

**CHILDHOOD MALTREATMENT AND
STRESS SENSITIZATION IN DEPRESSION: MODERATION BY
AGE GROUP AND DEPRESSION HISTORY**

by

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Abstract

Major Depressive Disorder is a highly prevalent and recurrent psychological disorder, affecting approximately 12% of Canadians across their lifetime and 5% each year. Studies have shown that a history of childhood maltreatment increases risk for depression by conferring a vulnerability to the effects of stressful life events (i.e., stress sensitization). The goal of the current investigation was to examine whether the relation between childhood maltreatment and stress sensitization in depression is influenced by age group and depression history. This study also sought to investigate whether specific characteristics of the maltreatment experience differentially relate to stress sensitization. Two hundred and seven clinically depressed adolescents (i.e., 12 – 17 years; $n = 59$) and adults (i.e., 18 – 64 years; $n = 148$) participated in this study. Childhood accounts of maltreatment were assessed using the Childhood Experience of Care and Abuse Scale, and stressful life events experienced 3-months prior to depression onset were assessed using the Life Events and Difficulties Schedule. Results revealed that individuals with a history of severe maltreatment reported lower severity levels of stressful life events prior to depression onset than did those without such a history, but only among adolescents. Further, this relation was specific to independent stressors (i.e., those totally or nearly totally independent of the behaviour of the individual) and not dependent stressors (i.e., those at least partly due to the individual's behaviour), and was specific to emotional abuse. Results also suggested that it is the experience of severe maltreatment, rather than particular aspects of it, that sensitizes individuals to the effects of stress. In summary, this study provides support for the relation of childhood maltreatment to stress sensitization in adolescents. Maltreated adolescents may be especially vulnerable to the depressogenic effects of stress, perhaps because their maltreatment experience is more proximal to

depression onset. In contrast, other relevant processes (e.g., cognitive schema and neurobiological consolidation, chronic stress) may drive stress sensitization in adulthood; however, further research is required to investigate such mechanisms. Limitations and clinical implications are discussed.

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Chapter 1

Introduction

Major Depressive Disorder (MDD) is the most common psychological disorder, affecting 12.2% of Canadians across their lifetime and 4.8% each year (Patten et al., 2006). MDD affects males and females of all ages, regardless of individual characteristics such as ethnicity, geographic region, sexual orientation, and socioeconomic status. Currently, MDD is the second leading cause of disability worldwide among individuals 15 to 44 years of age and is projected to become the second leading cause among *all* age groups by 2020 (World Health Organization). The cardinal features of MDD include intense and prolonged sadness and/or a loss of interest or pleasure in almost all activities (American Psychiatric Association, 2000). In addition to these, individuals with depression experience a number of other symptoms that may include changes in weight and/or appetite, sleep disturbances, psychomotor changes, lack of energy, feelings of worthlessness or inappropriate guilt, difficulty concentrating, and recurrent thoughts of death (American Psychiatric Association, 2000). According to diagnostic guidelines of the Diagnostic and Statistical Manual of Mental Disorders – Fourth Edition (DSM-IV-TR, American Psychiatric Association, 2000), five of the nine possible symptoms must be present for a minimum duration of 2 weeks in order to meet criteria for MDD.

The nature and number of symptoms associated with MDD cause it to be a highly debilitating disorder. MDD is associated with significant impairment in occupational and/or educational functioning, interpersonal relationships, and physical health, and with an increased risk of suicide (Angst, Stassen, Clayton, & Angst 2002). These serious

consequences affect not only depressed individuals and their loved ones but also society as a whole. Each year in Canada, depression carries an economic burden of approximately \$14.4 billion, accounting for both direct treatment costs and indirect costs associated with reduced productivity and disability claims (Stephen & Joubert, 2001). One of the main factors that accounts for the immense personal and societal costs associated with MDD is the disorder's chronic nature. For example, in comparison to individuals with a less chronic course of depression, those with a more recurrent course have been found to experience a greater degree of chronic symptoms (Keller, Lavori, Rice, Coryell, & Hirschfeld, 1986), resistance to treatment (Kupfer, Frank, & Perel, 1989), and impairment in areas such as interpersonal relationships (Wittchen, Nelson, & Lachner, 1998), occupational and educational functioning (Wittchen et al., 1998), and cognitive functioning such as memory deficits (Basso & Bornstein, 1999). Unfortunately, the majority of individuals with MDD experience more than one major depressive episode (Mueller et al., 1999), and the risk of experiencing a recurrence increases with each successive episode (Solomon et al., 2000). Because of the chronic nature of depression and its associated disability, investigating etiological factors that cause and maintain MDD is an extremely important avenue of research.

Depression and Stress Sensitization

A substantial body of research suggests that stressful life events (SLEs) are strong predictors of the onset of MDD in adolescence (e.g., Lewinsohn, Joiner, & Rohde, 2001; Silberg, et al., 1999; Williamson et al., 1998) and adulthood (e.g., Daley, Hammen, & Rao, 2000; see Mazure, 1998). In particular, studies have demonstrated that depressed individuals are 3-6 times more likely than nondepressed individuals to report 'severe'

SLEs in the preceding three months (Goodyer, Kolvin, & Gatzanis, 1985). A severe life event is defined as a stressor that is associated with a high degree of psychological threat, such as one's spouse of 20 years unexpectedly files for divorce, or one is fired from one's job in the context of being the sole family earner. Approximately 80% of depressive episode onsets are preceded by a severe SLE (Mazure, 1998).

Although it has been well documented that severe SLEs are important contributors to the initial onset of depression (e.g., Brown & Harris, 1989; Hammen, 2005; Mazure, 1998), the role of stress in predicting subsequent depressive episodes changes over the lifetime course of the disorder (see Monroe & Harkness, 2005, for a review). Therefore, it is possible that the type of stressor that typically precedes a first episode of depression differs in nature from the type of stressor that generally predicts a recurrence. In order to help account for the changing role of stress in depression, Post (1992) proposed the stress sensitization (or "kindling") hypothesis. This hypothesis states that severe SLEs are important for triggering the first episode of depression; however, as the disorder progresses across recurrences, individuals become sensitized to the effects of SLEs *and* to their previous depressive episodes, such that more minor stressors (e.g., argument with a friend, failing an exam) become capable of precipitating recurrences (Monroe & Harkness, 2005; Post, 1992).

Two lines of evidence prompted Post (1992) to conceptualize the role of stress in depression in terms of sensitization. The first was the consistent clinical observation that severe SLEs were more likely to precede a first onset of depression than a recurrence (see Post, 1992, for a review). The second line of evidence came from neurobiological studies demonstrating that electrical or chemical stimulation could result in functional and

structural brain alterations responsible for subsequent reactivity to stress. Animal laboratory studies demonstrated clear evidence of electrophysiological kindling (e.g., Goddard, McIntyre, & Leech, 1969), with “kindling” referring to progressive changes in behaviour that result from repeated electrical stimulation. Goddard et al. found that with sufficient applications over time, less and less electrical brain stimulation, particularly to the limbic region, was required to elicit behavioural convulsions. Further, the authors noted that this long-lasting effect was not due to tissues damage, poison, edema, or gliosis, allowing them to conclude that the effect was due to electrical activation in the brain. The kindling effect has also been found to result from repeated administration of chemical stimulation (i.e., drugs). Research has shown that animals, such as rats, show increased behavioural responsiveness (e.g., specific motor movements) to the same or lower doses that follow repeated drug administration, as opposed to decreased responsiveness or “tolerance” to the same or higher doses (e.g., Laviola, Wood, Kuhn, Francis, & Spear, 1995; Post, Lockfeld, Squillace, & Contel, 1981). Electrical and chemical neurobiological stressors have been found to result in functional and structural alterations of brain regions that are responsible for subsequent sensitization or kindling. One such alteration, which has been identified by multiple research teams (e.g., Bullit, 1989; Ceccatelli, Villar, Goldstein, & Hokfelt, 1989; Nakajima, Post, Weiss, Pert, & Ketter, 1989), is the induction of gene transcription factors. Activation of gene transcription plays a role in altering neuromodulation and the functioning of various peptides and proteins, which ultimately results in kindling.

Using observations from kindling studies with animals as a framework, Post (1992) proposed the stress sensitization hypothesis to explain why the role of stress in

triggering episodes of depression appears to change as the disorder progresses. There are several important implications of the hypothesis that deserve individual mention (see Monroe & Harkness, 2005). The first is that there typically exists a greater association between more severe SLEs and the onset of a first depressive episode relative to a recurrent episode (Post, 1992). That is, a severe stressor is more likely to precede an individual's first episode of depression than, for example, his or her fifth or sixth episode. The second implication is that as depression progresses, the unique importance of severe SLEs in predicting a recurrent episode decreases, such that nonsevere SLEs become capable of evoking recurrences (Monroe & Harkness, 2005). This occurs because individuals become sensitized to the effects of stress and prior depressive episodes (Post, 1992), lowering their threshold for responding to stress. As a result, stressors that were previously incapable of triggering a depressive episode acquire that capacity as the disorder progresses. A third component of the stress sensitization hypothesis is that although nonsevere SLEs gain the capacity to evoke depressive episodes, severe SLEs are still fully capable of initiating a recurrence (Monroe & Harkness, 2005). That is, although the association between severe stressors and episode onset decreases across recurrences, severe SLEs are still capable of triggering recurrent episodes. This implies that the overall role of stress in triggering depression increases as the scope of SLEs that may precipitate a recurrence expands.

Typically, studies purporting to investigate stress sensitization have not examined the model in full (e.g., Daley et al., 2000; Kendler, Thornton, & Gardner, 2000). For example, some studies have investigated the relation of the onset of depressive episodes and severe SLEs but overlooked nonsevere SLEs, whereas others have included

individuals with recurrent depression but not those experiencing their first episode.

Although studies in this area tend to have such shortcomings, support for the abovementioned aspects of the stress sensitization hypothesis has been demonstrated.

First, results from prospective studies have shown a stronger association between more severe SLEs and the first episode of depression relative to a recurrent episode. For example, with a sample of 1,470 adolescents, Lewinsohn, Allen, Seeley, and Gotlib (1999) examined the effects of 10 major SLEs (i.e., events thought to be severe and salient for this population; e.g., moved out of the home for the first time) on the onset of first depressive episodes and recurrences. The researchers found that these major SLEs were more predictive of first episodes relative to recurrent episodes. In a population-based prospective study of older adults (i.e., 57 years and older) with subsyndromal and full syndromal depression, Ormel, Oldenhinkel, and Brilman (2001) investigated whether severe and nonsevere SLEs played a differential role in the 3-month period preceding a first episode or recurrent episode of depression. Stressful life events were assessed with the Life Events and Difficulties Schedule (LEDS; Brown & Harris, 1978, 1989), a gold-standard contextual interview and rating system that assesses recent life events and chronic difficulties. The authors found that severe events were more strongly associated with first episodes compared to recurrences.

Second, evidence demonstrating that nonsevere SLEs become more and more capable of evoking a recurrence as depression progresses has also been found. For example, the Ormel et al. (2001) study showed that nonsevere SLEs predicted the onset of depression, but only for individuals with a prior history of depression. Monroe et al. (2006) set out to replicate this finding within a sample of clinically depressed individuals.

Using the LEDS procedure, these authors found a predictive relationship between nonsevere SLEs and depression recurrence among depressed individuals receiving maintenance pharmacological treatment. Lenze, Cyranowski, Thompson, Anderson, and Frank (2008) examined the predictive ability of severe and nonsevere SLEs in a sample of recurrently depressed women who were first treated to remission with interpersonal therapy and then followed for up to 2 years or until recurrence. The authors found that nonsevere events significantly predicted recurrence, whereas severe events did not.

Lastly, it has been shown that although nonsevere SLEs gain the capacity to evoke recurrent episodes of depression, more severe SLEs are still fully capable of doing so. Morris, Ciesla, and Garber (2010) demonstrated this in a 6-year longitudinal study that followed adolescents from 6th to 12th grade, a time period extremely relevant for examining changes in the relation of stress and depression across first onsets and recurrences. The authors found that the predictive relation between SLEs and depression strengthened as a function of the number of previous episodes, regardless of the magnitude of the stressor. This suggests that as depression progresses and individuals become sensitized to the effects of stress, both severe *and* nonsevere events are capable of evoking a recurrence. Therefore, stress begins to play a greater role in depression as the scope of SLEs that may precipitate a recurrence expands.

Taken collectively, the abovementioned studies provide support for the stress sensitization hypothesis by demonstrating that the role of stress in depression changes across the course of the disorder. Support for the stress sensitization hypothesis is important because it helps account for the progressive nature of depression. More specifically, it helps in understanding why the risk of experiencing a recurrence increases

as a function of the number of prior episodes (Solomon et al., 2000). As depression recurs, individuals become increasingly vulnerable to the effects of stress *and* depressive episodes, via neurobiological and cognitive changes involved in sensitization (see Harkness & Lumley, 2007, for a review; Monroe & Harkness, 2005). Because of this increased sensitivity, individuals are more likely to experience a recurrence in the face of major *or* minor SLEs with each successive episode. The stress sensitization hypothesis also helps in understanding why periods of remission decrease as a function of the number of prior episodes (Solomon et al., 2000). Minor SLEs, which gain the capacity to trigger recurrences as sensitization occurs, are much more common in daily life than major SLEs. Therefore, minor SLEs become more likely to precede recurrences at a higher rate than less common, but equally capable, major SLEs (Monroe & Harkness, 2005). Resulting from this, as individuals become increasingly vulnerable to the effects of both severe and nonsevere stress, depressive episodes become more likely, and happen more quickly, over time (Monroe & Harkness, 2005).

Childhood Maltreatment and the Stress Sensitization Hypothesis of Depression

The stress sensitization hypothesis is extremely helpful in understanding how depression progresses once its course is underway. It does not in its original form, however, account for the initial onset of depression. Because of this, researchers have recently considered whether pre-existing vulnerability factors might increase risk for stress sensitization *even prior to the very first onset of depression*. One such variable is a history of childhood maltreatment, which is defined here as a history of physical and/or sexual and/or psychological or emotional abuse. Childhood maltreatment is strongly prospectively associated with the onset of MDD (e.g., Boney-McCoy & Finkelhor, 1996;

Brown, Cohen, Johnson, & Smailes, 1999; Fergusson, Horwood, & Lynskey, 1997; Lansford et al., 2002; Tebbutt, Swanston, Oates, & O'Toole 1997). Indeed, a review of this literature determined that a history of childhood maltreatment increases the risk of developing depression in adolescence or young adulthood 2-5-fold (Harkness & Lumley, 2007). Therefore, it appears that childhood maltreatment is a likely causal candidate in the initial development of depression (Kendler et al., 2000).

Several studies have also demonstrated that adverse childhood experiences such as abuse, neglect, and family disruption heighten sensitivity to SLEs. For example, Kendler, Kuhn, and Prescott (2004) found that following a severe SLE, women with a history of childhood sexual abuse were more likely to develop depression than women without such a history. This suggests that a prior history of trauma serves as a diathesis that heightens vulnerability to depression in the face of major acute stress. In a 2-year prospective study of young women, Hammen, Henry, and Daley (2000) examined the threshold of stress experienced prior to an episode of depression while controlling for depression history and chronic difficulties. The authors found that women with a history of one or more childhood adversities (e.g., family violence, parental alcoholism, death of a parent) were more likely to become depressed following lower levels of stress than those without such a history. This suggests that childhood adversity resulted in a predisposition of a heightened sensitivity such that even minor stressors were capable of triggering a depressive reaction.

Harkness, Bruce, and Lumley (2006) expanded on these studies and used a continuous gradient of stress severity to investigate the role of childhood maltreatment in sensitizing adolescents to the effects of independent and dependent SLEs. Independent

events are totally or nearly totally independent of the behaviour of the individual (e.g., grandmother suffers a stroke), whereas dependent events are at least partly due to the individual's behaviour (e.g., fired from job because of repeated tardiness). Consistent with the stress sensitization hypothesis, adolescents with a history of maltreatment had lower severity ratings of SLEs prior to the onset of a depressive episode than adolescents without such a history. Further, this effect was specific to *independent*, and not dependent, events (Harkness et al., 2006). The authors note that this finding is consistent with literature pointing to independent events as more central in the etiology of depression than dependent events (e.g., Shrout et al., 1989). Overall, findings from this study and the abovementioned studies are in agreement with the premise that adversity experienced early in life is predictive of greater sensitivity to the depressogenic effects of stress.

The Harkness et al. (2006) study further expanded on previous investigations of the relation of childhood maltreatment to stress sensitization by examining whether depression history (i.e., first episode vs. recurrent episode) moderated this association. That is, the authors were interested in whether the sensitizing effects of childhood maltreatment added to the sensitizing effects of a prior depressive episode. Interestingly, the authors found that the association between maltreatment history and lower severity levels of SLEs was specific to individuals on their first episode of depression. The authors suggested that a history of maltreatment may have sensitized adolescents to the effects of stress such that less severe stressors were required to trigger the very first episode of depression than were required for non-maltreated individuals. Of those on a recurrent episode, there was no difference in the level of stress severity reported between

maltreated and non-maltreated adolescents, suggesting that once the course of depression is underway, more proximal risk factors may be more important in determining the relation of stress to the onset of an episode.

These results suggest that a history of childhood maltreatment serves as a greater risk factor for stress sensitization among adolescents on their first depressive episode relative to those on a recurrent episode. It is important to note, however, that childhood maltreatment is still very proximal to depression onset in adolescence, regardless of whether the depression is a first onset or a recurrence. The situation is quite different in adulthood, such that even individuals' first onsets may be years away from the experience of maltreatment. Therefore, the primary goal of the current study was to examine the moderating effect of depression history and age on the relation of childhood maltreatment to stress sensitization in a sample of depressed individuals who range in age from adolescence to late adulthood.

Childhood Maltreatment and Stress Sensitization: Moderation by Depression History and Age Group

Evidence to suggest that the relation of childhood maltreatment to later stress sensitization may depend on age comes from studies examining the differential main effect relation of childhood maltreatment to early- versus late-onset depression. For example, Hill, Pickles, Rollinson, Davies, and Byatt (2004) examined differential relations of various forms of adversity to juvenile- and adult-onset depression, using the Childhood Experience of Care and Abuse Scale (Bifulco, Brown, & Harris, 1994), a gold standard retrospective measure of childhood abuse and neglect. The authors found that

juvenile-onset depression (i.e., before age 16) was associated with emotional abuse, physical abuse, domestic violence, and sexual abuse involving intercourse, whereas adult-onset (i.e., at or after age 16) depression was related only to sexual abuse without intercourse. Similarly, Jaffee et al. (2002) found that juvenile-onset depression was more strongly associated with a host of early risk factors (e.g., prenatal and neonatal problems, parental psychopathology and conflict, loss of parent, sexual abuse) than was adult-onset depression. Findings from these two studies demonstrate that early adversity generally has a stronger relation to adolescent-onset depression than adult-onset depression.

The studies above do not, however, address the mechanism that accounts for the decreasing role of childhood adversity on depression onset from childhood/adolescence to adulthood. I suggest in the current study that childhood maltreatment is not as likely to predict depression in adulthood as it is in adolescence because it is not as likely to affect the sensitization to stress. Perhaps adolescence is a developmental period during which individuals are especially vulnerable to the depressogenic effects of maltreatment, whereas some underlying mechanism of depression pathology is more pertinent to the onset of depression in adulthood.

Moderators of the Childhood Maltreatment – Stress Sensitization Relation:

Characteristics of Maltreatment

None of the studies reviewed above provide fine-grained detail regarding the characteristics of childhood maltreatment that most strongly drive stress sensitization. Knowledge of such characteristics will help advance our understanding of mechanisms underlying stress sensitization and complement research that has been conducted on the

fine-grained aspects of SLEs involved in stress sensitization (e.g., independence vs. dependence; Harkness et al., 2006). Therefore, the second major goal of the current study was to examine three maltreatment characteristics that might further moderate the relation of maltreatment to stress sensitization. First, I examined aspects of maltreatment timing, including the developmental period in which maltreatment occurred (i.e., childhood, adolescence, or both childhood and adolescence) and the duration of maltreatment. With respect to the first aspect, consideration of when maltreatment occurs in development is important because rates of maltreatment differ across developmental periods (Statistics Canada, 2009); therefore, it is not appropriate to aggregate maltreated children and adolescents into a single group. In addition, there is a relative paucity of studies focusing on adolescent maltreatment. For this reason, the consequences of adolescent maltreatment are not well understood.

Research on the role of maltreatment in predicting depression, in general, has found conflicting results regarding the developmental period during which maltreatment occurs. Several research teams have found that maltreatment occurring in adolescence, or continuously throughout childhood and adolescence, is more strongly depressogenic than is maltreatment that occurs only in childhood (e.g., Éthier, Lemelin, & Lacharité, 2004; Feiring, Taska, & Lewis, 1999; Peters, 1988; Tebbutt et al., 1997; Thornberry, Ireland, & Smith, 2001). Findings from these studies imply that adolescence is a key developmental period during which maltreatment exerts its pathological effects, and several suggestions have been put forth to help account for this. First, it has been noted that adverse conditions that cannot be avoided (e.g., parental abuse, neglect) may be particularly stressful for adolescents during a time when they are seeking greater independence and

autonomy, subsequently increasing their risk of internalizing and externalizing problems (Agnew, 1992). Further, some suggest that emotional and behaviour disturbances are more likely to follow adolescent maltreatment than childhood maltreatment because of additional developmental changes associated with this period, such brain maturation and the consolidation of cognitive abilities (Conte & Schuerman, 1987). More specifically, it has been suggested that the formation of negative thought patterns often associated with maltreatment (see Harkness & Lumley, 2007) are particularly likely to have a depressogenic effect following maltreatment occurring in adolescence because of corresponding increases in neurobiological and cognitive-emotional stability (Paus, 2005). Conversely, the effects of maltreatment occurring in childhood alone may not be as severe because the maturing brain and cognitive abilities are less developed and more pliable.

Nevertheless, other researchers have found that the *earlier* maltreatment occurs in a child's life, the more likely it is that the child will develop both internalizing and externalizing psychopathology (Bolger, Patterson, & Kupersmidt, 1998; Kaplow & Widom, 2007; Keiley, Howe, Dodge, Bates, & Pettit, 2001). Reasons that have been proposed for more detrimental effects of early abuse include that abuse occurring early in childhood is associated with a failure to reach important developmental milestones (e.g., formation of secure attachments, development of self-esteem, self-regulation) (Cicchetti, 1989; Moran & Eckenrode, 1992), and that younger individuals lack cognitive resources necessary for coping (Moran & Eckenrode, 1992) and are less able to actively avoid harmful situations (Carlson, Furby, Armstrong, & Schlaes, 1997).

To date, no studies have examined the differential impact of maltreatment occurring in childhood versus adolescence to the subsequent sensitization to stress. Examining this relation might help account for the discrepancies in the studies above, and may be useful in further identifying individuals who are particularly vulnerable to developing depression in the face of maltreatment. Despite the conflicting findings regarding the association of maltreatment timing to later psychopathology, I expect sensitization to stress to be more apparent among individuals who experienced maltreatment during adolescence. That is, I predict that maltreatment occurring in adolescence, or chronic maltreatment that persists into this developmental period, will have a stronger depressogenic effect such that it will be associated with a lower threshold of stress prior to an index episode of depression.

There is some ambiguity, however, regarding a classification scheme that categorizes maltreated individuals into distinct developmental periods based on when they experienced maltreatment. For example, should an individual who experienced a single incident of physical abuse in childhood and a single incident of sexual abuse in adolescence (i.e., technically categorized as persistent abuse) be classified in the same way as someone who experienced chronic psychological abuse from birth until age 18? In order to address concerns such as this, I will supplement the examination of developmental periods by looking at the *duration* of maltreatment. Kaplow and Widom (2007) used a similar approach by classifying the onset of maltreatment both categorically and continuously.

Although the examination of maltreatment duration differs conceptually from that of developmental periods, I expect a similar pattern of results to emerge. More

specifically, I predict that maltreatment of a longer duration will have a more depressogenic effect than less chronic maltreatment, such that it will be associated with a lower threshold of stress prior to depression onset. Thus, in essence, I expect to find evidence for a dose-response relation, such that greater stress sensitization will be apparent among individuals with a history of more chronic maltreatment relative to those with a less chronic history. The reasoning behind this is that a number of studies have found that chronic maltreatment has a stronger depressogenic effect than transitory maltreatment (e.g., Éthier et al., 2004; Thornberry et al., 2001). Further, maltreatment of a longer duration is more likely to persist into the adolescent years, increasing the likelihood of negative cognitive schemas developing and solidifying.

The second and third maltreatment characteristics that were examined in the present study included maltreatment comorbidity (i.e., defined here as the presence of two or more types of abuse) and severity, respectively. Dose-response relationships have been found for both of these maltreatment characteristics in relation to depression, in general (e.g., Edwards, Holden, Felitti, & Anda, 2003; see Higgins & McCabe, 2001, for a review). For example, studies have shown that the probability of MDD is highest among individuals reporting the greatest number of types of maltreatment (Bifulco, Moran, Baines, Bunn, & Stanford, 2002; Wise, Zierler, Krieger, & Harlow, 2001). Bifulco et al. (2002) found that recurrent MDD was experienced by 76% of individuals with a history of 5 – 6 types of maltreatment, 58% with a history of 3 – 4 types, 40% with a history of 1 – 2 types, and 21% with no history of maltreatment. Further, it has been demonstrated that rates of depression are greatest among individuals with a history of severe maltreatment. Bifulco et al. (2002) found that among individuals with a history of

childhood psychological abuse, MDD was experienced by 83% of those with a history of “marked” abuse, 70% of those who suffered “moderate” abuse, 55% of those who reported “mild” abuse, “ and 37% of those with no history of abuse. A similar association was found between risk of MDD and abuse severity in a sample of women with a history of physical and/or sexual abuse (Wise et al., 2001). Furthermore, in this sample, the risk of depression was highest among those with a history of both physical and sexual abuse, in comparison to individuals with a history of only one type of abuse.

Again, examining sensitization to stress in individuals with comorbid and/or more severe maltreatment may provide a mechanism for understanding the above relations. I expect to find evidence of these dose-response relations, such that greater stress sensitization will be apparent among individuals with a history of comorbid and/or more severe maltreatment relative to those without such a history. In general, examining fine-grained characteristics of maltreatment will provide a broader understanding of how maltreatment experienced early in life predisposes individuals to the effects of stressors experienced prior to the onset of a depressive episode.

Objectives and Hypotheses

The overarching purpose of the present study was to examine further moderators of the relation between childhood maltreatment and the sensitization to independent and dependent SLEs in depression. In addition to hypothesizing replication of past findings (e.g., Hammen et al., 2000; Harkness et al., 2006; i.e., individuals with a history of severe maltreatment would report lower severity levels of SLEs prior to depression onset than non-maltreated individuals), several predictions were made based on two primary goals.

The first goal was to examine the moderating effects of depression history and age group on the relation of childhood maltreatment to stress sensitization. The second goal was to examine characteristics of maltreatment that may be differentially related to stress sensitization. These characteristics included (a) maltreatment timing (i.e., developmental period and duration), (b) maltreatment comorbidity, and (c) maltreatment severity.

Goal 1: The moderating effect of depression history and age. I predicted a three-way interaction between a history of severe maltreatment, depression history (i.e., first episode vs. recurrence), and age group, such that maltreatment would be associated with lower severity levels of independent SLEs prior to onset, but only among adolescents (i.e., 12 – 17 years) on a first episode of depression. I expected no differences between maltreated and non-maltreated individuals with respect to severity levels of dependent SLEs. This prediction was based on Harkness et al.'s (2006) finding that the relation of childhood maltreatment to stress sensitization is specific to *independent*, and not dependent, events. Further, I expected no differences in stress sensitization would be evident between maltreatment and non-maltreatment among individuals on a recurrent episode of depression or among those in the adult group (i.e., 18 years and older). The motivation for these hypotheses stems from the finding that the childhood maltreatment – stress sensitization relation is specific to those on a first episode of depression relative to a recurrence (Harkness et al., 2006), and from research demonstrating a stronger association between a history of early adversity and adolescent-onset versus adult-onset depression (e.g., Hill et al., 2004).

Goal 2: Maltreatment characteristics and stress sensitization. Because I was interested in examining differential relations of maltreatment characteristics to stress

sensitization, all subsequent predictions concern only those individuals with a history of severe maltreatment. First, I hypothesized that individuals who experienced adolescent (i.e., 12 years and older) or persistent (i.e., maltreatment in both childhood and adolescence) maltreatment would report lower severity levels of SLEs prior to current episode onset than those with a history of childhood-only maltreatment (i.e., 0 – 11 years). Second, and also with respect to maltreatment timing, I hypothesized that greater sensitization prior to current episode onset would be evident for individuals who experienced more chronic maltreatment (i.e., longer duration) in comparison to those who experienced less chronic maltreatment. These predictions were based on the suggestion that maltreatment occurring in adolescence or chronically from childhood into adolescence is more depressogenic than that occurring in childhood only (e.g., Éthier et al., 2004; Thornberry et al., 2001). Third, I predicted that individuals with a history of comorbid maltreatment (i.e., 2 or more forms of maltreatment) would report lower severity levels of SLEs prior to current episode onset than individuals who experienced a single form of maltreatment. Fourth, I expected to find greater sensitization prior to current episode onset among individuals who experienced more severe maltreatment in comparison to those who experienced less severe maltreatment. These latter two hypotheses were motivated by research demonstrating dose-response relations between risk for depression and both maltreatment comorbidity and maltreatment severity (e.g., Bifulco et al., 2002; Wise et al., 2001). Further, as discussed above, I hypothesized that all of the above relations between the respective maltreatment characteristic and stress sensitization would be specific to independent, and not dependent, SLEs. Finally, I predicted that these relations would be further moderated by age group, such that they

would be apparent in the adolescents but not in the adults, and depression history, such that they would be evident only among those on a first episode.

Chapter 2

Method

Participants

Participants were 207 individuals who met DSM-IV criteria for a current non-chronic, non-bipolar mood disorder and who took part in one of three larger studies investigating the relation of stress to depression. The diagnoses held by participants included the following: (1) MDD ($n = 181$); (2) MDD in partial remission ($n = 10$); (3) Dysthymia ($n = 4$); (4) Adjustment disorder with depressed mood ($n = 4$); and (5) Depressive disorder not otherwise specified ($n = 8$)¹. Exclusion criteria were the presence of a psychotic disorder, bipolar disorder, developmental or conduct disorder (latter two diagnoses relevant to adolescents), substance dependence, and a medical condition that could cause depression. All participants had a minimum grade 7-education level and were fluent in reading English.

Participants from Study 1 were 75 adolescent and young adult males ($n = 18$) and females ($n = 57$), ranging in age from 12 to 21 ($M = 16.03$, $SD = 1.87$). Study 1 is an ongoing research project at Queen's University, which is examining how genetic factors interact with childhood maltreatment to heighten sensitivity to stress. These participants were recruited through mental health agencies and high schools in Kingston (see Harkness, Lumley, & Truss, 2008). Participants in Study 2 were 68 adult men ($n = 18$) and women ($n = 50$), ranging in age from 18 to 64 ($M = 33.61$, $SD = 13.50$). These

¹ Analyses described below were conducted on the full sample of participants ($n = 207$). Because a portion of participants did not meet full diagnostic criteria for MDD, analyses were also conducted on the subsample of participants with MDD ($n = 181$) while excluding those with other non-bipolar mood disorders. The same pattern of results were found; therefore, these analyses are not described.

participants were part of a study examining the role of childhood maltreatment and SLEs on treatment outcome in depression, and were recruited from advertisements and doctor referrals in the Greater Toronto Area (see Bulmash, Harkness, Stewart, & Bagby, 2009). Finally, participants in Study 3 were 64 adult women, ranging in age from 18 to 57 ($M = 35.98$, $SD = 10.42$). Study 3 was a research project conducted in Eugene, Oregon that examined the relation of childhood maltreatment and recent SLEs to depression. These women were recruited by way of advertisements (see Harkness & Monroe, 2006).

Two age groups were created that roughly correspond to lifespan stages of development: 1-adolescence (ages 12 – 17; $n = 44$ females, 15 males) and 2-adulthood (ages 18 – 64; $n = 127$ females, 21 males). Although age is a continuous variable, dichotomizing it into these two groups was thought to be a more appropriate way of detecting the hypothesized relation in the present study (i.e., moderation by age group of the relation of childhood maltreatment to stress sensitization). One reason that motivated this decision is that adolescence and adulthood are conceptually different stages of life, involving differences in social roles and contexts. For example, adolescents experience far more institutionally and culturally imposed structure and less opportunity for diversity in life paths than do adults (see Schulenberg, Sameroff, & Cicchetti, 2004). Subsequently, the transition from adolescence to adulthood, which typically involves a decrease in these restraints and an increase in self-direction, results in significant changes in independence, identity, affiliation, emotional regulation, achievement, and normal neurobiological changes (e.g., Cohen, Kasen, Chen, Hartmark, & Gordon, 2003; Schulenberg, Maggs, & O'Malley, 2003; Spear, 2000). Further, it has been suggested that although some of these developmental transitions are continuous in nature, others follow a discontinuous or

nonlinear trajectory across age (Schulenberg et al., 2003). A further motivation for dichotomizing age was speculation regarding differences in the experience of depression across developmental stages (e.g., triggers and symptom profiles, neurobiological correlates, treatment response, risk of converting to bipolar disorder; for reviews, see Kaufman, Martin, King, & Charney, 2001; Kovacs, 1996). This speculation originates from a large literature base, which has often used the classification of age into distinct stages (e.g., adolescence and adulthood) to identify these important differences. As such, a linear relation between age and SLEs was not predicted in the present study, deeming it more appropriate to examine age categorically.

Measures

Demographic questionnaire. This information sheet included questions asking for participants' gender, age, ethnicity, educational attainment, relationships/marital status, and occupational status. Occupational status was coded by two raters on an 8-point scale according to the Hollingshead Index of Social Position (Hollingshead, 1975). For the adolescents, an average of the parents' Hollingshead ratings was used in analyses. For adult participants in a committed relationship, an average Hollingshead rating was also used. Questions about current and past treatment (i.e., medication, therapy) were also included in the demographic questionnaire.

Diagnostic. Participants in Study 1 were administered the child and adolescent version of the Schedule for Affective Disorders and Schizophrenia (K-SADS; Kaufmann, Birmaher, Brent, Rao, & Ryan, 1996). Participants in Studies 2 and 3 were administered the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I/P; First, Spitzer,

Gibbon, & Williams, 2002). The K-SADS and the SCID-I/P are semi-structured interviews used to determine present and past DSM-IV diagnoses. Both the K-SADS (Ambrosini, 2000) and the SCID-I/P (Williams et al., 1992) have demonstrated excellent reliability and validity in their respective populations. In all studies, these interviews were administered by advanced graduate students in clinical psychology who were trained to “gold standard” reliability status (see Grove, Andreasen, McDonald-Scott, Keller, & Shapiro, 1981). In order to achieve this status, interviewers had to observe interviews being conducted by gold standard interviewers, conduct these interviews while being observed by gold standard interviewers, and match diagnoses on at least three consecutive interviews. Across the samples included in this study, high levels of agreement were found between raters with approximately 87% agreement for diagnosis and 73% agreement for depression recurrence status when 25% of cases were independently rated by separate raters.

Beck Depression Inventory – II (BDI-II; Beck, Steer, & Brown, 1996). This is a 21-item self-report measure that assesses the presence and severity of depression symptoms, based on the depression criteria of the DSM-IV. Participants responded on a scale from 0 to 3, with higher scores indicating more severe depressive symptoms. The BDI-II has demonstrated excellent reliability and validity in studies of depression with adolescents (e.g., Krefetz, Steer, Gulab, & Beck, 2002) and younger and older adults (e.g., Dozois, Dobson, & Ahnberg, 1998; Segal, Coolidge, Cahill, & O’Riley, 2008). Standardized internal consistency estimates were high across the samples included in this study (*Cronbach’s alphas* = 0.95).

Childhood Experience of Care and Abuse Scale (CECA; Bifulco et al., 1994).

This is a retrospective semi-structured interview that assesses the quality of parental care and abuse from as far back as a participant can remember to age 18. In particular, the CECA assesses the following variables: (a) antipathy – hostility or coldness toward the child by parents; (b) indifference – parental neglect of the child’s physical and/or emotional needs; (c) physical abuse – violence toward the child by parents; (d) sexual abuse – nonconsensual sexual contact by any perpetrator; and (e) psychological abuse – cruelty via verbal and nonverbal acts from someone in a position of power or responsibility over the child (see Bifulco et al., 1994; Moran, Bifulco, Ball, Jacobs, & Benaim, 2002; see Appendix A for sample questions). Antipathy, indifference, and psychological abuse are often referred to collectively as emotional abuse.

Advanced graduate students in clinical psychology interviewed participants. Interviewers were trained to not query the participants’ depression status, the effect of the experiences on the participants’ depression status, or the participants’ subjective responses to their experiences. The interviews were audio taped for subsequent rating. The aforementioned variables were rated on a 4-point severity scale (1 = *marked*, 2 = *moderate*, 3 = *some*, 4 = *little/none*) by raters unaware of the participants’ depression status or life event information. The raters used the CECA manual, which contains hundreds of examples and rating rules, to assign a severity rating to each variable. High levels of agreement between raters were found with kappa coefficients ranging from .86 – 1.00 when 20% of the CECA tapes were independently rated by separate raters. Across the studies, interviewers and raters received extensive training and ongoing supervision in the Bedford College procedures for rating childhood adversity.

Because the distributions of the CECA variables were skewed with relatively few participants reporting *moderate* or *marked* maltreatment, each variable was dichotomized into ‘severe’ (1-marked or 2-moderate) or ‘nonsevere’ (3-some or 4-little/none) levels. The percentages of participants who reported severe antipathy, indifference, physical abuse, sexual abuse, or psychological abuse were 27.1% ($n = 56$), 20.3% ($n = 42$), 26.1% ($n = 54$), 22.7% ($n = 47$), and 9.7% ($n = 20$), respectively. Then, a childhood maltreatment composite variable, denoting the presence versus absence of severe maltreatment (i.e., severe antipathy and/or indifference and/or physical abuse and/or sexual abuse and/or psychological abuse) was created. One-hundred and three (49.8%) participants were classified as having a history of severe maltreatment.

Several variables were further computed to examine the specific features of childhood maltreatment that are associated with stress sensitization. First, two variables were created based on the timing of maltreatment: developmental period and duration of maltreatment. Only participants with a history of physical, sexual, or psychological abuse were classified in this way. Participants with a history of only severe antipathy and/or indifference (i.e., neglect) were not included because antipathy and indifference are forms of maltreatment that, by their nature, almost always persist throughout childhood and adolescence. Therefore, these variables were based on 91 participants. Developmental period was classified as: (1) childhood (maltreatment did not continue past 11 years; $n = 20$); (2) adolescent (maltreatment had its onset at age 12 or older; $n = 15$); and (3) persistent (maltreatment had its onset prior to age 11 and persisted into adolescence; $n = 56$) (e.g., Bolger et al., 1998; Thornberry et al., 2001). Duration of maltreatment was

defined as the total time in years that the abuse (i.e., physical and/or sexual and/or psychological) lasted.

Second, all 103 participants with a history of severe maltreatment were categorized as having comorbid maltreatment ($n = 60$) if they reported at least two different types of maltreatment, or non-comorbid maltreatment ($n = 43$) if they only experienced one type of maltreatment.

Third, a continuous variable was created to assess total maltreatment severity for participants with a history of severe maltreatment ($n = 103$). In order to create this variable, severity scores for each type of maltreatment (i.e., antipathy, indifference, physical abuse, sexual abuse, psychological abuse) were first computed and reverse coded. Severity scores for antipathy and indifference were weighted by the duration (in years) of these maltreatment experiences, and scores were summed across all perpetrators. Severity scores for physical, sexual, and psychological abuse were weighted by both the duration (in years) and the frequency of abuse, and scores were summed across all perpetrators. Possible scores on this variable ranged from 2 to 20, with higher scores denoting more severe maltreatment.

Life Events and Difficulties Schedule (LEDS-II; Bifulco et al., 1989; adolescent version, Frank, Matty, & Anderson, 1997). This is a semi-structured, contextual interview and rating system that assesses recent SLEs in ten domains: education, occupation, housing, finances, role changes, legal, health, romantic relationships, other relationships, and deaths (see Appendix B for sample questions). Stress sensitization is a theory of depression onset; therefore, the focus in this study was

on life events that occurred in the 3 months prior to the onset of the depressive episode (Brown & Harris, 1989).

Advanced graduate students in clinical psychology interviewed participants. Interviewers were trained to obtain contextual or objective information regarding the event. Further, they were trained to not query about participants' subjective reactions to a stressor or the effect of the stressors on participants' depression. The interviews were audio taped for subsequent rating. A research assistant listened to the tapes and prepared brief vignettes to summarize each event, omitting any information regarding the participant's depression status and emotional response to the stressors. These vignettes were presented to a team of 2 – 4 raters, who then rated the severity of each event, using the adolescent and adult LEDES manuals to base their ratings. Together, the LEDES manuals include over 5,000 examples as well as explicit rules and criteria for rating life events in order to provide anchoring and standardization of the ratings. Studies using the LEDES have shown higher reliability and validity in the prediction of MDD over checklist measures of stress (e.g., Brown & Harris, 1989; McQuaid, Monroe, Roberts, Kupfer, & Frank, 2000). Across the samples included in this study, pairwise comparisons among raters on the severity ratings of events ranged from $\kappa = .76$ to $\kappa = .94$ ($M = .90$).

Life events were rated for their level contextual severity on a 4-point scale (1 = *marked*, 2 = *moderate*, 3 = *some*, 4 = *little/none*). Severity is based on factors such as the degree of unpleasantness, threat or danger about the future, and serious practical inconvenience related to the event. This severity rating is determined independent of the respondent's reported feelings about the event. Each rater provided his or her own severity rating, justifying the rating by appealing to a specific example in the LEDES

manuals. Discrepancies among raters were discussed and a consensus rating was achieved, which was used in all analyses.

Life events were also rated on independence/dependence. As previously noted, independent events are those totally or nearly totally independent of the action of the individual (e.g., grandmother develops cancer, mother loses job). Dependent events are those at least partly due to the action of the individual (e.g., fired from job because of repeated tardiness). The raters made decisions about independence by consensus using the LEDS manuals.

Severity scores for SLEs were reverse coded, such that higher scores represented greater severity. Next, the severity scores were summed over the number of events in the 3-month period prior to onset separately for independent and dependent events.

Procedure

Ethical approval was obtained for each Study from the Research Ethics Board of the respective institution. Written informed consent was provided by all participants and additionally by a parent or guardian for participants under 18 years of age. In Study 1, participants engaged in two 2-hour assessments separated by one week. During session 1, adolescents were administered the K-SADS interview and questionnaires. During session 2, they received the LEDS and the CECA interviews. In Study 2, women participated in one 3-hour assessment in which the SCID-I/P was administered first, followed by questionnaires and the LEDS and CECA interviews. Participants in Study 3 were part of a treatment trial; therefore, the timing of interview administration differed in this study. Prior to receiving treatment, participants completed the SCID-I/P and questionnaires.

Following 16 weeks of treatment (i.e., either cognitive-behavioural therapy, interpersonal therapy, or antidepressant medication), participants were administered the LEDS and the CECA interviews, with the LEDS interview covering the period from six months prior to onset of the index episode through the treatment trial (Bulmash et al., 2009). For each study, it was important for the diagnostic interview to be administered prior to the life events and childhood history interviews to prevent bias in the diagnosis of depression.

Data Analysis

All analyses were conducted using the Statistical Program for the Social Sciences (SPSS) Software, Version 18. A small number of participants were missing data for several demographic variables. Rather than exclude these individuals, these data were accounted for by a mean replacement procedure that was stratified by each participants' maltreatment history, age group, and depression history. Preliminary univariate analyses were first conducted to examine differences across study sites with respect to relevant demographic, clinical, SLE, and childhood maltreatment variables. Demographic variables included gender, age, ethnicity, and occupational status. Clinical variables included age of first depressive episode, total number of previous episodes, depression severity, presence versus absence of comorbid diagnoses, and presence versus absence of current treatment. SLE variables included the cumulative severity rating of independent and dependent events, respectively, in the 3-month period preceding the onset of depression. Finally, childhood maltreatment variables included severe maltreatment (i.e., presence vs. absence), the developmental period during which maltreatment occurred (i.e., childhood vs. adolescence vs. persistent), duration of maltreatment, comorbid maltreatment (i.e., presence vs. absence), and maltreatment severity. Preliminary

univariate analyses were also conducted to examine the relation of demographic and clinical covariates to both the SLE variables and the childhood maltreatment variables. Demographic and clinical variables found to be significantly related to the primary study variables of interest were included as covariates in the main models below.

The primary study goals were assessed using Analysis of Covariance models. The dependent variables in all of the ANCOVA models were the cumulative severity rating of independent or dependent events, respectively, in the 3 months preceding the onset of depression. Covariates included any demographic and clinical variables that differed significantly across the SLE and maltreatment variables. The ANCOVA procedure used Type III sum of squares to account for intercorrelations among the independent variables and unequal cell sizes. Partial η^2 is reported as the index of effect size. Guidelines by Ferguson (2009) were used to help interpret effect sizes, such that .04, .25, and .64 corresponded to small, medium, and large effects, respectively. Significant interactions among the childhood maltreatment, age group, and depression history factors were followed up by pairwise comparisons between means, using the Bonferroni correction to adjust for the number of comparisons. Estimated marginal means are reported in all figures.

Chapter 3

Results

Preliminary Univariate Analyses

Site differences. Site differences were examined and reported strictly for descriptive purposes. Tables 1 and 2 present descriptive data separately for each site. Study sites did not differ in terms of ethnicity or treatment status. As can be expected given the makeup of the samples, age and gender differed significantly across studies (p 's $< .001$). Occupational status differed across sites, $F(2, 204) = 16.99, p < .001$, partial $\eta^2 = .14$, with Study 1 reporting significantly higher occupational status (in this case, of their parents) than either Study 2 or 3. Further, and not surprisingly, those in Study 3 were more likely to be experiencing a recurrent episode of depression, $\chi^2(2, N = 207) = 34.51, p < .001$, and reported significantly more episodes of depression, $F(2, 204) = 31.15, p < .001$, partial $\eta^2 = .23$, than those in Studies 1 and 2. Study 2 had significantly higher BDI scores than Study 1, $F(2, 204) = 3.41, p = .035$, partial $\eta^2 = .03$. Finally, participants in Study 1 ($n = 33/75, 44.0\%$) and Study 3 ($n = 28/64, 43.8\%$) were more likely to have a comorbid diagnoses than participants in Study 2 ($n = 9/68, 13.2\%$; $\chi^2[2 N = 207] = 19.17, p < .001$).

Cumulative severity ratings of independent SLEs did not differ significantly across study sites. Cumulative severity ratings of dependent SLEs were differentially distributed across studies, $F(2, 204) = 6.86, p = .001$, partial $\eta^2 = .06$, such that those in Study 3 reported a greater severity of dependent events than those in both Studies 1 and 2.

Study 3 had a significantly higher proportion of individuals with a history of severe maltreatment, $\chi^2(2, N = 207) = 16.97, p < .001$, in comparison to Studies 1 and 2. Of those with a history of severe maltreatment, Studies 2 and 3 had the highest proportion of individuals with a history of persistent abuse, whereas Study 1 had the highest proportion of individuals with a history of childhood abuse, $\chi^2(4, N = 91) = 11.01, p = .026$. Duration of maltreatment differed across studies, $F(2, 88) = 9.49, p < .001$, partial $\eta^2 = .18$, with individuals in Study 3 reporting more years of abuse than those in Study 1. The presence of maltreatment comorbidity was not differentially distributed across studies. Maltreatment severity differed across studies, $F(2, 100) = 7.08, p = .001$, partial $\eta^2 = .12$, with individuals in Study 3 reporting more severe maltreatment than those in Studies 1 and 2.

Table 1

Descriptive Statistics of Demographic and Clinical Variables Stratified by Study Site

	Study Site		
	Study 1 (<i>n</i> = 75)	Study 2 (<i>n</i> = 68)	Study 3 (<i>n</i> = 64)
Age (<i>M/SD</i>)	16.03/1.87	33.61/13.50	35.98/10.42
Sex (<i>n/%</i>)			
Male	18/24.0	18/26.5	0/0.0
Female	57/76.0	50/73.5	64/100
Ethnicity (<i>n/%</i>)			
White	70/93.3	24/35.3	59/92.2
Other	5/6.7	6/8.8	5/7.8
Hollingshead index (<i>M/SD</i>)	3.59/1.81	5.41/1.77	4.94/2.27
Age at first onset (<i>M/SD</i>)	13.72/2.93	26.29/10.33	
Number of episodes (<i>M/SD</i>)	1.83/1.47	2.97/4.57	7.45/6.09
Depression history (<i>n/%</i>)			
First onset	38/50.7	41/60.3	8/12.5
Recurrence	37/49.3	27/39.7	56/87.5
BDI score (<i>M/SD</i>)	25.86/12.34	30.28/8.95	28.44/8.44
Comorbidities (<i>n/%</i>)			
Panic disorder	4/5.3	1/1.5	17/26.6
Simple phobia	6/8.0	3/4.4	3/4.7
Social phobia	11/14.7	3/4.4	12/18.8
OCD	2/2.7	0/0.0	0/0.0
PTSD	6/8.0	0/0.0	13/20.3
GAD	9/12.0	1/1.5	1/1.6
Substance abuse	5/6.7	2/2.9	0/0.0
Treatment status (<i>n/%</i>)			
Yes	46/61.3	38/55.9	33/51.6
No	28/37.3	30/44.1	24/37.5

Note. BDI = Beck Depression Inventory; OCD = Obsessive Compulsive Disorder; PTSD = Post Traumatic Stress Disorder; GAD = Generalized Anxiety Disorder.

Table 2

Descriptive Statistics of Stress and Childhood Maltreatment Variables Stratified by Study Site

	Study Site		
	Study 1 (<i>n</i> = 75)	Study 2 (<i>n</i> = 68)	Study 3 (<i>n</i> = 64)
Cumulative severity of independent events (<i>M/SD</i>)	1.29/1.81	1.01/2.37	1.84/2.34
Cumulative severity of dependent events (<i>M/SD</i>)	1.57/2.01	1.29/2.00	2.70/2.88
No. of maltreatment types (<i>n</i> /%)			
No. participants with 0	48/64.0	37/54.4	19/29.7
No. participants with 1	12/16.0	13/19.1	18/28.1
No. participants with 2	12/16.0	5/7.4	6/9.4
No. participants with 3+	3/4.0	13/19.1	21/32.8
Developmental period (<i>n</i> /%)			
Childhood	9/33.3	6/19.4	5/11.1
Adolescence	6/22.2	4/12.9	5/11.1
Persistent	8/29.6	16/51.6	32/71.2
Duration of maltreatment (<i>M/SD</i>)	4.68/4.27	7.70/5.12	10.55/5.63
Maltreatment severity (<i>M/SD</i>)	7.27/2.19	8.03/2.61	9.84/3.60

Demographic and clinical covariates. Univariate analyses were next conducted to identify potential demographic and clinical covariates. I did not correct alpha for multiple tests because the purpose of conducting these tests is to be liberal in the search for possible confounding variables.

Life events. Females reported significantly higher severity levels of independent SLEs prior to depression onset, ($M = 1.50$, $SD = 2.32$), than did males ($M = 0.92$, $SD = 1.34$; $t[205] = 2.04$, $p = .045$). Participants with a comorbid disorder also reported significantly higher severity levels of independent SLEs prior to depression onset ($M = 1.81$, $SD = 2.43$) than those without a comorbid disorder ($M = 1.18$, $SD = 2.02$; $t[205] = 1.98$, $p = .049$). No other demographic or clinical covariates emerged as significantly related to life events.

Child maltreatment. Descriptive characteristics by maltreatment group (i.e., severe maltreatment: absent vs. present) are presented in Table 3. Compared to those without a history of severe maltreatment, those with a history of severe maltreatment were more likely to be female, $\chi^2(2, N = 207) = 10.69$, $p = .001$, and reported a significantly lower occupational status, $t(205) = 2.06$, $p = .041$, more severe depressive symptoms, $t(205) = 4.70$, $p < .001$, and a significantly greater number of depressive episodes, $t(147.23) = 3.93$, $p < .001$. No other demographic or clinical covariates emerged as significantly related to the presence versus absence of severe maltreatment.

Table 3

Descriptive Statistics of Demographic and Clinical Variables Stratified by Maltreatment Group

	Severe Maltreatment	
	Absent (<i>n</i> = 104)	Present (<i>n</i> = 103)
Age (<i>M/SD</i>)	26.23/13.48	29.73/12.88
Sex (<i>n/%</i>)		
Male	27/26.0	9/8.7
Female	77/74.0	94/91.3
Ethnicity (<i>n/%</i>)		
White	77/74.0	76/73.8
Other	7/6.7	9/8.7
Hollingshead index (<i>M/SD</i>)	4.31/2.01	4.90/2.15
Age at first onset (<i>M/SD</i>)	19.81/10.25	19.52/8.97
Number of episodes (<i>M/SD</i>)	2.63/2.97	5.26/6.12
Depression history (<i>n/%</i>)		
First onset	52/50.0	35/34.0
Recurrence	52/50.0	68/66.0
BDI score (<i>M/SD</i>)	24.92/9.12	31.32/10.45
Comorbidities (<i>n/%</i>)		
Panic disorder	7/6.7	15/14.6
Simple phobia	4/3.8	8/7.8
Social phobia	7/6.7	19/18.4
OCD	0/0.0	2/1.9
PTSD	7/6.7	12/11.7
GAD	10/9.6	1/1.0
Substance abuse	4/3.8	4/3.9
Treatment status (<i>n/%</i>)		
Yes	53/51.0	64/62.1
No	45/43.3	37/35.9

Note. BDI = Beck Depression Inventory; OCD = Obsessive Compulsive Disorder; PTSD = Post Traumatic Stress Disorder; GAD = Generalized Anxiety Disorder.

Among those with severe maltreatment, those with persistent abuse reported a significantly lower occupational status than those with adolescent abuse, ($M = 5.33$, $SD = 2.25$ vs. $M = 3.67$, $SD = 1.72$; $F[2, 88] = 3.78$, $p = .027$, partial $\eta^2 = .08$). Greater maltreatment duration was significantly related to lower occupational status, $r(N = 91) = .31$, $p = .003$, and more depressive episodes, $r(N = 91) = .32$, $p = .002$. Finally, more depressive episodes were related to both maltreatment comorbidity (present: $M = 6.23$ $SD = 6.96$; absent: $M = 3.91$, $SD = 4.42$; $t[99.71] = 2.07$, $p = .041$) and greater maltreatment severity, $r(N = 103) = .33$, $p = .001$.

Goal 1: The Relation of Childhood Maltreatment, Depression History, and Age Group to Stress Sensitization

The relation of severe maltreatment, depression history, and age group to the severity of SLEs prior to depression onset was examined in two 2 X 2 X 2 factorial ANCOVAs. The dependent variables in the two models were severity levels of independent and dependent events, respectively. Gender, occupational status, and depression severity were entered as covariates. Comorbidity was also entered as a covariate in analyses involving independent events.

Independent events. Over and above the effects of the covariates, the interaction between age group and severe maltreatment was significant, $F(1, 195) = 9.17$, $p = .003$, partial $\eta^2 = .05$. This interaction was followed up with pairwise comparisons using a critical alpha value of .0125. As displayed in Figure 1, and consistent with hypotheses, in the adolescent age group, those with a history of severe maltreatment reported significantly lower severity levels of independent SLEs than did those without such a

history ($M = 0.67$ vs. 2.02 , $SE = 0.48$ vs. 0.38 ; $F[1, 199] = 7.19$, $p = .008$, partial $\eta^2 = .04$). In contrast, in the adult age group, no differences emerged between those with ($M = 1.61$, $SE = 0.28$) versus those without ($M = 1.06$, $SE = 0.27$) a history of maltreatment, $F(1, 199) = 1.70$, $p = .194$, partial $\eta^2 = .01$. See Appendix C for ANCOVA test results, excluding main effects of covariates.

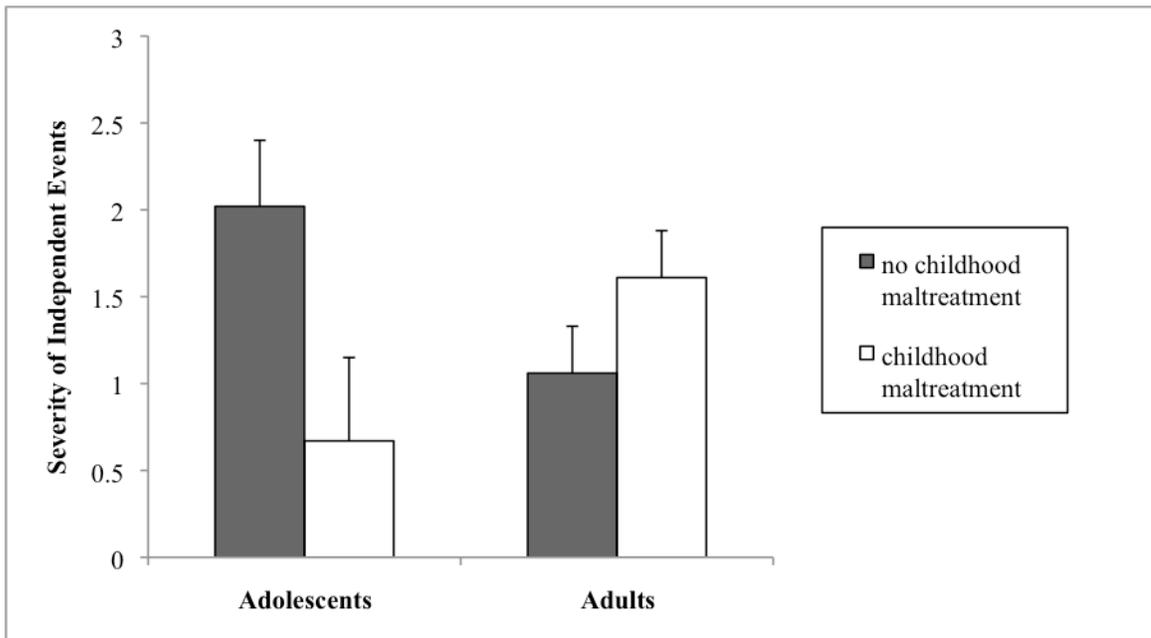


Figure 1. Relation of age group and childhood maltreatment to the cumulative severity level of independent stressful life events.

As a follow-up to the above results, I sought to determine whether the interaction between age group and severe maltreatment was specific to a particular type of maltreatment. The motivation for this was that research has demonstrated that particular types of maltreatment convey a greater risk for depression than others (e.g., Gibb, Chelminski, & Zimmerman, 2007; Hankin, 2005). Three 2 (presence vs. absence of physical, sexual, or emotional abuse, respectively) X 2 (age group) factorial ANCOVAs were conducted. Emotional abuse is a composite denoting the presence versus absence of

antipathy and/or indifference and/or psychological abuse. No significant main effects or interactions emerged for the model with physical abuse. For the model with sexual abuse, the interaction between age group and maltreatment approached significance, $F(1, 199) = 3.49, p = .063, \text{partial } \eta^2 = .02$. Although not significant, this was followed up with pairwise comparisons; however, the follow-up tests were nonsignificant with negligible effect sizes. Finally, in the model with emotional abuse, the age group by maltreatment interaction was significant, $F(1, 199) = 11.88, p = .001, \text{partial } \eta^2 = .06$ (see Figure 2). Consistent with the primary analysis above, those with a history of severe emotional abuse reported lower severity levels of independent SLEs than did those without such a history, but only among adolescents ($M = 0.27$ vs. $1.94, SE = 0.58$ vs. $0.33; F[1, 199] = 6.10, p = .014, \text{partial } \eta^2 = .03$) and not for adults ($M = 1.90$ vs. $0.99, SE = 0.29$ vs. $0.22; F[1, 199] = 5.09, p = .024, \text{partial } \eta^2 = .03$). Further, among those with a history of severe emotional abuse, adolescents reported significantly lower severity levels of independent SLEs than did adults, ($M = 0.27$ vs. $1.90, SE = 0.58$ vs. $0.29; F[1, 199] = 6.54, p = .011, \text{partial } \eta^2 = .03$). For those without a history of severe emotional abuse, there was no significant difference in severity levels of independent SLEs for adolescents and adults ($M = 1.94$ vs. $0.99, SE = 0.33$ vs. $0.22; F[1, 199] = 5.52, p = .020, \text{partial } \eta^2 = .03$). See Appendix D for ANCOVA test results by maltreatment type, excluding main effects of covariates.

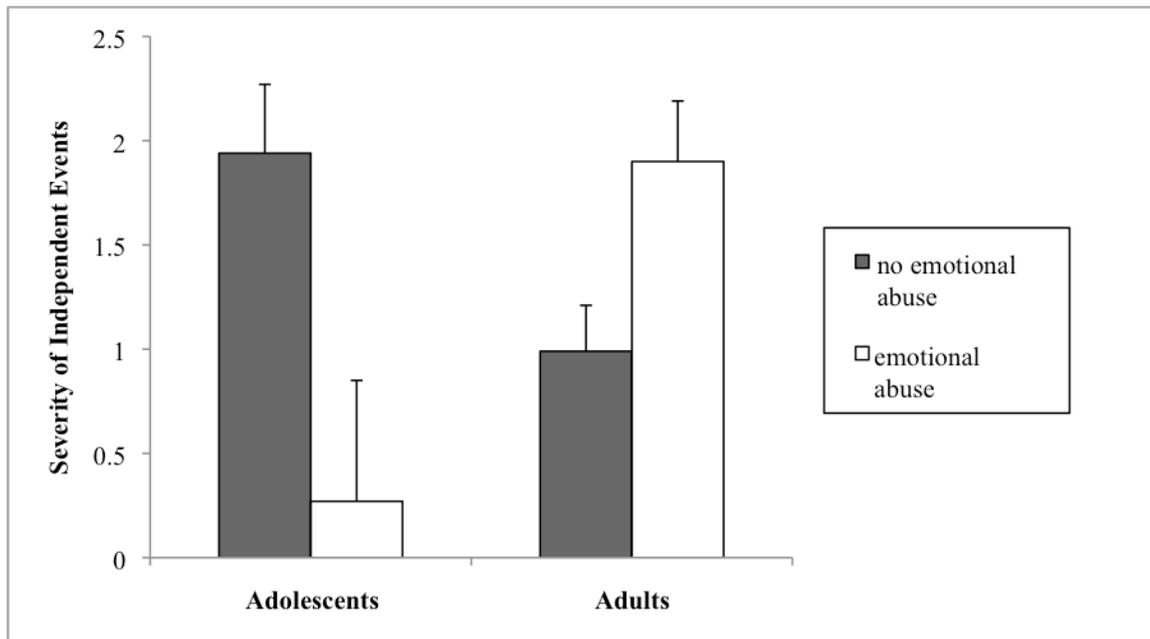


Figure 2. Relation of age group and emotional abuse to the cumulative severity level of independent stressful life events.

Dependent events. Over and above the effects of the covariates, the main effect of severe maltreatment approached significance, $F(1, 196) = 3.20, p = .075$, partial $\eta^2 = .02$, such that those with a history of maltreatment reported *higher* levels of dependent events than did those without such a history ($M_s = 2.02$ and 1.29 , respectively, $SE_s = 0.30$ and 0.26 , respectively). Further, this main effect was specific to sexual abuse (present: $M = 2.55, SE = 0.36$; absent: $M = 1.62, SE = 0.19$; $F[1, 202] = 5.18, p = .024$, partial $\eta^2 = .03$) and not physical (present: $M = 2.15, SE = 0.34$; absent: $M = 1.72, SE = 0.20$; $F[1, 202] = 0.88, p = .350$, partial $\eta^2 = .00$) or emotional abuse (present: $M = 2.22, SE = 0.30$; absent: $M = 1.64, SE = 0.21$; $F[1, 202] = 2.43, p = .121$, partial $\eta^2 = .01$). No other main effects or interactions emerged as significant. See Appendix C for ANCOVA test results, excluding main effects of covariates.

Goal 2: The Relation of Maltreatment Characteristics and Age Group to Stress

Sensitization

The relations of the maltreatment characteristics and age group to the severity of SLEs prior to depression onset were examined with a series of ANCOVAs within the subsample of participants with a history of severe maltreatment ($n = 103$). The dependent variables in all models were severity level of independent and dependent events, respectively. Depression history was not included as a factor in these analyses because it failed to interact with other variables and did not emerge as a significant main effect in the above analyses. For analyses involving independent SLEs, gender and comorbidity were entered as covariates. For all models, additional variables relevant to the maltreatment characteristic of interest were also entered. More specifically, occupational status was entered in analyses involving both the developmental period of maltreatment and maltreatment duration. Further, total number of episodes was entered in analyses involving maltreatment duration, maltreatment comorbidity, and maltreatment severity. See Appendix E for ANCOVA test results by maltreatment characteristics, excluding main effects of covariates.

Maltreatment timing. The relation of the developmental period of abuse to SLEs was examined with two 3 (developmental period: childhood only, adolescence only, persistent) X 2 (age group) factorial ANCOVAs. No significant main effects or interactions emerged for either independent or dependent SLEs. The relation of maltreatment duration to SLEs was examined with two one-way ANCOVAs, modeling maltreatment duration as a continuous factor in the interaction with age group. Again, no

significant main effects or interactions were found for either independent or dependent SLEs.

Maltreatment comorbidity. The relation of maltreatment comorbidity to SLEs was examined with two 2 (comorbid maltreatment) X 2 (age group) factorial ANCOVAs. The main effect of age group was significant, $F(1, 98) = 4.28, p = .041$, partial $\eta^2 = .04$, such that adults reported higher severity levels of dependent SLEs prior to depression onset than did adolescents ($M_s = 2.50$ and 1.14 , respectively, $SE_s = 0.30$ and 0.58 , respectively). No other main effects or interactions emerged as significant for either independent or dependent SLEs.

Maltreatment severity. The relation of maltreatment severity to SLEs was examined with two one-way ANCOVAs, modeling maltreatment severity as a continuous factor in the interaction with age group. No significant main effects or interactions were found for independent or dependent SLEs.

Chapter 4

Discussion

The overarching purpose of the current investigation was to examine whether the relation of childhood maltreatment to stress sensitization in depression is moderated by age group and depression history. Although research has clearly demonstrated that the experience of maltreatment early in life confers a vulnerability to depression through stress sensitization (e.g., Hammen et al., 2000; Harkness et al., 2006), questions regarding the existence of this vulnerability across development and depression recurrences are just starting to be addressed. Further, questions still remain regarding fine-grained characteristics of maltreatment that may differentially influence this relation. In investigating these relations, this study had two primary goals. The first goal was to examine the moderating effects of age group and depression history on the childhood maltreatment – stress sensitization relation. The second goal was to investigate three characteristics of maltreatment that may be differentially related to stress sensitization, including maltreatment timing (i.e., developmental period and duration), maltreatment comorbidity, and maltreatment severity. Results pertaining to each of these two goals are summarized and discussed below.

Stress Sensitization, Age Group, and Depression History

Support for the stress sensitization hypothesis was found in the present study, such that individuals with a history of severe childhood maltreatment had significantly lower severity levels of SLEs prior to depression onset than did those without such a history. Further, consistent with hypotheses, this relation was found in adolescents and not adults

and was specific to independent, and not dependent, SLEs. In addition, this interaction was specific to emotional abuse and not physical or sexual abuse.

These results are consistent with Harkness et al.'s (2006) findings and suggest that childhood maltreatment may have resulted in a depressogenic vulnerability to stress for adolescents. As such, adolescents with a history of maltreatment may require lower severity levels of independent SLEs to trigger depression onset than those with no such history. These results are also in line with longitudinal studies linking a variety of negative childhood experiences (e.g., sexual abuse, family violence, parental divorce, parental psychopathology) to stress sensitization (e.g., Hammen et al., 2000; Kendler et al., 2004). Further, this relation provides support for Hankin and Abramson's (2001) cognitive vulnerability-transactional stress model of depression, which posits that distal risk factors for depression, such as a history of childhood adversity, contribute to the development of depression through more proximal risk factors (e.g., minor SLEs). Finally, and most importantly, findings from the present study extend previous research by demonstrating that the relation between childhood maltreatment and stress sensitization differs across developmental stages. This is important as it suggests that different mechanisms may be involved in triggering depression in adolescence versus adulthood. Following a discussion pertaining to possible processes underlying the childhood maltreatment – stress sensitization relation in adolescence, age group differences in potential depressogenic responses to stress will be discussed.

This study did not examine the precise mechanisms of stress sensitization in depressed adolescents. Therefore, in order to help account for the findings, I will turn to a psychobiological mechanism proposed by Harkness and Lumley (2007) to underlie the

translation of childhood maltreatment to a depressogenic vulnerability to stress, with ideas originating from several research teams (e.g., Gold, Goodwin, & Chrousos, 1988; Plotsky, Owens, & Nemeroff, 1998; Segal, Williams, Teasdale, & Gemar, 1996). These authors suggest that the effects of cognitive and neurobiological alterations resulting from the experience of maltreatment likely mediate its effect on the sensitization to later stress. The cognitive component refers to the development of negative internal working models, negative cognitive schemas, and/or negative attributional styles in the context of a history of maltreatment (Gibb et al., 2001; see Harkness & Lumley, 2007). Cross-sectional and prospective studies have found that these negative thinking patterns mediate the relation of childhood maltreatment to depression (e.g., Feiring, Taska, & Lewis, 2002; Lumley & Harkness, 2007, 2009), even after controlling for levels of initial psychopathology (Feiring et al., 2002). It is believed that over time and across frequent usage, these negative cognitive processes consolidate and become more rigid through neurobiological and cognitive-emotional stability (Harkness & Lumley, 2007; Paus, 2005).

The neurobiological aspect of Harkness and Lumley's proposed model refers to dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis response to stress, presumably resulting from the experience of maltreatment. The HPA axis involves interactions between several glands (i.e., hypothalamus, pituitary, and adrenal glands) and results in the release of the glucocorticoid hormone cortisol from the adrenal cortex, which prepares the body to respond to stress by facilitating the "fight-or-flight" response (Sapolsky & Plotsky, 1990). Typically, an inhibitory feedback mechanism terminates this response once the stressor has passed. However, in the face of chronic stressors (e.g., childhood maltreatment), the HPA axis remains activated, which can result in chronically

elevated levels of glucocorticoids (Sapolsky, Krey, & McEwen, 1984) and subsequent neural changes, such as hippocampal atrophy (Sapolsky, Uno, Rebert, & Finch, 1990; Vythilingam et al., 2002) and increased cortisol release (Heim, Newport, Bonsall, Miller, & Nemeroff, 2001). It has been suggested that as these alterations stabilize across development through neurobiological maturity (Paus, 2005), their effects become more severe and long-lasting (Harkness & Tucker, 2000).

Harkness and Lumley (2007) suggested that even prior to the very first episode of depression, the abovementioned cognitive and neurobiological changes associated with maltreatment may result in a vulnerability to depression that can be activated in the face of proximal stress. More specifically, when faced with stress, negative cognitive networks are more easily activated and the HPA axis responds in a hyper-reactive way, resulting in a greater depressogenic response for individuals with a history of maltreatment compared to those without. At this point, it can only be speculated that these mechanisms help account for the findings of the present study. Considerable work is further required to characterize the nature of depression vulnerability acquired from a history of childhood maltreatment. In order to examine the full model suggested by Harkness and Lumley, longitudinal studies examining within- and between-subject differences in these potential mediating variables are needed.

Although the relation between childhood maltreatment and stress sensitization was not observed in the adult sample, this does not necessarily imply that stress sensitization is absent among these individuals. Stress sensitization has been found consistently in adult samples (e.g., Lenze et al., 2008; Ormel et al., 2001; Stroud, Davila, Hammen, & Vrshek-Schallhorn, 2011) and there is no reason to assume its absence in the

present study. What may differ for this age group, relative to adolescence, is the role or extent that childhood maltreatment contributes to sensitization. Specifically, I suggest that childhood maltreatment may play a more prominent role in triggering adolescent depression in the face of stress, relative to adulthood, because the onset of depression is more proximal to the actual traumatic experience. Over time and across development, other factors may become more important than a history of adversity in driving stress sensitization, such as the consolidation of negative cognitive schemas and neurobiological alterations and the experience of chronic difficulties. As such, the relation between childhood maltreatment and stress sensitization may become negligible in adulthood.

As discussed above, negative cognitive schemas resulting from the experience of maltreatment (e.g., Gibb et al., 2001; Nolen-Hoeksema, Girgus, & Seligman, 1992) are believed to become more rigid and engrained in individuals across development (e.g., Segal et al., 1996). Perhaps over time, these schema structures become so integral to an individual's identity and so easily activated in the face of a minor setback, that they play a progressively larger role in driving stress sensitization and triggering subsequent depressive episodes. The same concept can be applied to neurobiological changes. Studies have demonstrated the emergence of more significant neural changes across development. For example, a relation between childhood maltreatment and reduced hippocampal volume has been found in adults (e.g., Vythilingam et al., 2002) but not in children (e.g., De Bellis et al., 2002). This suggests that although structural and functional neural changes may begin to emerge soon after maltreatment (i.e., during childhood or adolescence), these changes only consolidate once neurobiological stability associated with development has occurred. Stemming from this, perhaps as neural changes stabilize

over time, they come to play a more primary role in stress sensitization. Consequently, other risk factors that were previously the primary contributors to stress sensitization (e.g., a history of childhood maltreatment) may become less significant.

Another possibility is that stress sensitization in adulthood originates from having an elevated baseline level of chronic difficulties, which are ongoing stressors that typically last several months. Studies have consistently shown that individuals with a history of maltreatment experience higher levels of chronic stressors, such as interpersonal problems, financial difficulties, and health problems, than those without such a history (e.g., Bifulco, Bernazzani, Moran, & Ball, 2000; Cicchetti & Toth, 2005; Kendler et al., 1993). Further, adults in general may naturally experience more ongoing problems than younger individuals because their realm of experiences, responsibilities, and social interactions tends to be wider, allowing for greater opportunity for problems. When such an elevated baseline level of chronic difficulties is present, minor SLEs may be all that are required to trigger an episode of depression (Brown & Harris, 1986). In support of this, studies have found evidence for the mediating role of chronic difficulties in the relation of childhood adversity to adult depression (e.g., Bifulco et al., 2000; Kendler et al., 1993; Kessler & Magee, 1994). Therefore, it is possible that stress sensitization occurring in adulthood is better accounted for by a chronically adverse environment rather than distal risk factors, such as a history of childhood trauma.

Overall, the discussion above suggests that although the relation between childhood maltreatment and stress sensitization may be apparent when depression onset is more proximal to maltreatment (e.g., during adolescence), other factors may become more important in driving stress sensitization as development occurs and more time has

elapsed between maltreatment and depression onset. In order to address this hypothesis, longitudinal studies examining the within-subject course of stress sensitization across development are needed. Furthermore, it is important for future studies to account for pre-existing levels of chronic difficulties to help shed some light on the mechanism driving stress sensitization in adulthood.

Because of the heterogeneous nature of childhood maltreatment, I examined the relative specificity of particular types of abuse (i.e., physical versus sexual versus emotional) to the childhood maltreatment – stress sensitization relation. The results of these descriptive analyses revealed that the relation of childhood maltreatment to stress sensitization was specific to emotional abuse, and was not found for physical or sexual abuse. This finding is consistent with the literature demonstrating a preferential association between emotional abuse and negative cognitive style (e.g., Gibb, 2002), and implicating the consequences of emotional abuse as more severely depressogenic than those of physical or sexual abuse (e.g., Boudewyn & Liem, 1995). For example, Hankin (2005) demonstrated that of emotional, physical, and sexual abuse, emotional abuse was the only type that prospectively predicted changes in depressive symptoms, while controlling for initial depression levels and overlap between maltreatment types. Similarly, Gibb et al. (2007) found that among individuals with a history of childhood maltreatment, those who experienced emotional abuse were more likely to meet diagnostic criteria for MDD than were those with a history of physical or sexual abuse.

Rose and Abramson (1992) suggested that childhood emotional abuse may contribute a particular cognitive vulnerability to depression because the experience directly provides the individual with information from the perpetrator that can serve the

formation of depressive cognitions (e.g., negative views of the self; “I am stupid/worthless/ugly”). In contrast, the authors suggested that physically or sexually abused individuals may attribute their experiences to other causes outside of the self. This reasoning can be applied to the specificity of emotional abuse to the childhood maltreatment – stress sensitization relation, such that adolescents with a history of emotional abuse may be more likely to respond to minor SLEs with a depressive response because they are more cognitively primed to do so than those with a history of physical or sexual abuse. Additional studies are required to replicate this finding, ideally within the psychobiological framework suggested by Harkness and Lumley (2007).

The specificity of the childhood maltreatment – stress sensitization relation to independent events is consistent with findings implicating independent events as more etiologically relevant to the precipitation of depression than dependent events (e.g., Shrout et al., 1989; Stroud et al., 2011). Harkness et al. (2006) suggested that independent SLEs may be especially central to the onset of depression in adolescence because many of the severe stressors that adolescents experience are in fact outside of their control (e.g., parental separation, an unwanted move across the country that resulted in the loss of only friend). In contrast, if these or similar events were rated for adults, they would likely be considered dependent on the actions of the individual. This is not to imply that adolescents do not experience dependent events because surely they do. Rather, Harkness et al. (2006) suggested that adolescents may not experience the same range or severity of dependent events as adults. As such, the relevance of independent SLEs to depression onset and the prevalence of these events in adolescence may help account for the specificity of sensitization to independent SLEs in the present study.

Contrary to expectations, depression history did not moderate the relation of childhood maltreatment to stress sensitization among adolescents. This finding runs counter to that of Harkness et al. (2006) who found that the relation of childhood maltreatment to stress sensitization in adolescents was specific to those on their first episode. It is somewhat surprising considering that many of the depressed adolescents in this study came from the same sample used by Harkness and colleagues. Methodological issues likely contributed to the failure to replicate these past findings. More specifically, the main goal of this study was to identify a very specific three-way interaction between maltreatment history, age group, and depression history. Out of the eight possible cells, I was looking to find evidence for stress sensitization among only one (i.e., maltreated adolescents on their first depressive episode). Finding such an interaction likely requires more power than the present had. Another methodological consideration is that the adolescents and adults were not well matched with respect to depression history. The majority of adolescents were experiencing either their first episode of depression or their first recurrence (59.3% and 27.1%, respectively), whereas adults had a broader range of previous episodes. Further, age of first onset differed across the groups with adults reporting a significantly older age than adolescents (i.e., 24 years versus 13 years). Therefore, there may have been too much variability across age groups with respect to depression history to find a main effect or interaction of this variable. In order to address these concerns, efforts should be made to match adolescents and adults with respect to depression history, including number of previous episodes and age of first onset.

Finally, for dependent SLEs, a main effect of age was found, such that adults reported significantly higher severity levels of dependent SLEs prior to depression onset

than adolescents. Although not predicted, this finding is consistent with the abovementioned discussion regarding the experience of independent and dependent SLEs across the lifespan. More specifically, the range and severity of dependent SLEs may be greater for adults than adolescents because adults generally have larger social spheres and more responsibility in various domains (e.g., occupation, family). As such, there may be greater opportunity for dependent SLEs to be generated. Similarly, as suggested by Harkness et al. (2006) and alluded to above, many events that are considered dependent on the individual's behaviour in adulthood (e.g., separation from spouse) may have been rated as independent for adolescents (i.e., the adolescent's parents' separation would have been rated). As such, it is easy to see how adults may experience a greater number and severity of dependent SLEs prior to a depressive episode than adolescents (see also Harkness, Alavi, Monroe, Slavich, Gotlib, & Bagby, 2010).

Maltreatment Characteristics and Stress Sensitization

Support was not found for predictions regarding specific characteristics of maltreatment (i.e., timing, comorbidity, and severity) that may differentially impact the childhood maltreatment – stress sensitization relation. Despite these null findings, the pattern of results provides some interesting preliminary insights into the role childhood maltreatment plays in stress sensitization and potential avenues for future research. In a general sense, the findings suggest that it is simply the experience of severe maltreatment, and not particular characteristics of it, that subsequently sensitizes individuals to the effects of stress. That is, once an individual's experience of maltreatment has reached a 'severe' threshold, other aspects of the experience do not appear to further sensitize the individual. It is interesting to note, however, that the one characteristic not explicitly

hypothesized in this study – type of maltreatment – was preferentially related to stress sensitization, with emotional abuse showing the strongest association. As previously discussed, this finding is consistent with the literature implicating the effects of emotional abuse as more depressogenic than those of physical or sexual abuse (e.g., Hankin, 2005).

Although it is possible that fine-grained aspects of maltreatment are largely irrelevant in driving stress sensitization once a severe level of maltreatment has been experienced, several methodological issues may have played a role in the null findings. First, only individuals with a history of ‘severe’ maltreatment (i.e., “marked” or “moderate” ratings of maltreatment) were included in analyses pertaining to maltreatment characteristics. Since the goal was to essentially identify dose-response relations between these characteristics and stress sensitization, the inclusion of a greater spectrum of experiences (i.e., as might be seen in maltreatment ranging from little or mild to severe) may have influenced the presence of these relations. Second, as previously alluded to, there is some ambiguity associated with categorizing maltreated individuals based on factors such as maltreatment timing and comorbidity. As such, some of the classification decisions made in the present study may differ from those of others. However, it is important to note that prior research was used as a guideline for making these decisions whenever possible.

The literature examining the relation of fine-grained characteristics of maltreatment to stress sensitization in depression is scant and deserving of further study. Additional studies that take the abovementioned concerns into consideration are needed to confirm whether maltreatment characteristics, with the exception of type, are largely

unimportant in sensitizing individuals to the effects of stress over and above a history of severe maltreatment.

Limitations

The results of this study should be considered in light of a number of limitations. First, the present study was limited by small cell sizes. These small cell sizes may have contributed to the failure to find a significant three-way interaction between maltreatment history, age group, and depression history, as well as the failure to find support for some of the models examining the more fine-grained maltreatment characteristics. Therefore, replication of these models in studies of larger samples is warranted. Second, the effect sizes for most significant results were small (Ferguson, 2009), which may limit the interpretability of the findings. However, effect sizes for nonsignificant effects were negligible and similar effect sizes for significant results have been found in previous research (e.g., Harkness et al., 2006). Third, the continuous variable of age was dichotomized into age group, which may have resulted in a loss of statistical power, lower reliability, and an increased chance of type I or type II errors (see Chen, Cohen, & Chen, 2007). Such a procedure has been suggested to be particularly problematic when underlying rationale is lacking; however, in the present study, the decision to dichotomize age was based on theoretical reasoning suggesting that conceptual differences exist between the two age groups. Nevertheless, future studies will need to take this issue into account when examining stress sensitization across development. Fourth, this study relied on volunteer participants who may not be entirely representative of the population of adolescents and adults with depression. It is worth noting that with the exception of ethnicity, the present sample included a heterogeneous group of individuals, ranging in

diversity with respect to age, socioeconomic status, geographic region, childhood upbringing, and depression severity. Nevertheless, additional studies using large epidemiological samples are needed to further investigate the relations of the present study.

Finally, this study relied on retrospective self-reports of SLEs and childhood experiences, which were subsequently rated for severity. This procedure may be viewed as problematic in that there are two possible sources of biases, which include a bias on the part of respondents in recalling their experiences and a bias on the part of the raters. The LEDS and CECA interviews and rating systems have built-in safeguards to address such concerns. First, to address the concern of respondent bias, the format of the interviews is well suited to priming autobiographical memory by encouraging participants to tell a story about their experiences, probing for both positive and negative contextual detail. In addition, interviewers are trained to query only about the practical details of the participants' experiences and not about their emotional reaction to stressors or the relation of stressors to depression. Second, with respect to rater bias, the information provided to the raters is first filtered through a research assistant who deletes any subjective information or unnecessary detail that may influence the rating. Furthermore, ratings are based on manualized examples that raters use to justify their decisions, helping to ensure standardization. Because of their rigorous and contextual nature, the LEDS and CECA have largely been regarded as the gold standards of retrospective accounts of life events and childhood histories, respectively, with superior reliability and validity over traditional self-report questionnaires (e.g., Bifulco et al., 1994; Brown & Harris, 1989; McQuaid et al., 2000).

Conclusion

The current investigation sought to examine whether the relation between childhood maltreatment and stress sensitization differs across developmental stages and depression history. This study was the first to demonstrate that childhood maltreatment is associated with lower severity levels of independent SLEs prior to depression onset in adolescents and not adults. However, contrary to expectations, this relation was not specific to adolescents on their first episode of depression. This study also investigated whether particular aspects of the maltreatment experience were differentially associated with stress sensitization. In general, the results suggest that it is simply the experience of severe maltreatment, and not particular characteristics of it, that sensitizes individuals to the effects of stress. However, maltreatment type was an important contributor, with the relation of childhood maltreatment to stress sensitization being specific to emotional abuse and not physical or sexual abuse.

The present findings have potentially important clinical implications and may be helpful in informing treatment options for individuals with a history of maltreatment. Specifically, adolescents with a history of maltreatment who are seeking psychotherapy for depression may respond best to cognitive and behavioural strategies that focus on identifying and coping with SLEs that may trigger depression. Further, clinicians may need to be especially vigilant in assessing a full and accurate account of maltreatment history in adolescents seeking treatment for depression, since a history of emotional abuse appears to be most strongly depressogenic. Similarly, depressed adults with a history of maltreatment may also benefit from cognitive and behavioural strategies, particularly those that focus on restructuring negative cognitive schemas and/or identifying and

managing sources of chronic stress. However, more sound suggestions regarding treatment options for adults require investigation into factors that drive stress sensitization in adulthood.

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Appendix A

Sample Questions from the Childhood Experience of Care and Abuse Scale (CECA)

1. RELATIONSHIP WITH PARENT FIGURES

- MOTHER** How well did you get along with your mother?
Were you close?
- (Affection) Was she affection towards you?
How would she show it? Did you ever wish she were more affectionate?
- (Companionship) Did your mother spend much time with you when you were little?
Did you enjoy this time?
What sorts of things did you do together? Were there any special activities or games?

Could you share a laugh together?
- (Antipathy) Was she hard to please?
IF YES: In what sort of way? What makes you say that?
Was she very critical of you?
Was she ever cold and distant?
Did you ever feel like she didn't want you or didn't like you?
Did she ever say anything rejecting? What sorts of things would she say? How often would she say these things?
Did you argue much with her? What about? How often? What were the arguments like? What sorts of things were said?

Did she ever push/slap/hit you?
IF YES: Where on your body? How often did this happen? Were you ever injured as a result?
IF YES: Did you receive or require medical attention?

Was your relationship the same when you were younger? Did it change at all over childhood?
IF YES: When was that? In what way did it change? Why do you think that was?
- FATHER** (Same questions as above)

6. SEXUAL ABUSE

Have you ever had an unwanted sexual experience?

IF YES, THEN ASK SEXUAL ABUSE QUESTIONS.

IF NO, THEN ASK SOME ADDITIONAL SCREENING QUESTIONS:

- (i) Has anyone *ever* tried or succeeded in having sexual intercourse with you against your will?
- (ii) Can you think of any upsetting sexual experiences you have had? Or any unwanted sexual experience with someone in authority, like a teacher or doctor?
- (iii) What about a situation where you were nearly involved in an unwanted sexual incident, but avoided it?

FOR ALL SEXUAL ABUSE EXPERIENCES, ASK THE FOLLOWING:

INCLUDE - ANY SEXUAL CONTACT WITH AN ADULT
 - ANY NON-CONTACT SEXUAL ENCOUNTER WITH ADULT RELATIVE

Who was involved?
 Was _____ living in the same house as you when that happened?

Can you tell me what happened?

(Date of Abuse) How old were you when the abuse first happened?
 How long did it go on for?
 How many times or how often did it happen?
 When did it end? What were the circumstances? Why did it end then?

(Physical Contact) Did it involve touching or not?

IF YES:
 Can you tell me what exactly happened?

Did you have to touch him/her? Where?
 Did it involve masturbation? Was that to him/her or to you?

IF APPROPRIATE:
 Did it involve anything else, like assault with an implement?
 Did it involve sexual intercourse?

IF NO CONTACT:
 Did he/she ask you to have sex with him/her?
 What was said? Was he/she persistent?

How did you avoid him/her touching you?
 Did you get away?

Were there other times when he/she tried but you were able to avoid it?

(Violence) Did he/she make any threats or use violence?
 Did he/she ever use physical force on you?
 Did he/she ever threaten to hurt anyone else?

(Support) Were you able to tell anyone?
 Who was that? What was their reaction?
 Were they sympathetic and helpful?

When did you first confide?
 Was anyone else helpful?
 Was anyone critical or unhelpful about it?

(Official Contact) Did you see a doctor or social work at that time? Police?
IF YES:

Were they helpful or sympathetic?
Did anyone say or do anything that made you feel worse?
Was there a court case?
Did you have to be present? What happened?

ASK ALL: Have there been any OTHER times when you've been sexually approached against your wishes before age 17?

IF YES, THEN REPEAT ABOVE SECTION

Appendix B

Sample Questions from the Life Events and Difficulties Schedule (LEDS)

SECTION I. HEALTH

In this section, you are also looking for change points in health and/or interpersonal difficulties. Look for both positive and negative changes in condition. If the illness affects S or a family member, also ask about family history of disease or ailment. Ascertain exactly what the subject knows regarding diagnosis, prognosis, etc.

Has anyone in the family or any close friends been ill?

What about you? Your spouse, children, parents?

What was the illness?

When did that occur?

How serious was it? What were the symptoms?

Did they take any medication for it? Are they still? Any other treatment (e.g., surgery, radiation, etc.)?

Hospital stay?

What is the prognosis?

Anyone off work because of it? For how long?

When were they able to do things again, stop using crutches/cane, stop taking medication, etc.?

How did this affect you?

Has anyone been admitted to or left the hospital during the time period?

Who? When did that occur?

For what illness?

Medical tests or procedures done

Surgery

Was it inpatient or outpatient?

Local or general anesthetic?

Intense Care Unit

Critical Care Unit

Was it a routine admission or an emergency?

Did you go to the emergency room? Why?

For how long?

What changes were involved for you?

Did your daily routine change at all? Did you have additional responsibilities?

What is the medical outlook?

If someone outside household: How involved were you?

Have any relative or close friends died?

Who? What of? When did this occur?

Was it expected or sudden?

How close were you to _____?

Did you attend the funeral/service? Was this your first funeral/service?

Were you involved at all? Any role in the funeral? Any role/responsibility afterward?

Have you received any bad news during the time period about an illness that's been going on for some time? Like a chronic illness?

Whose illness? When did you find out? What was the news?

How long has it been going on for? What are the symptoms? Treatments? What is the prognosis?

How does this affect you?

Appendix C

Table 4

ANOVA Test Results for Independent and Dependent Stressful Life Events

	Independent Events			Dependent Events		
	<i>F</i>	<i>p</i>	partial η^2	<i>F</i>	<i>p</i>	partial η^2
Main effects						
Childhood maltreatment	1.13	.289	.006	3.20	.075	.016
Age group	0.00	.985	.000	1.92	.168	.010
Depression history	0.12	.735	.001	0.02	.886	.000
Interaction effects						
Childhood maltreatment x age group x depression history	0.16	.687	.001	0.11	.740	.001
Childhood maltreatment x age group	9.17	.003	.050	1.60	.208	.008
Childhood maltreatment x depression history	0.13	.718	.001	0.00	1.00	.000
Age group x depression history	0.16	.686	.001	0.62	.430	.003
Pairwise comparison follow-up						
Maltreatment vs. no maltreatment, adolescents	7.19	.008	.040			
Maltreatment vs. no maltreatment, adults	1.70	.194	.008			
Adolescents vs. adults, no maltreatment	4.50	.035	.022			
Adolescents vs. adults, with maltreatment	2.62	.107	.013			

Appendix D

Table 5

ANOVA Test Results by Abuse Type for Independent Stressful Life Events

	<i>F</i>	<i>p</i>	partial η^2
Model 1: Physical abuse			
Physical abuse	0.81	.369	.004
Age group	0.05	.826	.000
Physical abuse x age group	0.19	.662	.001
Model 2: Sexual abuse			
Sexual abuse	.031	.576	.002
Age group	0.68	.411	.003
Sexual abuse x age group	3.49	.063	.017
Model 3: Emotional abuse			
Emotional abuse	0.92	.339	.005
Age group	0.82	.365	.004
Emotional abuse x age group	11.88	.001	.056
Emotional abuse vs. no abuse, adolescents	6.10	.014	.030
Emotional abuse vs. no abuse, adults	5.09	.024	.028
Adolescents vs. adults, no emotional abuse	5.52	.020	.027
Adolescents vs. adults, with emotional abuse	6.54	.011	.032

Appendix E

Table 6

ANOVA Test Results by Maltreatment Characteristics for Independent and Dependent Stressful Life Events

	Independent Events			Dependent Events		
	<i>F</i>	<i>p</i>	partial η^2	<i>F</i>	<i>p</i>	partial η^2
Model 1: Developmental period						
Developmental period	0.26	.776	.006	0.69	.503	.016
Age group	0.36	.553	.004	0.32	.574	.004
Developmental period x age group	0.51	.604	.012	0.44	.645	.010
Model 2: Maltreatment duration						
Maltreatment duration	0.59	.444	.007	0.00	.957	.000
Age group	0.69	.409	.008	0.00	.938	.000
Maltreatment duration x age group	0.37	.543	.004	0.46	.502	.005
Model 3: Maltreatment comorbidity						
Maltreatment comorbidity	0.12	.729	.001	0.95	.331	.010
Age group	2.23	.138	.023	4.28	.041	.042
Maltreatment comorbidity x age group	1.17	.283	.012	0.06	.811	.001
Model 4: Maltreatment severity						
Maltreatment severity	0.07	.786	.001	0.04	.841	.000
Age group	0.24	.626	.002	0.65	.421	.007
Maltreatment severity x age group	0.97	.326	.010	0.03	.864	.000