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Moderate Childhood Stress Buffers Against Depressive Response to Proximal Stressors: A Multi-Wave Prospective Study of Early Adolescents

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Abstract

Although the majority of research in the field has focused on childhood stressors as a risk factor for psychopathology, a burgeoning body of literature has focused on the possible steeling effect of moderate types of stressful events. The current study investigated the effects of proximal life stressors on prospective changes in depressive symptoms, and whether a history of moderate childhood adversity would moderate this relationship in a multi-wave study of a diverse community sample of early adolescents ($N = 163$, 52% female, 51% Caucasian). Hierarchical linear modeling was run with four waves of data. Adolescents with greater moderately severe early life events evinced a blunted depressive symptom response to changes in proximal stressful events in the previous 9 months, compared to those with fewer early moderately severe experiences of adversity. These results held after controlling for between-subject factors such as race, gender, severe early life stress, and average stress over the four waves of data. Findings indicate that greater exposure to moderate childhood stressors may buffer against the negative effects of subsequent stressors, suggesting the importance of a nuanced developmental approach to studying the effects of early life stress.

Keywords

early life stress; depression; attenuated stress response; buffering; steeling; resilience

To varying degrees, stress is a part of everyday life. The preponderance of evidence suggests that exposure to severe or chronic stressful life events predicts increased risk for the development of psychopathology (Coyne & Downey, 1991; Grant, Compas, Thurm, McMahon, & Gipson, 2004; Kessler, Price & Wortman, 1985). Although this relationship is evident for many psychological outcomes, an accumulation of research demonstrates a

consistent and strong association between exposure to stressful life events and an increase in depressive symptoms and onset of major depression (Abela & Skitch, 2006; Hammen, 2005; Kessler, 1997; Mazure, 1998), particularly during the adolescent years (Ge et al., 1994). However, a burgeoning body of literature has indicated the possibility that exposure to moderate stressors may decrease vulnerability through a steeling effect (c.f. Boyce & Ellis, 2005). The present study explores the possible buffering effects of moderate childhood adversity on adolescents' reaction to current life stress in developing depressive symptoms.

Early Childhood Stressors

There are normative developmental changes in how individuals react to and process stressful life events (Compas, 1987; Dahl & Gunnar, 2009). Although several factors have been shown to lead to dysregulation of the stress response and changes in normal trajectories, exposure to severe early life stressors consistently has been demonstrated to have long-term deleterious effects on individuals' stress responses (Gunnar & Quevedo, 2007; Tarullo & Gunnar, 2006) and the development of psychopathology (McLaughlin et al., 2010). Specifically, forms of severe childhood life stress, such as physical abuse and neglect and sexual abuse, have been linked to heightened physiological responses to later stressful events (Harkness, Steward, & Wynne-Edwards, 2011; Heim et al., 2002; Shea et al., 2005), and are associated with elevated risk for developing mood and anxiety disorders in adolescence and adulthood (McLaughlin et al., 2010a).

Although childhood trauma and maltreatment may increase the risk of depression, research has shown that the role of stress may change over the course of depression and exposure to stressful events over the lifetime (for reviews, see Monroe & Harkness, 2005; Stroud, Davila, & Moyer, 2008). 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 depressive recurrences. This change in sensitivity after an episode of depression has been extended to examine other variables that may confer a similar sensitization to life stress. For example, childhood sexual, physical, and emotional abuse and neglect have been linked to a lower-threshold for subsequent stressors to elicit a depressive response (Harkness, Bruce, & Lumley, 2006; McLaughlin, Conron, Koenen, & Gilman, 2010b; Shapero et al., 2014), which suggests that individuals experience heightened reactivity to current stressors as a result of greater exposure to severe childhood adversity. When children experience adversity, they may try to understand the causes and consequences of these events and may be more likely to make depressogenic inferences about themselves if these events are severe or recurrent. Indeed, research suggests that early life stress may lead to the development of cognitive vulnerabilities, which confer a heightened risk to developing depression, particularly when confronted by additional stress (Alloy et al., 2006 for a review; Gibb et al., 2001; Rose & Abramson, 1992).

In addition to childhood trauma, research also has found that severe early negative life events (such as parental loss or death) sensitized individuals to depression when exposed to proximal stressful events (Hammen, Henry, & Daley, 2000; Rudolph & Flynn, 2007). Importantly, the sensitization theory suggests that a lowered threshold to episodes of depression is present for those who experienced severe early childhood adversity. Thus,

when exposed to low levels of proximal stress, individuals who experience severe childhood adversity would be at greater risk for depression than those without such adversity. In contrast, following high levels of proximal stress, individuals with and without childhood adversity would have higher depressive symptoms regardless of previous exposure to childhood stress. In this sense, less stress would be sufficient to elicit a depressive response for individuals who have been exposed to severe childhood adversity.

Attenuated Stress Response

Although there is support for the stress sensitization theory (e.g., Stroud et al, 2011), recent research and theory posit that exposure to moderate adversity in childhood environments may buffer against the effects of proximal stress and promote a more adaptive response to future stress (Ellis & Boyce, 2008). Most research on the attenuated stress response has been investigated and supported in animal models and examinations of the neuroendocrine response system.

Overall, evidence from animal models provides support for the development of a steeling effect from encountering moderate types of early life stress (Lyons & Parker, 2007; Lyons, Parker, & Schatzberg, 2010). For example, experiments in young primates who were experimentally exposed to repeated experiences of brief stress before returning them to their usual housing conditions showed a reduction in sensitivity to later stress experiences (Lyons & Parker, 2007). Additionally, Macri and Wurbel (2007) examined whether stressful changes in early environments based on maternal attention affected later behavioral and biological responses to adult stressors in rodents, finding that moderately challenging maternal environments led to a down-regulation in the biological response to later stress (Macri & Wurbel, 2007). Thus, in multiple animal models, early life stressors of moderate severity have been linked to a reduction in reactivity to subsequent stressors as opposed to the vulnerability that is evident after severe early stressors.

In addition, in the last decade, advances in research examining the neuroendocrine system in humans support the assertion that early negative life experiences alter the stress response. Whereas some research suggests that exposure to childhood adversity, trauma, and abuse promotes hyperactivity of the hypothalamic-pituitary-adrenal axis system that results in sensitization to the effects of later stressors (c.f. Heim & Nemeroff, 2001), other research suggests an attenuation or blunting of cortisol output in those who experience negative events during childhood. For example, Gunnar and colleagues (2009) compared the responses to stress in individuals who experienced low, moderate, or severe experiences of early life stress. They found that individuals who experienced a moderate level of early life stress exhibited a blunted stress response, whereas those who experienced a severe level of early life stress did not differ from the low stress group in their reactivity to stress. Taken together, evidence from studies examining the stress response suggest that experiences of some stress during childhood may attenuate the response to stress during adulthood and perhaps enhance one's ability to overcome the stress associated with everyday life.

Few studies have evaluated the attenuated stress response in relation to episodes and symptoms of depression. However, a recent longitudinal study found that individuals who

were exposed to some lifetime stress reported better mental health outcomes compared to those with high levels of stress and those with no history of adverse life experiences (Seery, Holman, & Sliver, 2010). Additionally, in a study that examined the differences between specific forms of childhood maltreatment and risk for depression in adults, the severity of childhood maltreatment was related to increased risk (Fisher et al., 2013). Childhood experiences were coded as mild, moderate, and severe and participants who had the most severe experiences were at increased risk for depression compared to those with moderate and mild severity of experiences (Fisher et al., 2013). These findings suggest that some stress may be adaptive and protect against the development of depression following proximal stress.

Moderate Stress Exposure and Adolescence

Importantly, more research is needed in humans to evaluate the potential buffering nature of exposure to not only moderate levels of early life stressors, but also moderate types of early life stressors in the development of psychopathology. Although much research has supported the adverse effects of severe forms of early life stress, considerably less work has examined the effects of moderate types of stressors (see Edge et al., 2009 for exception). To date, there is no general consensus on what differentiates moderate versus severe forms of stressors; however, severe forms of stress have been characterized as including experiences of abuse and neglect (e.g., sexual and physical), death of a close family member, traumatic life experiences (e.g., victim of crime), or getting into serious trouble with the law (e.g., Pettit et al., 2010). On the other side of the spectrum may be daily hassles, such as not having enough money for hobbies, working under strain, or mild teasing by other children (Kanner et al., 1987, Pettit et al., 2010). In contrast, moderate types of stressors include more commonplace events that are more impactful than daily hassles such as conflict with or neglect by family or peers, academic failure, and difficult living situations, among others (Edge et al., 2009). Exposure to stress that is not overwhelming (e.g., moderate stressors) may promote the development of adaptive regulation in response to later stress. Whereas early investigations of the effects of moderate life stress have been conducted in adults (e.g., Seery et al., 2010), examination in the transitional developmental period of adolescence is lacking.

Adolescence is a pivotal developmental time period in which school transitions, adjustments in social support, and puberty give rise to dramatic increases in environmental stressful events (Ge et al., 1994; Rudolph & Hammen, 1999). Indeed, concurrently the rates of depression increase during adolescence (Hankin et al., 1998), making it an important time to examine factors that may influence depressive reactivity to proximal stressors.

Current Study

The majority of prior studies in humans have examined effects of relatively severe forms of life stress, such as maltreatment and trauma (e.g., Espejo et al., 2006; Harkness et al., 2006; Kendler, Kuhn, & Prescott, 2004). However, the current study builds on prior research to examine whether the occurrence of moderate adversity in childhood buffers against the depressogenic effects of proximal stressors in adolescence. Whereas some research supports

sensitization to the effects of severe forms of early life stress on responses to later stressors, there may be a protective effect among individuals who experience some stress during childhood in overcoming proximal stressors. That is, it may not be prudent to characterize all effects of early life stressors as harmful, as some research suggests that some exposure to stressful events as a child may be helpful in developing the ability to cope with the inevitable occurrence of stressful events later in life. Taken together, we hypothesize that experiences of moderate stress during childhood will attenuate the depressive response to proximal stressors in adolescence. Although, consistent with prior studies examining the effects of stress, we believe that proximal stress will elicit higher levels of depressive symptoms overall; however, we predict that the depressive reaction to proximal stressors will be weaker among adolescents exposed to moderate stressors during childhood.

Method

Sample Recruitment

The overall sample was recruited as part of a longitudinal study designed to evaluate the emergence of depressive disorders among male and female, Caucasian and African American adolescents. One of the goals of the broader study was to examine racial differences, and thus, adolescents with other racial backgrounds were excluded. Prior research suggests possible differences between Caucasian and African American adolescents in the experiences of stress, depressogenic cognitions, and internalizing symptoms (Anderson & Mayes, 2010; Hamlat et al., 2014). However, examining racial differences was not a primary aim of the current study. Adolescents were recruited from Philadelphia-area middle schools through either school mailings and follow-up phone calls (approximately 68% of the sample) or advertisements placed in area newspapers (approximately 32% of the sample). Potential participants completed a screening by phone to determine eligibility, which required being 12 or 13 years old, self-identifying as Caucasian/White, African-American/Black, or Biracial (Hispanic adolescents were eligible if they also identified as White or Black), and having a mother or primary female caregiver also willing to participate in the study. Given the small number of adolescents identifying as biracial ($n = 9$), they were included in the non-white group. Adolescents were not eligible for the study if there was no mother/primary female caregiver available to participate; the mother or adolescent was psychotic, mentally retarded, had a severe learning disability or a severe developmental disorder; or the mother or adolescent was unable to read or speak English or could not complete assessments for any other reason (see Alloy et al., 2012 for further details regarding recruitment).

Study Sample

The larger study aimed at investigating risk for depression in early adolescence recruited 413 adolescents from the community (Alloy et al., 2012). The current sample was drawn from this larger sample based on completing at least three follow-up assessments. The current sample consisted of 163 adolescents (Mean age at enrollment = 12.83 years; $SD = 0.63$) who completed an initial baseline assessment (Time 1) and at least three of four follow-up assessments (Times 2, 3, 4, and 5), each of which were approximately nine months apart. Of the 163 adolescents included in the current sample, 66 (40%) completed

three follow-up assessments and 97 (60%) completed all four assessments. Participants in the current study did not differ from the overall sample on Time 1 depressive symptoms, Time 1 stress, early life stressors, or any demographic variables. Fifty-two percent of the sample was female and 49% were African-American. Twenty-three percent of participants had family incomes falling below \$30,000, 28% between \$30,000 - \$59,999, 20% between \$60,000 - \$89,999, and 29% above \$90,000. Forty-one percent of participants were eligible for free school lunch, a measure of financial need that accounts for the number of dependents being supported on the family's income.

Procedures

At Time 1, adolescents completed a self-report questionnaire evaluating current depressive symptoms, and adolescents' mothers completed a questionnaire assessing the occurrence of stressful events that occurred to the adolescent during childhood prior to study enrollment. At the Time 2, 3, 4, and 5 assessments, adolescents completed self-report questionnaires assessing current depressive symptoms and the occurrence of negative events since the previous assessment, and then were interviewed about those negative life events with a semi-structured interview. Rates of participation in the follow-up surveys were as follows: Time 2 ($N = 141$, 86.5%), Time 3 ($N = 124$, 76.1%), Time 4 ($N = 147$, 90.2%), and Time 5 ($N = 147$, 90.2%).

Measures

Depressive Symptoms—The Children's Depression Inventory (CDI; Kovacs, 1985) is a self-report questionnaire that contains 27 items to assess affective, behavioral, and cognitive symptoms of depression in youth ages 7 to 17. Each item is rated on a scale from 0 to 2; items are summed for a total score (ranging from 0 to 54), with higher scores indicating more depressive symptoms. The CDI has good reliability and validity (Klein, Dougherty, & Olino, 2005). This measure was given at all 5 assessments. Internal consistency in our sample ranged from α 's = .85 to .88.

Childhood Stressful Life Events—The Children's Life Events Scale (CLES; Crossfield, Alloy, Gibb, & Abramson, 2002) is a checklist of 50 negative events of moderate to major severity that children may experience in their lifetime expanded from the Source of Stress Inventory (Chandler, 1981). The current study purports to examine the effect of moderate adversity on current responses to proximal stressful events. Moderate early life adversity was operationalized as the experience of greater exposure to (or higher levels of) less extreme types of stress in childhood. Thus, to be consistent with the main study goals, the original CLES was coded to create events of moderate versus severe types of early childhood experiences. Events were coded by a team of four doctoral students/clinical psychologists into moderate or severe categories ($\kappa = .83$). Severe forms of abuse (e.g., physical/sexual abuse, neglect) or traumatic events (e.g., death of parent or sibling, out of home placement of child) were included in the severe category and had a low base rate in the current sample (4% of reported events). Moderate items include events in the following domains: achievement failures (e.g., bad grades in school; academic failure), family difficulties (e.g., increase in number of arguments with parents; family financial difficulties), interpersonal problems (e.g., frequent teasing of child by peers; increased arguments with

siblings; romantic break-up), and other life stressors (e.g., family moved, close friend moved away, overcrowded living situation). Mothers, not adolescents, completed the CLES because children may have been too young when some stressful events occurred to recall all of them independently. Scores on the CLES can range from 0 to 46, with higher scores indicating more experiences of moderate types of negative events. Several studies (e.g., Crossfield et al., 2002; Grandin, Alloy, & Abramson, 2007) have demonstrated predictive validity of the CLES. The CLES was given at Time 1.

Proximal Negative Life Events—Proximal life events were evaluated with the Adolescent Life Events Questionnaire (ALEQ; Hankin & Abramson, 2002), which was designed to assess the occurrence of a broad range of negative or stressful events often reported by adolescents, including familial (e.g., “you fought with your parent(s) over personal goals, desires, or choice of friends/boyfriend/girlfriend”), peer (e.g., “you had an argument or fight with a friend(s)”), and school/achievement events (“you did poorly on or failed a test or school project”). Adolescents and their mothers separately completed the 63-item ALEQ at Times 2, 3, 4, and 5 and indicated events that occurred in the adolescent’s life since the previous assessment. Following completion of the ALEQ, adolescents completed the Life Events Interview (LEI; Safford, Alloy, Abramson, & Crossfield, 2007) with trained interviewers who evaluated whether events endorsed by the adolescents and/or their mothers on the ALEQ met a priori event definition criteria for each event, which was designed to limit the influence of depressogenic bias on event reporting. For example, if an adolescent reported that his/her parents had an argument, a priori criteria for this event stated that the argument had to include a serious consequence, such as yelling, crying, name-calling, door slamming, and not speaking to each other. Any events that did not meet criteria were disqualified, reducing potential problems related to subjective report biases. Interviewers also verified that events occurred within the timeframe since the previous assessment. The life event variable used in the current study reflects the sum of the number of types of events reported by adolescents that were verified by the LEI. Reliability and validity have been established for the ALEQ (Hankin, 2008; Hankin & Abramson, 2002; Stange, Hamilton, Abramson, & Alloy, 2014) and for the LEI (Safford et al., 2007).

Results

Statistical Approach

Hierarchical linear modeling (HLM; Raudenbush & Bryk, 2002) was used to investigate differences between adolescents’ depressive reactions to recent stressful life events as a function of their experiences of moderate childhood stress. HLM is a rigorous statistical method for approaching this question because it can represent both changes within a person over multiple time points while also ascertaining how individuals may differ from one another over time (Curran & Willoughby, 2003). Ordinary Least Squares (OLS) 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 163 individuals and 559 observations were included in the final models using full maximum likelihood estimation. Independent samples t-tests were used to assess whether demographic variables were associated with depressive symptoms; these tests showed that race was not significantly associated with CDI scores, $t(161) = 0.54$, *ns*, or stress, $t(161) = .34$, *ns*, but sex was associated with depressive symptoms, $t(161) = 326$, $p < .01$. Both race and sex were included in the analyses as possible confounds in the relationship between childhood stress and sensitivity to proximal stressors¹. Bivariate correlations of the main study variables showed that there were significant positive correlations between proximal stressful life events, depressive symptoms, and childhood stressors overall (Table 1). Severe childhood stressors generally were not directly correlated with proximal stressors or symptoms at each time period.

Preliminary HLM analyses examined the main effects of early moderate stress on average depressive symptoms to reveal whether moderate early life stress is an overall and unique predictor of depression across this longitudinal period. First, only moderate early life stress was entered in the between-subject level and depressive symptoms were entered as the outcome. Results suggest that moderate early life stress predicted higher average depressive symptoms when entered individually, $\beta = .15$, $t(161) = 2.39$, $p < .05$. However, as seen in Table 2, when including other between-subject variables, moderate early life stress was no longer significantly predictive of average depressive symptoms, whereas more proximal stressors were more strongly correlated with depressive symptomatology.

Main Study Analysis

In the main HLM analysis, the within-subject (time-varying) level assessed whether levels of proximal stressful events in the prior period predicted changes in depressive symptoms. The cross-level interactions assessed whether between-subject history of higher levels of moderate childhood stressors moderated the relationship between proximal stressors and depressive symptoms. The outcome variable for all analyses was depression symptom (CDI) scores in the current period (T), controlling for previous period CDI (Lagged CDI) scores (T-1). 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 Stress, which represents the number of negative life events that occurred since the previous period, similar to prior studies (e.g., Auerbach et al., 2011; Calvete, Orue, & Hankin, 2013; Shapero, Hankin, & Barrocas, 2013). 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. The participant's sex and race were entered at the between-subject level of analysis to control for possible sex and race differences. Additionally, we included Average Stress in level 2 to account for individual differences in stress to isolate within-subject fluctuations in stress after accounting for between-subject effects. This measure was grand-

¹Based on prior studies examining gender differences in adolescent depression, a three-way interaction was used to explore whether sex moderated the relationship between moderate early life stressors and proximal stress on symptoms. Results did not support a three-way interaction, $\beta = .01$, $t(161) = .99$, $p > .05$. Additionally we ran a three-way interaction to explore whether race moderated this relationship. Results did not support a three-way interaction, $\beta = -.01$, $t(161) = .53$, $p > .05$.

mean centered to account for overall differences. The continuous measure of history of moderate childhood stressors (CLES) was entered to examine whether a between-subject history of higher levels of moderate stressors during childhood attenuated depressive reactivity to proximal stress as measured by depressive symptom levels. Severe childhood stressors were included as a covariate to account for the occurrence of severe forms of adversity, although base rates of severe childhood events were low. Finally, the cross-level interaction of moderate childhood stressors (CLES) x Stress enabled us to evaluate the attenuation hypothesis of the study. The intercept-only model, $\beta = 5.04$, $t(161) = 14.49$, $p < .001$, revealed an intra-class correlation (ICC) of .51. Therefore, 51% of the variance in depressive symptoms is due to between subject variance and 49% of the variance in depressive symptoms is due to within subject variance.

Results suggested that history of moderate childhood stressful events moderated the effect of total proximal stressful life events on depressive symptoms (Table 2). The main effects of this model suggested that individuals with higher levels of depressive symptoms, $\beta = .40$, $t(161) = 6.65$, $p < .001$, and higher levels of total proximal stressors, $\beta = .28$, $t(382) = 5.49$, $p < .001$, at the previous time period, had higher levels of current depressive symptoms, suggesting an increase in symptoms over time. In addition, between-subject factors, such as higher average stress, $\beta = .32$, $t(161) = 7.12$, $p < .001$, and more moderate childhood stressful events, $\beta = .07$, $t(161) = 2.06$, $p < .05$, predicted increases in current depressive symptoms, suggesting the possible risk associated with moderate early life stressors. Of note, however, in the interaction model, a history of moderate childhood stressful events moderated the relationship between the T-1 total stressors and subsequent changes in depressive symptoms, $\beta = -.02$, $t(382) = 2.45$, $p < .05$. The coefficient for this interaction indicated that when faced with additional proximal stressful events, individuals with a history of higher moderate childhood stressful events experienced an attenuated depressive response as compared to individuals with a lower history of childhood stress (See Figure 1). The simple slopes of the model were tested for one standard deviation above and below the mean (Aiken & West, 1991) for childhood moderate stressful events. Following the procedure suggested by Preacher, Curran, and Bauer (2006), the slope for the relationship between proximal total stressors and depressive symptoms for adolescents who experienced a low level of moderate early childhood stressors was significant and was larger, ($t = 5.34$, $p < .001$) than for adolescents who experienced a high level of moderate early childhood stressors ($t = -2.43$, $p < .01$); (t for the difference between the slopes = 3.21 , $p < .01$).

Discussion

Although there is considerable evidence that severe forms of early stressful life events predict increases in depressive symptoms among youth and adults (e.g., Hammen, 2005; Mazure, 1998), less is known about the influence of exposure to moderate childhood stress on the development of depressive symptoms following proximal stressors. Consistent with prior research on the impact of stressful events on depressive symptoms during adolescence (e.g., Ge et al., 1994), the present study found that higher levels of proximal stressful events longitudinally predicted increases in depressive symptoms during adolescence. However, adolescents who experienced higher levels of proximal stressful events were not equally vulnerable to increases in depressive symptoms. Consistent with our hypotheses, the present

study suggested the presence of a steeling effect, such that exposure to moderate stressors during childhood contributed to an attenuated depressive response to proximal negative events during adolescence. Taken together, this is the first known study to find evidence of an attenuated depressive response following proximal stressors among a community sample of adolescents exposed to moderate childhood early life stressors.

Our finding of a steeling effect of childhood early life stress indicates that exposure to moderate stressors during childhood may buffer youth from the detrimental effects of stressful events during adolescence. These findings are consistent with recent studies by Seery and colleagues (Seery Holman, & Silver, 2010; Seery, Leo, Lupien, Kondrak, & Almonte, 2013) indicating that some exposure to childhood adversity is beneficial for psychological well-being and mental health among adults, but extends these findings to an adolescent sample and to moderate types of childhood adversity in addition to moderate levels of childhood adversity. Particularly given the dramatic increase in the occurrence of stressful events and depressive symptoms during adolescence (Ge et al., 1994; Hankin et al., 1998; Larson & Ham, 1993), this is an important period to identify factors that confer vulnerability to or promote resilience against depression in the face of proximal stress.

The current findings also extend past research on the effects of childhood adversity on depressive responses to proximal stressors. Most research on childhood adversity has focused on the effects of more severe forms of childhood stress, such as sexual, physical, and emotional abuse and neglect. These studies consistently have demonstrated that severe childhood adversity sensitizes individuals to later stressors, such that a lower number of proximal stressful events are needed to trigger a depressive response (e.g., Hammen et al., 2000). In contrast, our study focused on more moderate types of childhood stress (e.g., moving to a new town, parental hardship), which appears to differentially influence responses to later stressors. Given that more severe childhood adversity elicits a sensitization effect (e.g., Rudolph & Flynn, 2007) and more moderate childhood adversity appears to buffer against the depressive effects of proximal stressors, the distinction between severe and moderate childhood stressors may be a significant indicator of how individuals will respond to proximal stressors later in life. These findings highlight the importance of assessing many different types of childhood stressors during adolescence, and future research is needed to further elucidate the differentiation between mild, moderate, and severe events during childhood.

There are several possible mechanisms through which moderate childhood stressors may contribute to an attenuated depressive response to proximal stressors during adolescence. For instance, moderate childhood stressors may provide youth with more opportunities to learn how to effectively manage stressful events later in life. Specifically, youth may learn more coping strategies for responding to stress, such as utilization of social support and positive cognitive appraisals, relative to children who are exposed to little or no adversity (Compas, 1987; Compas et al., 2001). In fact, research has found that internal abilities of coping and cognitive hardiness buffer against the effects of life stress (Beasley, Thompson, & Davidson, 2003). Additionally, youth exposed to moderate stressors may develop beliefs about their self-efficacy and mental “toughness” for responding to stress (Dienstbier, 1989), which may encourage feelings of control and make later stressors seem more manageable.

Thus, the occurrence of moderate stressors in childhood may better prepare youth cognitively and emotionally for the challenges of adolescence, particularly as it relates to coping with proximal stressors. In contrast, severe childhood stressors may overwhelm youths' resources to manage stress and disrupt their ability to cope. Whereas moderate stressors might give youths a "healthy" dose of stress to overcome, severe stressors may leave "scars" that increase vulnerability for depression (e.g., Rohde, Lewinsohn, & Seeley, 1990). Specifically, severe childhood stressors are associated with poorer emotion regulation strategies (Cicchetti & Toth, 1998) and more dysfunctional cognitions (Gibb, 2002), which may ultimately heighten reactivity and confer risk for depression. Further, Rose and Abramson's (1992) extension of the hopelessness theory helps explain the pathway for severe childhood emotional abuse predicting the development of cognitive vulnerabilities, as the depressogenic interpretations are directly supplied to the individual by the abuser. Individuals with a history of childhood abuse may interpret current negative events in a more depressogenic manner consistent with the hopelessness hypothesis, which may be one mechanism for subsequent increases in depressive symptoms. For example, research supports the notion that childhood emotional abuse is associated with later depression, and that negative cognitive styles mediated this relationship (Gibb et al., 2001). Additionally, individuals who are exposed to little or no adversity during childhood also may be at greater risk due to their overprotected environment. For instance, adolescents who have not previously encountered stressful events may not be prepared to manage stressful events later in life, particularly during the vulnerable period of adolescence. Adolescents without adversity may not have the coping skills or have developed the social support system to handle stressful events, and thereby may have heightened reactivity to proximal stressors in the form of depressive symptoms.

Although this is among the first studies to investigate the buffering effect of childhood adversity in relation to depressive symptoms among adolescents, our results are consistent with recent studies of animal models (e.g., Parker & Maestripieri, 2011). Animal models are able to conduct highly controlled experiments that lead to conclusions of causality, but are limited in understanding the impact of psychological stressors that are generalizable to humans. The current study builds on this research in identifying the accumulation of moderate stressors that may buffer against depression in early adolescence and that may be used to inform the quality of stressors in animal models. Further, several studies of biological reactivity also are consistent with the notion that moderate childhood adversity may be more beneficial than mild or severe childhood stressors (Gunnar et al., 2009). Several recent theoretical and empirical studies also suggest a curvilinear relationship in the effect of early life stress, demonstrating that moderate levels of childhood stress reduce biological reactivity to induced stress (Boyce & Ellis, 2005). Thus, it is possible that exposure to moderate childhood adversity may result in a more global physiological and psychological habituation or resilience to future stressors, which may also explain the reduced depressive response to stressors.

The present study had a number of strengths. First, the current study used a prospective, multi-wave design and employed an idiographic approach to data analysis, which allows for the investigation of individual differences in depressive symptoms and proximal stressors over time. The current study also included multiple informants for the measurement of

stressful events, including maternal report of moderate childhood stressful events and both maternal and adolescent reports of proximal stressful events. The adolescent report of proximal stressors was further assessed using an interview method to address possible reporter bias due to individual differences and is considered to be the standard for measuring life events (Hammen, 2005). Additionally, this study examined a large community sample of demographically diverse adolescents, which strengthens the generalizability of our findings.

Although the present study had strengths, there also were a number of limitations that should be noted. First, as with many studies assessing emotional experiences, the current study only utilized self-report measures of depressive symptoms. However, given that depressive symptoms in adolescence are associated with functional impairment (Kessler, Zhao, Blazer, & Swartz, 1997) and predict the onset of major depression in adulthood (van Lang et al., 2007), depressive symptoms warrant attention in addition to diagnoses. Nonetheless, future work should conduct diagnostic interviews to determine the generalizability of these findings to high-risk or clinical populations. Additionally, we used stress at the prior time point to predict changes in depressive symptoms through a lagging process. This type of analysis allows for a clear distinction between prior stress and current reported depression controlling for past reported depression, but the potentially long duration between stressful events and depressive symptoms may imprecisely measure the more proximal effects of stress on mood. The effects of stressful events on depressive symptoms in individuals may occur more quickly than reported in the current manuscript and thus, our reported effects may be underestimations of the true effect sizes of proximal stress on depressive symptoms. This is an important area for further inquiry using alternative methodological approaches such as daily diary studies or ecological momentary assessment (e.g., Hankin, Fraley, & Abela, 2005; Mezulis & Rudolph, 2012). Additionally, future research should evaluate the congruency between domains of early life stressors and resiliency or vulnerability to later types of stressors in the same domain, as well as the effects of the severity of the proximal life events. This also may be helpful to ascertain experiences of childhood adversity as the impact of events on a particular person may vary based on their personal context.

Importantly, the current study only examined the effects of moderate childhood stressful life events due to the measurement that was used, and did not test the effects of childhood stressors that were severe or mild. Therefore, future research should conduct a more direct examination of a curvilinear relationship between early life experiences and later reactivity, which would provide more information regarding the potential adaptive nature of moderate levels of early life stress in buffering reactions to proximal stressors (Ellis & Boyce, 2008). Further, the present study evaluated moderate types of stressors; however, there is no general consensus in the literature on the types of stressors that constitute moderate versus mild and severe, and there may be individual differences in perception of stressors as mild, moderate, and severe in childhood. Research is generally clear on the differences between severe and non-severe stress, but the distinction of a more fine-grained understanding of moderate stress is needed. Further, it is unclear what frequency of moderate stressful events may be adaptive versus maladaptive. Additionally, the mechanism through which moderate childhood adversity attenuates depressive symptoms to proximal stressors remains unclear. Thus, future research should evaluate the potential processes through which moderate childhood adversity buffers the effects of proximal stressors on depression, such as coping

strategies and other resilience factors (Rutter, 1987; Seery et al., 2013). Results also cannot rule out possible formal external interventions, such as therapy or other forms of treatment, as a mechanism through which responses to current stressors were attenuated. Finally, whereas this study included a large sample of Caucasian and African American adolescents, it did not include participants from other racial backgrounds; thus, conclusions cannot necessarily be generalized to adolescents from other races.

Although most prior work has conceptualized childhood adversity as a risk factor for negative mental health outcomes among adolescents and adults (e.g., Turner & Lloyd, 1995), our study is among the first to demonstrate that moderate childhood stress attenuates depressive response to subsequent proximal stressors in humans. Further, this is the first study to document this attenuating effect among adolescents, which is a particularly important period within which to examine the effects of stressful events on the increase in depressive symptoms. These findings have important clinical and research implications, as our results indicate that moderate childhood stress may protect individuals from the depressogenic effects of proximal stressful events during adolescence. Specifically, these findings highlight the importance of implementing interventions that consider the unique and potentially beneficial role of exposure to stress, such as Stress Inoculation Training (SIT), which may help individuals cope with stressful events as well as “inoculate” individuals to future stressors (Meichenbaum, 1996). Additionally, our study represents an important direction for future studies on childhood adversity, stressful events, and depression during adolescence. It will be important for future research to simultaneously examine the effects of moderate childhood stress on psychological and physiological reactivity to proximal stress in order to more fully examine the buffering effects of moderate childhood stress on proximal stressors. Further, future research should evaluate the specific circumstances under which childhood adversity amplifies, sensitizes, or attenuates the effect of proximal stressors during adolescence.

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Figure 1. Effects of Proximal Stress on Depression Scores as a Function of Childhood Experiences

Note: CLES = Moderate early childhood stressful life events; values are indicated for 1 SD above and below the mean. Total proximal stressful life events are shown for 2 SD above and below the mean.

Table 1

Bivariate correlations between main study variables at each period

	T# CDI	T# Total Stress	CLES Severe	CLES Moderate	Mean (SD)
T1 CDI	-	.48***	.16*	.22**	5.19 (4.74)
T1 Stress		-	.05	.14	10.72 (5.90)
T2 CDI	-	.62***	.07	.06	5.60 (5.94)
T2 Stress		-	.13	.15	10.88 (6.49)
T3 CDI	-	.47***	.01	.03	4.54 (5.21)
T3 Stress		-	.01	.04	8.90 (5.77)
T4 CDI	-	.48***	.04	.16*	5.07 (5.76)
T4 Stress		-	.12	.07	8.84 (6.35)
CLES Severe			-	.30***	0.40 (0.65)
CLES Moderate			-	-	9.63 (4.33)

Note: Main study variables are correlated with each other at each period, thus the # indicates that each column is correlated only with that period; CDI = Child Depressive Symptoms, CLES = Early Child Stressors;

p < .001,

**

p < .01,

* p < .05

Table 2

Effect of Stress on Depression Scores for Childhood Moderate Stressful Events

Variable	Coefficient (SE)	Coefficient (SE)	Coefficient (SE)
Within-Subject Level			
Lagged Stress		.281 (.051)***	.494 (.104)***
Lagged Depressive Symptoms		.404 (.061)***	.434 (.058)***
Between-Subject Level			
Sex	-.422 (.550)	-.243 (.355)	-.289 (.347)
Race	.132 (.581)	.287 (.399)	.248 (.393)
Moderate Childhood Stress	.080 (.052)	.074 (.036)*	.067 (.035)
Severe Childhood Stress	.063 (.524)	-.075 (.348)	-.118 (.339)
Average Lagged Stress	.493 (.071)***	.317 (.044)***	.312 (.042)***
Cross-Level Interactions			
Moderate Childhood x Lagged Stress			-.018 (.009)*
Variance Components			
Within-Subject Level		1.87	1.20

Note: Lagged Stress was person-mean centered; Average Lagged Stress was grand-mean centered;

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