 37 participants met criteria for a major depressive episode.

Procedure

In phases 1 and 2 of the CVD Project, participants completed the SADS-L and cognitive style measures to determine eligibility and cognitive vulnerability. During regular prospective assessments (RPA), scheduled at 6-week intervals over the roughly 2 1/2 years of follow-up, participants completed the measures assessing depressive symptoms and stressful life events in person (Safford, Alloy, Abramson, & Crossfield, 2007). Based on evidence that stressful life events can have an almost immediate affect on depressive symptoms (e.g., O'Neill, Cohon, Tolpin, & Gunthert, 2004), measurement of stress and depressive symptoms were minimized to the shortest possible interval.

Depressive symptom data were collected at each RPA using the 2-week version of the BDI for each 2-week interval within that RPA. Life events were compiled into 2-week intervals according to their estimated dates of occurrence from the stress interview; thus, stress levels for each 2-week interval were determined and analyses could be completed within this shorter, 2-week interval. In analyses, changes in depressive symptoms were predicted using stressful life events from the 2-week period prior, while controlling for prior depressive symptoms. At the end of the second year of follow-up, participants completed a retrospective assessment of childhood experiences of abuse before 15 years of age.

Measures

Cognitive Style Questionnaire (CSQ; Alloy et al., 2000)—The CSQ is a revised version of the Attributional Style Questionnaire (ASQ; Peterson et al., 1982) and was created for the CVD project to assess the cognitive vulnerability featured in the hopelessness

theory. It measures inferential style based on the participant's ratings of 24 hypothetical events (12 positive and 12 negative). Participants rate the cause of the event on the dimensions of internality (caused by the participant), stability (cause is stable over time), and globality (cause is widespread across situations) and degree to which negative consequences and negative self-worth implications will follow. A negative composite score was created by summing participants' responses on the stability, globality, consequences, and self dimensions for the 12 negative events. The CSQ in the CVD project demonstrated good internal consistency (a = .88), retest-reliability (1-year, r = .78), and predictive validity for depressive episodes (Alloy et al., 2000, 2006b).

Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978)—The DAS assesses the maladaptive attitudes featured as vulnerabilities in Beck's theory. In the CVD project, an expanded DAS was used in which participants responded to 64 items assessing attitudes toward evaluation, performance criticism, causal attributions, expectations of control, and rigid ideas about the world. Participants rated statements on a 7-point Likert scale ranging from 1 (*totally agree*) to 7 (*totally disagree*). The measure was used with the CSQ (Alloy et al., 2000) to determine overall cognitive-risk status. The DAS in the CVD Project had good internal consistency (a = .90), retest-reliability (1-year, r = .80), and predictive validity for depressive episodes (Alloy et al., 2000, 2006b).

Beck Depression Inventory (BDI; Beck et al., 1979)—The BDI is a 21-item self-report measure that assesses depressive symptoms. Participants completed the BDI at every prospective assessment and reported for every 2-week period between assessments. Participants rated items on 0 to 3 scales and total scores ranged from 0 to 63, with higher scores indicating more severe depressive symptoms. The BDI has good reliability and validity (Beck, Steer, & Garbin, 1988).

Life Events Scale (LES) and Life Events Interview (LEI; Safford et al., 2007)—The LES and LEI were administered at every regular prospective assessment, approximately every 6 weeks, to assess the number, type, and dates of occurrence of negative life events experienced since the last interview. The 134-item LES was created for the CVD project to capture stressful life events and not include events that clearly reflect depressive symptoms.

Following the LES, participants were administered the LEI, a semistructured interview conducted by a trained interviewer blind to the participant's cognitive-risk status, symptoms, and diagnoses. LEIs were used to reduce any problems related to subjective reporter bias. For every event endorsed on the LES, the interviewer asked follow-up questions to determine if the event qualified for the manualized definitional criteria for each event, as well as the date of occurrence. Events that did not meet definitional criteria were excluded from the final count (4% of events). Common events included "Minor illness or injury," "Significant fight with a family member," and "Hurt by friend or peer."

Stress was operationalized as the objective count of negative events experienced over each of the 2-week intervals corresponding to the 2-week assessment of depressive symptoms. Three independent raters coded each event on its independence/dependence from 0 to 3 (0 = totally independent to 3 = definitely dependent) with an intraclass correlation of .87. These scores were then dichotomized into either independent or dependent. The LES and LEI have been shown to have excellent reliability and validity in terms of their ability to accurately capture the life events experienced (Safford et al., 2007).

Lifetime Experiences Questionnaire (LEQ; Gibb et al., 2001)—The LEQ is a 92-item self-report questionnaire designed to assess history of emotional, physical, and sexual maltreatment by peers and adults before 15 years of age. For each event, participants

reported on whether they had experienced the event, the age at which the event occurred, the frequency of the event, and the perpetrator. The LEQ was based on Cicchetti's (1989) child maltreatment interview to obtain comprehensive and specific information regarding the occurrence of specified events. Physical abuse events included being hit with a fist or object, being choked, and being the victim of deliberate physical pain. Emotional abuse events included being rejected, humiliated, degraded, terrorized, isolated, and denied emotional responsiveness. Sexual abuse events included sexual behaviors or exposure to pornography or unwanted intercourse (see Gibb et al., 2001 for more information).

Subscales consisted of physical (9 items; a = .65), emotional (20 items; a = .85), and sexual (20 items; a = .82) abuse questions and have demonstrated high correlations with structured maltreatment interviews and good predictive validity for episodes of depression (Gibb et al., 2001). The current study used a continuous measure of abuse by summing the total number of distinct events per abuse category with higher scores indicating more abuse events. The focus of the current study is emotional abuse; therefore, analyses controlled for levels of sexual and physical abuse.

Results

Overview of Statistical Approach

HLM (Raudenbush & Bryk, 2002) was used to investigate differences between individuals' depressive reactions to recent stressful life events as a function of their childhood emotional abuse history. Each model was run for different types of recent stress to examine whether reactions to particular events were affected by emotional abuse history. HLM is a rigorous statistical method for approaching these questions because it can represent changes within a person over multiple time points, as well as ascertaining how individuals may differ from one another over time (Curran & Willoughby, 2003). Ordinary least squares regression assumes that each data point is an independent sample, which may result in biased standard errors and inefficient coefficient estimates.

Preliminary Analyses

A total of 281 individuals and 14,994 observations were included in the final models. Two-week periods with missing data were excluded from analysis. Across follow-up, 68 of the potential 14,994 observation-level data points (2-week intervals) contained missing level-1 data. Such a high completion rate was possible because of the longitudinal nature of the study and the ability to assess data at subsequent prospective follow-up periods. Observation-level missing data were handled using listwise deletion. Thus, because of the lagged nature of the HLM analysis, when data were missing at interval T1, analyses to predict to the dependent variable in interval T were omitted. On average, participants completed 28 2-week intervals (*SD* = 15.5) during the 2-year period, which did not differ based on risk status, t (279) = .26, p = .79. However, all data up to drop out were included in study analyses. Noncompleters did not differ significantly from completers on risk status.

Independent samples t tests were used to assess whether demographic variables were associated with the main outcome variables; these tests showed that sex did not significantly affect baseline BDI scores, t (279) = −.50, *ns*, but risk status did, t (279) = −6.49, $p < .001$, and was thus controlled in analyses. A one-way analysis of variance showed that age at study enrollment was not significantly related to baseline BDI scores, F(12, 280) = .93, *ns*. Bivariate correlations of the main study variables showed that there were significant correlations between stressful life events, risk status, and emotional abuse.

In addition, all stress variables were correlated with each other. Depressive symptoms were correlated significantly with all stress variables and emotional abuse only. As a further test of the effects of emotional abuse, a hierarchical linear regression was run to examine the effects of emotional abuse on depressive symptoms at Time 1 (T1) controlling for risk status. Individuals with higher levels of emotional abuse had significantly higher levels of depressive symptoms at T1, $\beta = .22$, t (280) = 2.70, $p = .007$, controlling for cognitive risk. All of the abuse categories were correlated significantly, highlighting the need to control for overlap of variance in further analyses.

Abuse as a Vulnerability Analysis

For analyses, the within-subject (time-varying) level assessed whether levels of stressful events in a 2-week period predicted changes in depressive symptoms in the next 2-week interval. The cross-level assessed whether childhood emotional abuse history moderated the relationship between stress and depressive symptoms. The outcome variable for all analyses was depression symptom (BDI) scores in the current 2-week interval (T), controlling for previous interval BDI (lagged BDI) scores (T1) and time in the study (Time; number of follow-up visits). This enables the model to examine change in depressive symptoms at each interval and control for autocorrelation of depressive symptoms across time. The within-subject level also included the variable lagged stress, which represents the number of negative life events in the previous 2-week interval (T1). Lagged stress was person-mean centered, so the coefficient for this variable represents the effects of within-person changes in lagged stress. The between-subject level consists of static characteristics of the participants.

T1 cognitive vulnerability (risk status; dichotomous) was used as a control to allow for a rigorous examination of the effects of childhood emotional abuse above the potential role of cognitive style. The participant's sex was also entered at the between-subject level of analysis to control for sex differences. The continuous measure of childhood emotional abuse history (emotional abuse) was entered to examine whether a history of higher levels of abuse increased depressive reactivity to stress as measured by depressive symptom levels. In addition, we included the continuous measures of physical and sexual abuse as covariates to control for the co-occurrence of other types of abuse and examined the unique contribution of an emotional abuse history. Furthermore, because of the examination of stress sensitization, participant's history of past episodes of depression (MDD Hist) and the prospective episodes of major depression (MDD Prosp) were controlled for in analysis to reduce the contamination of episode history.

Finally, the cross-level interaction of emotional abuse (grand-mean centered) x lagged stress enabled us to evaluate the child abuse sensitivity hypothesis of the study. Of note, the following model represents childhood emotional abuse and stress. After examining overall stress, analyses were repeated for differences between independent and dependent stressors separately (see Table 2).

Within-subject

$$\mathrm{BDI}_{ij} = \pi_{0j} + \pi_{1j}(\text{Lagged BDI}) + \pi_{2j}(\text{Lagged Stress}^{a}) + \pi_{3j}(\text{Time}) + e_{ij}$$

Between-subject:

$$\pi_{0j}=\beta_{00}+\beta_{01}(\mathrm{EA})+\beta_{02}(\mathrm{Risk})+\beta_{03}(\mathrm{SA})+\beta_{04}(\mathrm{PA})+\beta_{05}(\mathrm{Sex})+\beta_{06}(\mathrm{MDD\ Hist})+\beta_{07}(\mathrm{MDD\ Prosp})+R_{0j}$$
$$\pi_{1j}=\beta_{10}+R_{1j}$$
$$\pi_{2j}=\beta_{20}+\beta_{21}(\mathrm{Emotional\ Abuse}^{\mathrm{b}})+R_{2j}$$
$$\pi_{3j}=\beta_{30}+R_{3j}$$

Three analyses were conducted and are summarized in Table 2. Variables were entered in a stepwise fashion for each stress category, with the first column indicating the model without the interaction term and the second column indicating the model with the childhood abuse x stress interaction. Overall, the results suggested that emotional abuse moderated the effect of dependent stressful life events, but did not moderate overall or independent events. The main effects for the overall, independent, and dependent stress models were similar. That is, in the overall stress model, individuals with high risk status, β = .85, *t* (280) = 6.29, *p* < .001, had higher levels of current depressive symptoms. In addition, women were more likely to be depressed than men, β = .36, *t* (280) = 3.06, *p* < .01. As expected, both higher levels of T1 stress, β = .02, *t* (280) = 2.57, *p* = .01, and higher T1 BDI scores, β = .36, *t* (280) = 11.56, *p* < .001, predicted increases in current depressive symptoms.

Of note, in the interaction models, childhood emotional abuse status moderated the relationship between the T1 dependent stressors and subsequent changes in depressive symptoms, β = .015, *t* (280) = 2.06, *p* < .05. The coefficient for this interaction indicates that when faced with an additional stressful event, individuals with a history of higher emotional abuse levels experience greater increases in depressive symptoms as compared to individuals with a lower abuse history. This interaction was examined for those without a history of emotional abuse compared to those with higher levels of emotional abuse (one standard deviation above the mean).

For each increase in emotional abuse events, the effect of dependent events increased depressive symptom outcomes by a standardized coefficient of .07 for those with higher abuse compared with a coefficient of .006 for those without a history of abuse. Although seemingly a small effect, this coefficient may indicate an important change in depressive reactivity, particularly for individuals with a chronic history of emotional abuse. In addition, we note that this represents unique variance not accounted for by other known risk factors (i.e., prior depressive episodes, sexual and physical abuse, and cognitive styles).

Follow-up analysis investigated the specificity of the interaction effect examining childhood sexual and physical abuse separately. The same base model was used to test the relationship between stress and depression, but the interaction term was substituted to indicate the effect of sexual or physical abuse controlling for the overlap of the other two types of childhood abuse. Three models were run for each type of abuse for overall, independent, and dependent stressful life events. Childhood physical abuse did not moderate the relationship between T1 stress and subsequent changes in depressive symptoms for any types of stress, overall: β = .010, *t* (280) = 1.04, *p* = .30; independent β = .009, *t* (280) = .787, *p* = .43; or dependent β = .027, *t* (280) = 1.45, *p* = .15. Similarly, childhood sexual abuse did not moderate the relationship between T1 stress and subsequent changes in depressive symptoms for any types of stress, overall: β = −.005, *t* (280) = 1.57, *p* = .12; independent β = −.005, *t* (280) = 1.34, *p* = .18; or dependent β = −.011, *t* (280) = 1.71, *p* = .09. Thus, only childhood emotional abuse moderated the relationship between dependent stress and depression.

Discussion

Our results provided support for the stress sensitivity hypothesis for emotional abuse history, in that individuals with a history of higher levels of emotional abuse experienced greater increases in depressive symptoms when confronted with dependent stressful life events. These findings are consistent with research examining the effects of early life stress on later stress response (Hammen et al., 2000; Kendler et al., 2004; Harkness et al., 2006; Hazel et al., 2008). This effect was significant only for dependent life events (Harkness et al., 1999; Kendler et al., 2006; for an exception see Harkness et al., 2006) and not independent events. This study is the first to examine the unique effect of childhood emotional abuse, a type of abuse that is consistently associated with depression (Alloy et al., 2006a), on individuals' reactions to current stressful events while controlling for the overlapping effects of other types of abuse. This suggests that emotional abuse confers a unique effect on an individual's stress response while also taking into account their overall levels of stress, prior levels of depression, and cognitive-risk status.

In addition, this effect was specific to childhood emotional abuse, as childhood sexual and physical abuse did not affect the relationship between stress and depression while controlling for the overlap of each other type of abuse. Experiences of early emotional abuse may be particularly maladaptive because negative evaluation is supplied directly by the primary attachment figures (Rose & Abramson, 1992). According to attachment theory (Bowlby, 1982), attachment figures help develop representational or internal working models of the world. Instead of developing a self-model that is characterized by self-worth, those who experience emotionally abusive comments may develop a more negative self-model and become prone to internalizing symptoms when confronted by dependent life events. Indeed, emotional abuse has been linked to enduring feelings of shame, humiliation, anger, and feelings of worthlessness (e.g., Barnet, Miller-Perrin, & Perrin, 2005). Further, experiences of emotional maltreatment may lead to an automatic association between life events and self-worth; thus, those who experience childhood emotional abuse have an increased automatic negative self-association in adulthood (Harmelen et al., 2010).

Early life stress may affect the stress system directly by conferring disruption in neurobiological development (Cicchetti, 2004). Early adversity has been associated with neurobiological changes, specifically in the hypothalamic-pituitary-adrenal (HPA) axis, the major neuroendocrine stress response system (Shea, Walsh, MacMillan, & Steiner, 2004). Heim and colleagues (2000) found that adult women with a history of childhood abuse had robust HPA abnormalities. In addition, early experiences of emotional abuse may lead to the development of an involuntary defeat strategy (Sloman, 2000), characterized by a psychobiological response to threats aimed to reduce the likelihood of further risk (Sloman, Gilbert, & Hasey, 2003). Although this response can be adaptive at times, it may also activate stress mechanisms that ultimately lead to a negative effect on mood (Sloman et al., 2003).

Finally, more recent research highlights the possibility of differential susceptibility to childhood emotional abuse based on genetic liability (Antypa & Van der Does, 2010). Antypa and Van der Does (2010) found that the polymorphism of the serotonin transporter gene moderated the relationship between childhood emotional abuse and depressive symptoms. Further research is needed to examine the contributions of emotional abuse to the vulnerability to depression and neuroendocrine response to stressful life events.

This study highlights an important pathway through which early experiences may confer a risk for depression, in that a history of childhood emotional abuse may sensitize an individual to current life stress, but other pathways are important to consider as well. In a

two time-point study, Hankin (2005) tested multiple potential pathways by which childhood emotional, physical, and sexual abuse may lead to depression in young adulthood. He found that only childhood emotional abuse was predictive of prospective changes in depressive symptoms, and that insecure attachment style, negative life events, and negative cognitive styles mediated this association.

In addition, research suggests that early life stress, including childhood abuse, may lead to the development of cognitive vulnerabilities, which confer a heightened risk to developing depression (Rose & Abramson, 1992; Gibb et al., 2001; see Alloy et al., 2006a for a review). Rose and Abramson's (1992) extension of the hopelessness theory may better explain the developmental pathway for childhood emotional abuse, as opposed to physical or sexual abuse, because the depressogenic interpretations are directly supplied to the individual by the abuser. Individuals with a history of childhood emotional abuse may interpret current negative events in a similarly more depressogenic manner consistent with the hopelessness hypothesis, which accounts for subsequent increases in depressive symptoms. For instance, Gibb et al. (2001) found that childhood emotional, but not physical or sexual, maltreatment was associated with both cognitive vulnerability and depression, and negative cognitive styles partially mediated the relationship of emotional maltreatment and the development of depression. Taken together, this study builds on prior research examining vulnerability models for depression as childhood emotional abuse may affect sensitivity to stress.

The current study of childhood abuse effects on an individual's stress reactivity has many strengths. It builds on prior research by examining both men and women in a relatively more diverse sample at two sites. In addition, this study employed rigorous statistical methods that take advantage of the multiwave prospective design over relatively brief intervals, which allowed for a sensitive analysis of the relation between life stress and depressive symptoms and individual differences in reactivity to stressful events. The current study builds on prior research by controlling for cognitive vulnerabilities to depression as well as other co-occurring forms of abuse. Further, controlling for depression in the models helps to reduce possible bias introduced by retrospective reports.

Although confidence in the current findings is heightened by the aforementioned strengths, results must be interpreted with caution because of the following limitations. First, as in previous studies (e.g., Hammen et al., 2000; Harkness et al., 2006), this study relied on a retrospective self-report of childhood abuse history, which may be biased based on individuals' subsequent experiences. In addition, the retrospective reports of emotional abuse may be more susceptible to biases than reports of physical and sexual abuse. However, research suggests that adults' recall of childhood events is relatively accurate (Brewin, Andrews, & Gotlib, 1993; Bifulco, Brown, Lillie, & Jarvis, 1997); consequently, research consistently employs this method, which has been found to be accurate and reliable (e.g., Gibb et al., 2001).

Second, the report of current life stress also relied on self-report and it may be biased because of a history of abuse. The use of a semistructured interview was employed to combat reporter bias on current life stress. Semistructured stress interviews are currently the gold standard in assessing life stress (Monroe & Reid, 2009); the current study employed an interview based on the Brown and Harris (1978) stress interview, whereby reported events must meet objective a priori definitional criteria. In contrast to self-report checklists of life events, interviews are less biased by the participants' potential (e.g., mood congruent bias) subjective interpretations of events. Moreover, interviewers were blind to participants' abuse history and risk status. Third, a strength and limitation of the study was that participants comprised a selected sample of college-age students based on their risk for depression. This

design was used to increase variability on the constructs of interest; however, the results may be limited in their generalizability.

This study examined the differential effect of independent and dependent events on stress response based on the importance of this distinction in prior research (e.g., Kendler et al., 2006). Other categories of stressors merit consideration, including but not limited to, interpersonal and achievement stressors, which have been shown to be associated with depressive outcomes (e.g., Eberhart & Hammen, 2010; Carter & Garber, 2011). In addition, we operationalized stress as the sum of discrete stressful events that occurred. Further research could differentiate the level of stress by objective effect, as stressful events may not all be equal.

Finally, findings from the current study did not examine episodes of depression or clinician-rated symptom levels as the data obtained were of self-reported depressive symptoms. Thus, interpretations of the results should be made with the aforementioned methodological considerations. Despite its limitations, this study provides evidence for stress sensitization using a two-site, multiwave, high-risk, and longitudinal design with conservative analytic methods.

Importantly, implications from this study suggest that an individual's history of emotional abuse leads to an increased reactivity to stressful life events after controlling for the reactivity associated with cognitive vulnerabilities. This suggests that childhood emotional abuse may be particularly maladaptive, highlighting the need for more research targeted at emotional abuse specifically within a longitudinal framework, as much previous research examines only sexual and physical abuse (Alloy et al., 2006a) or uses composite scores of total abuse (Harkness et al., 2006).

A second important direction for research is to determine the mechanisms through which childhood emotional abuse influences individuals' reactivity to future life stress. Results emphasize the importance of early intervention for those who experience higher levels of emotional abuse. Individuals with a history of abuse may especially benefit from therapy that focuses on understanding the relationship between stress and depression and effectively coping with negative life events, which has been found to be efficacious in the treatment and prevention of depression (Nemeroff et al., 2003; Garber et al., 2009). Although the finding of a specific relationship between childhood emotional abuse and stress reactivity warrants further replication, this study highlights the particular pathogenic nature of emotional abuse.

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Table 1

Demographic, Stress, and Abuse Information Based on Study Inclusion

	Low risk *M* (*SD*)	High risk *M* (*SD*)	*t*	Total *M* (*SD*)
Age at T1	18.91 (1.92)	18.60 (1.23)	1.64	18.76 (1.64)
Time in study	28.25 (15.54)	28.32 (15.61)	−.26	28.28 (15.57)
Female	.68 (.47)	.67 (.47)	0.15	.68 (.47)
Depressive symptoms at T1	1.82 (2.97)	5.89 (6.68)	−6.49***	3.76 (5.47)
Overall events at T1	5.08 (5.66)	6.87 (7.44)	−2.25*	5.93 (6.62)
Dependent events at T1	1.78 (2.56)	2.35 (2.75)	−1.81	2.05 (2.67)
Independent events at T1	3.31 (4.01)	4.51 (5.22)	−2.16*	3.88 (4.66)
Emotional abuse	3.07 (3.60)	3.37 (3.79)	−.66	3.20 (3.67)
Physical abuse	1.07 (1.43)	1.30 (1.42)	−1.31	1.18 (1.42)
Sexual abuse	.73 (1.54)	.76 (2.07)	−.15	.75 (1.82)
Total N	147	134		281

Note. M = mean; SD = standard deviation.

Independent samples *t* tests compared low risk vs. high risk.

* $p < .05$.

** $p < .01$.

*** <.001.

Table 2

Effect of Stress on Depression Scores for Childhood Emotional Abuse

	Overall stress		Independent stress		Dependent stress	
Variable	**Coefficient (*SE*)**	**Coefficient (*SE*)**	**Coefficient (*SE*)**	**Coefficient (*SE*)**	**Coefficient (*SE*)**	**Coefficient (*SE*)**
Within-subject level						
Lagged stress	.021 (.008)**	.017 (.007)**	.017 (.009)§	.015 (.008)§	.063 (.020)**	.056 (.017)**
Lagged depressive symptoms	.363 (.031)***	.363 (.031)***	.366 (.031)***	.365 (.031)***	.362 (.031)***	.361 (.031)***
Time	−.006 (.002)**	−.006 (.002)**	−.006 (.002)**	−.006 (.002)**	−.006 (.002)**	−.006 (.002)**
Between-subject level						
Female	.361 (.118)**	.361 (.118)**	.360 (.118)**	.360 (.118)**	.361 (.118)**	.362 (.118)**
Risk status	.851 (.134)***	.852 (.135)***	.848 (.134)***	.849 (.134)***	.853 (.135)***	.854 (.136)***
Prior depression	.296 (.156)§	.296 (.156)§	.295 (.155)§	.295 (.156)§	.296 (.156)§	.297 (.156)§
Prospective episodes	.909 (.340)**	.910 (.340)**	.905 (.339)**	.906 (.339)**	.911 (.341)**	.912 (.341)**
Childhood emotional abuse	.014 (.025)	.015 (.025)	.014 (.025)	.014 (.025)	.015 (.025)	.015 (.025)
Childhood physical abuse	.000 (.059)	.000 (.059)	.000 (.058)	.000 (.059)	.000 (.059)	.000 (.059)
Childhood sexual abuse	.067 (.039)§	.068 (.039)§	.067 (.039)§	.067 (.039)§	.068 (.039)§	.068 (.039)§
Cross-level interactions						
Emotional abuse x lagged stress		.006 (.004)		.006 (.005)		.015 (.007)*
Variance components						
Within-subject level	5.49	5.47	5.49	5.48	5.48	5.47
Proportion reduction in variance		0		0		0

Note. Lagged stressful events was person-mean centered; childhood emotional abuse was grand-mean centered.

§ $p < .1$.

* $p < .05$.

** $p < .01$.

*** < .001.