EARLY ADVERSITY AND SEVERE EVENTS: SENSITIZING FACTORS FOR DEPRESSION

A Thesis

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by

Heather L. Holleman

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Cindy Bergeman, Director

Graduate Program in Psychology
Notre Dame, Indiana
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INTRODUCTION

Major depression is one of the most prevalent psychological disorders, with a lifetime prevalence of nearly 17% of the population in Western countries (Kessler et al., 2005). Among all medical disorders, major depression is one of the leading causes of disability in the United States and worldwide (World Health Organization; WHO, 2001). Major depression significantly impairs an individual’s general functioning, health-related quality of life, and work productivity, resulting in significant economic and social impairment (Booth et al., 1997; Eaton, Anthony, Mandel, & Garrison, 1990; Greenberg et al., 2003). In addition, experiencing major depression significantly increases the risk of suicide. The National Institute of Mental Health (NIMH, 2006) has estimated that 60% of people who successfully commit suicide have a history of mood disorders (including unipolar depression, dysthymia, and bipolar depression). What’s more, the cumulative morbidity of depression is even greater if one includes subsyndromal symptomatology (Angst, Kasper, & Weiller, 2000). Finally, major depression often has a severe and long-lasting impact on an individual’s life, and often recurs several times over the life course. Indeed, it is estimated that 60% of individuals who have had one episode of major depression will have another, 70% of individuals who have had two episodes will have a third, and 90% of individuals who have had three episodes will have a fourth (American Psychiatric Association, 2000). In light of these tremendous burdens imposed on people with depression, their wider social networks, and society, research into the origins of this major mental health problem is a high priority.
It is clear that most episodes of major depression are preceded by stressful life events. Indeed, in a review of studies on community samples, Mazure (1998) noted that up to 80% of depressed cases were preceded by major life events. What is not clear, however, is the variability in the depressogenic effects of major stress. That is, whereas stressful events commonly predate onset of a depressive episode, most people do not become depressed after experiencing a major stressor (Hammen, 2005). For example, Brown and Harris (1978) estimated that only 20% of individuals who experience a severe life event become depressed, suggesting other mechanisms are responsible for bringing about a depressive episode in the context of adversity. It therefore essential to understand the factors that may sensitize an individual to stress, thereby affecting susceptibility to depression upon exposure to a major life event.

There are a number of factors that are hypothesized to sensitize an individual to stress and make the person more likely to develop depression. For example and most basically, having once experienced an episode of depression predicts a greater likelihood that the individual will experience a subsequent depressive episode, suggesting possible sensitization (Solomon et al., 2000). More recent research indicates a strong genetic component in the development of major depression, which may be another source of sensitivity to developing depression under stress (Costello et al., 2002). Finally, early adversity, in the form of abuse, neglect, or death of a parent, appears to have a substantial effect on an individual’s susceptibility to adulthood depression (Hammen, 2000). In the following, findings from these literatures will be outlined and highlighted.
I. Possible factors contributing to stress sensitization. Clearly, many factors may influence an individual’s sensitivity to stress, making him or her more or less susceptible to experiencing a depressive episode following a major event. These hypothetical sensitizing factors likely play a large part in determining which people will develop major depression following a severe event and which people will be stress resistant. In order to fully understand the impact that severe life events have on the development of a major depressive episode, we must understand these other factors that play a permissive role in bringing about depression under adversity.

II. History of depressive episodes and the kindling hypothesis. Severe stressful life events commonly precede episodes of major depression, but these types of stress appear to be more common prior to the onset of depression for individuals with fewer prior episodes. Indeed, nearly 50% of first episode cases of depression are preceded by a severe life event (Monroe, Slavich, Torres, & Gotlib, 2007), and onset is likely to occur within the first three weeks, and perhaps up to six months, after experiencing a negative event (Brown & Harris, 1989). In contrast, far fewer recurrences for people with many lifetime episodes of depression are preceded by these major life events (e.g., Monroe et al., 2007).

Several theories have been proposed to explain why major stressful life events are more typically associated with first or early lifetime episodes of depression when compared to later recurrences of the disorder. The kindling hypothesis (Post, 1992) suggests that neurobiological changes occur with life stressors and with each
successive depressive episode, and that these combined changes lead to an increasing sensitivity to stress for triggering subsequent mood disorder episodes. Within this framework, individuals are progressively more susceptible to the recurrence of depression as the episodes appear to be less strongly associated with major environmental adversity (Kendler, Thornton & Gardner, 2000).

The kindling hypothesis explains how major life stress more readily precipitates onset of early depressive episodes, but the major forms of stress seem to lose importance with regard to precipitating onset of later episodes. Post’s (1992) kindling model is based on animal research that demonstrates the effects of repeated electrophysiological stimuli in the precipitation of seizures. Results from these studies indicate that progressively lower levels of electrical stimulation become capable of triggering the seizure response over time. Post (1992) extended this work to affective disorders and proposed that progressively lower levels of stress (i.e., stimulus) become capable of precipitating onset of affective disorders over repeated episodes. Thus, it might be that major forms of stress are required to first bring about a depressive episode, but with sensitization, progressively more minor forms of stress acquire the capability of triggering a recurrence.

The basic finding that major life events are more commonly associated with first or early lifetime episodes of depression has been well replicated (Monroe & Harkness, 2005; Post, 1992). For example, in a sample of older adults Ormel, Oldehinkel, and Brilman (2001) explored the association between life stress and onset of major depression, particularly the differential prediction of onset in first episodes.
versus recurrent episodes. All participants were aged 57 or older and had experienced at least one depressive episode (full or subsyndromal) in the past nine months. Results from this study indicate that severe life events (as assessed by the LEDS) have a stronger association with first episodes of depression than with recurrent episodes.

III. Genetic sensitization. Sensitization to stress may also occur on a genetic level, suggesting that one’s sensitivity to stressful events is dependent on genetic make-up (Costello et al., 2002). Research in behavioral genetics consistently demonstrates an increased risk for depression among individuals who are at high genetic risk compared to those at low genetic risk (e.g., Kendler et al., 1995), although specific genes responsible for this increased risk are only recently being investigated.

Caspi and colleagues (2003) sought to test this G x E interaction hypothesis in relation to depression with 847 Caucasian adults (52% male). Participants were divided into three groups: s/s homozygotes (n=147, 17%), s/l heterozygotes (n=435, 51%), and l/l homozygotes (n=265, 31%). Participants were assessed for stressful life events occurring between age 21 and 26, and depression occurring at age 26. Results from a moderated regression revealed a significant interaction between 5-HTTLPR and life events such that the effect of life stress on self-reported depression at age 26 was more robust (p=.02) in individuals with an “s” allele than in homozygotic “l/l” individuals. Additionally, the interaction indicated that major depression was predicted by stressful life events in individuals with an “s” allele but not in “l/l”
homozygotes (p=.056). Similarly, when the measurement of stressful life events was extended to childhood stress, results indicated that childhood maltreatment predicted adulthood depression in individuals with an “s” allele, but not among “1/1” homozygotes (p=.05). This groundbreaking work by Caspi and colleagues has launched a new era of studies on diathesis-stress interactions, and points to the potential key role that biogenetic susceptibility may play in translating major stress into major depression.

IV. Considering kindling and genetics combined. Of course, the possibility exists that processes similar to kindling and genetic sensitization may be operative in terms of moderating stress impact on depression. Related to understanding such matters, Kendler et al. (2001) proposed a “speed-of-kindling” model and a “pre-kindling” model. These models were developed to clarify the relationship between genetic risk, stressful life events, and depressive episodes. The “speed-of-kindling” model suggests that everyone begins with a similar degree of association between risk of depression in response to environmental adversity, but genetic risk is correlated with the speed of kindling process over successive recurrences (i.e., how fast the changing association between stressful events and depression occurs). According to this “speed-of-kindling” model, individuals who are at high genetic risk for depression are “rapid kindlers,” such that the brain learns more quickly to become depressed with progressively lower amounts of stress.
In contrast, the “pre-kindling” model proposes that genetic risk determines the initial strength of association between risk for depression and environmental adversity. Individuals who are at low genetic risk require considerable stress to precipitate depression, and consequently are less likely to develop depression under milder degrees of stress or spontaneously. Individuals at high genetic risk, however, would be “pre-kindled” and thus more likely to develop depression in the face of milder stress or spontaneously. In this case, major depression can come about without severe life stress either through multiple previous episodes or through high genetic risk.

Kendler and colleagues (2001) tested the “speed-of-kindling” and “pre-kindling” models with female-female twin pairs. Across all genetic risk groups, stressful life events and previous depressive episodes each predicted the risk of onset for a major depressive episode. The “pre-kindling” model, however, was supported for the women at higher genetic risk. That is, for these women there was relatively little change in the presence of major stress over successive recurrences. Results indicated that the association between stressful life events and onset of depression for individuals with low genetic risk had significantly declined by the third depressive episode. The kindling pattern appeared to be most prevalent for individuals in the low genetic risk group, as those in the high genetic risk group had a weaker association between stressful life events and depression. Indeed, individuals at high genetic risk appeared to be “prekindled” such that they looked similar to the low genetic risk group after their third episode. The traditional kindling model best described the
findings for women at low genetic risk. That is, for these women the association between stressful life events and onset of depression was strongest for those with the fewest prior depressive episodes. These results again suggest two distinct pathways to kindling or sensitization, one through history of depressive episodes (in the low genetic risk group) and one through heritability (in the high genetic risk group).

V. Early adversity. Early life stress has been shown to be associated with a variety of psychological disorders in adulthood, in particular mood disorders, and more specifically major depression (Brown & Anderson, 1991; McCauley et al., 1997). For example, it has been reported that women who were abused in childhood are four times more likely to develop depression in adulthood (Mullen et al., 1996). There are a number of ways that early life stress influences biological and psychological factors that may sensitize an individual to depression. Gold, Goodwin, and Chrousos (1988) suggested that biological stress response mechanisms may be sensitized as a result of childhood adversities, leading to increased risk of depression. Kessler and Magee (1993) proposed that early life stress leads an individual to acquire certain cognitive and biological vulnerabilities that decrease the amount of stress needed to precipitate a depressive episode.

Although at a general level there is considerable consistency in demonstrating early adversity as a risk indicator for major depression (Hammen, 2000), there is also a good deal of speculation about what types of early adversities are most deleterious for the development of subsequent depression. The most commonly proposed factor
is early loss, either literally (i.e., death of a parent), or practically (e.g., divorce, parental incarceration, and neglect; Brown & Harris, 1978). Other types of early adversities found to be related to adult depression include physical, emotional, or sexual abuse, neglect (Garnefski, van Egmond, & Straatman, 1989; Hammen, 2000), family violence (Kessler & Magee, 1994), marital discord, and parental mental illness or substance abuse (Hammen, 2000).

Numerous stress researchers have documented that early exposure to stressful life circumstances (e.g., abuse and neglect) makes an individual more likely to become depressed as an adult. Using data from the 1980 Islington series, Bifulco, Brown, and Harris (1994) assessed childhood adversity in terms of severe neglect, physical abuse, or sexual abuse before age 17. Results indicated that an index based on the presence of at least one of these adversities was associated with a two-fold increase in risk for depression during the study period. In another study, Kessler, Davis, and Kendler (1997) found that indexes of childhood adversity, prior psychiatric status, and current life stress predicted onset of major depression over 5 years, even when controlling for prior episodes of depression. Results from these studies underscore the significance of early family environment in the prediction of adult depressive disorders. In an epidemiological survey, Kessler and Magee (1993) found that early childhood adversity was a vulnerability factor for the development of major depression, both for first onset and recurrence. Bifulco and colleagues (1998) defined vulnerability factors “in terms of their capacity to increase risk in the
presence of stressful life events” (p. 40), and suggested that current vulnerability mediates the risk for depression stemming from childhood adversity.

Several theories have been proposed to explain how childhood adversity impacts adult experiences and results in depression. Early life experiences seem to work in one of two ways: 1) to increase the burden of stress in adulthood, or 2) to reduce the individual’s threshold for handling stress. In the first pathway, adversity continues throughout life, creating major stressors that can precipitate depression (i.e., stress generation; Hammen, 1991). In the second pathway, the psychobiological sensitivity of the person to stress may be altered, rendering the person depression-prone with lower levels of stress relative to persons without early adversity (i.e., stress sensitization; Hammen, 2000).

The role of early adversity and recent life stress in depression were examined in a recent study by Hammen and colleagues (2000). Employing a longitudinal study of depression, early adversity, and stress sensitization in women, Hammen, Henry, and Daley (2000) followed 121 recent high school graduates over two years. The participants were interviewed using the Structured Clinical Interview for DSM-III-R (SCID) to assess current and lifetime depressive episodes, and an episodic life stress interview (Hammen, 1991) to assess the severity and impact of specific stressors on the individual’s life. Follow-up interviews were administered at six months and then annually (by phone) and with questionnaires sent by mail.

Questionnaires used by Hammen et al. (2000) included the Beck Depression Inventory (BDI) and an adaptation of the Questionnaire of Early Childhood
Adversities (Kessler & Magee, 1993). Assessed childhood adversities included, a) serious parental drinking problem, b) parental mental health problem, c) death of a parent, d) experiencing or witnessing violence in the family, e) parent divorce or separation, and f) parent marital conflict. Follow-up interviews covered chronic stress in several domains (e.g., Hammen et al., 1987), current depressive symptoms, and stressful life events. Results revealed a stress-sensitization mechanism by which early adversity acted to lower the threshold for the amount of recent stress needed to trigger a depressive episode. When faced with similar degrees of total stress, women in this study who were exposed to one or more childhood adversities were more likely to become depressed than women who were not exposed to early adversity. Importantly, these effects were not accounted for by previous depressive history or by current chronic stressful conditions, suggesting that the aforementioned sensitization effect was not to long-term adversity, but rather was specifically related to childhood adversities.

Given that women in the study by Hammen et al. (2000) who had experienced early adversity developed depression after lower levels of stress exposure relative to their no-adversity counterparts, it suggests that they were sensitized to the depressogenic effects of life stress. There are several more specific pathways via which such sensitization processes may operate, and recent research in particular has provided some insights into the possible neurobiological underpinnings for such effects. Some of this intriguing work is reviewed next.
VI. Neurobiological consequences of early adversity. Stressors occurring during early stages of development might result in biological and cognitive changes in key stress regulatory systems, such that there is a subsequent lowering of the threshold for responding to later stressful conditions (Hammen et al., 2000). Indeed, as indicated previously, Gold, Goodwin, and Chrousos (1988) hypothesized that biological stress mechanisms are sensitized by exposure to acute stressors in childhood, and that some forms of depression arise from these stress mechanism defects.

There is an abundance of evidence that early adverse experiences are especially impactful on the developing organism during a window of vulnerability, increasing the subsequent risk for depression (Kessler et al., 1997; McCauley et al., 1997). During early development, the central nervous system (CNS) has considerable plasticity that may mediate the effects of early adverse experience on health and adaptation. After birth, there are critical periods during which certain areas of the brain are acutely susceptible to major structural changes brought about by adverse experiences that permanently affect cellular processes and structural integrity of particular neural pathways. These changes seem to take on greater significance when one considers them in the context of developmental demands, such that the stress and emotional trauma of childhood abuse could irreversibly shape the brain regions involved in emotional processing and stress responsiveness (Weiss & Wagner, 1998). It is also important to note that hormones and neurochemicals associated with stress reactions can have additional deleterious effects on the developing brain. Despite the
fact that humans have relatively low plasma levels of cortisol at birth, the levels of biologically active cortisol are enough to have clear physiological effects (Gunnar, 1992).

Using clinical human models, Heim and colleagues (2001) compared the pituitary adrenal responses of four groups of women. The study groups included 1) women with a history of childhood abuse who were not currently depressed, 2) women with a history of childhood abuse who were currently depressed, 3) women without a history of childhood abuse who were currently depressed, and 4) a control group of women who were not depressed and had no history of childhood abuse. Results indicated that women with a history of childhood abuse who were not currently depressed experienced sensitization of the pituitary and counter-regulative adaptation of the adrenal gland. The findings suggested that women with a history of abuse hypersecrete CRH upon exposure to stress, leading to depression and pituitary CRH receptor down-regulation. Upon exposure to intense, frequent stressors, the neurochemical systems that mediate the stress response become more sensitive to future stressful events. Increased receptor sensitivity alters the responsivity of the catecholamine systems (which stimulate release of ACTH and CRH) that are responsible for mediating stress reactions.

Some have suggested that many of the neurobiological (e.g., cortisol dysregulation) findings associated with depression are related to early adversity (Heim, Plotsky, & Nemeroff, 2004). It appears as though the combination of early life stress, biological mechanisms, and current life stress as an adult may ultimately shape
individual stress responsiveness and the manifestation of depressive disorders. In fact, Heim et al. (2004) proposed a new typology for depression based on neurobiological and developmental changes. The authors argued that early life stress should be strongly considered in depression models and neurobiological subtypes of depression should be based on the presence or absence of early life stress. Although these neurobiological considerations are not analyzed in the present study, it is important to bear in mind that they may underlie many of the relationships between early adversity and depression.

**VII. Psychosocial pathways from early adversity to depression.** Early adversity may lead to higher levels of stress in adulthood (Hammen, 2000). Indeed, in a study of women with a history of childhood abuse or neglect, Bifulco et al. (2000) found severe childhood abuse or neglect to be related to higher levels of stress and to severe adverse events in adulthood. Further, they found both stress and early abuse or neglect contributed to the occurrence of clinical depression in adulthood. Specifically, both factors were necessary to model the incidence of chronic or recurrent depression in adulthood. Durbin, Klein, and Schwartz (2000) theorized that early adverse home environments have long-term deleterious effects on an individual’s coping and interpersonal styles, resulting in poor social support and greater levels of stress in adulthood. Although the exact mechanisms that lead to increased stress for individuals with a history of abuse are unknown, researchers have posited several
interacting theories that are beyond the scope of this paper (e.g., stress generation, Hammen, 1991).

The research presented thus far offers some theoretical insight into the mechanisms that may predispose individuals with a history of early adversity to adulthood depression. Drawing from the kindling theory (Kendler et al., 2001; Post, 1992), people with a history of early adversity may be prekindled, not at birth like those with high genetic risk, but following early adversity (before puberty), making them more vulnerable to depressive episodes. Further, these individuals are likely to evidence an earlier age of onset for first lifetime episodes of depression. In this way, it seems that childhood adversity acts as an early stress sensitization factor. Finally, given the enduring alteration in susceptibility to stress for individuals with early abuse, one would expect a greater history of prior depressive episodes compared to depressed persons without an early abuse history.

VII. Statement of the problem and proposed research

The present study focuses on how early adversity is related to the association between major life events and past history of depressive episodes. Given that only 20% of individuals who experience a severe life event will become depressed (Brown & Harris, 1978), it is essential to investigate other mechanisms that bring about depressive episodes in the face of life stress. Several predictions about the expected relations between these variables follow from the previous discussion. First, we hypothesize that both early life stress and current, severe life stress predict personal
history of depression, but in the opposite direction. That is, current severe life events have been shown in the present sample to be inversely related to prior history of depression (i.e., fewer prior depressive episodes). In contrast, we hypothesize that early adversity will predict a greater number of past depressive episodes. Second, we would also expect, then, that within a depressed sample there will be an inverse relationship between the presence of early adversity and the presence of recent major life stress (i.e., those with severe stress will be less likely to have early adversity). Third, given the hypothesized sensitivity of individuals with a history of early life stress, we predict that they develop depression more quickly than individuals without a history of early life stress when each group has experienced a severe life event. Finally, we predict a relationship between early life stress and age of onset such that early adversity will be associated with earlier age of onset.
METHOD

I. Participants. One hundred adults diagnosed with major depressive disorder (MDD) were recruited through advertisements and referrals from two outpatient psychiatry clinics at Stanford University. Approximately half of the individuals in the sample \((n=46)\) were receiving treatment. Participants with a high likelihood of current depression and relatively recent onset were selected, resulting in 98% of the sample having had an onset of depression within 2.5 years. Selected individuals were invited to the Department of Psychology at Stanford University, where they completed several self-report questionnaires and a diagnostic interview. Participants who met formal inclusion criteria (see below) returned one week later to complete additional measures, and subsequently returned for a third session to complete the life stress interview. Participants were compensated at a rate of $25 per hour.

The diagnostic interview administered during the first visit was the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 1996). Individuals who met criteria for current MDD according to the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV; American Psychiatric Association, 1994) were considered for study inclusion. These individuals were then screened to exclude current comorbid panic disorder and social phobia, as well as to exclude lifetime history of mania, hypomania, or primary psychotic symptoms. Additionally, individuals were excluded if they had a recent history (within the past six months) of alcohol or psychoactive substance abuse or
dependence. Finally, participants were screened to ensure that there was no history of brain injury or mental retardation.

Advanced psychology graduate students and post-baccalaureate research assistants administered all interviews. An independent rater, blind to group membership, evaluated 15 randomly selected audiotapes of SCID-I interviews to assess diagnostic interrater reliability. The selected audiotapes included both individuals who did and did not meet formal inclusion criteria. The independent rater matched the diagnostic decision made by the original interviewer ($k = 1.00$).

Participants for the present study were a subgroup of the original sample for whom there were data available on abuse history ($N = 97$; i.e., three individuals from the original sample had missing data for abuse and were excluded from the analyses). They were between the ages of 18 and 58 years ($M = 34.90$, $SD = 12.21$), and were predominantly female ($n = 73$, 75%; male $n = 24$, 25%). The majority of participants were Caucasian ($n = 49$, 50.5%), followed by Asian ($n = 40$, 41.2%), African American ($n = 4$, 4.1%), Latino or Hispanic ($n = 2$, 2.1%), and other ($n = 1$, 1%).

Marital status of the participants was mainly single ($n = 57$, 58.8%), with 32 (33.4%) married or living with a partner, and 6 (6.2%) separated, widowed, or divorced. Most individuals in the sample had pursued higher education, with 45.4% ($n = 44$) having completed college with no advanced studies, 30.9% ($n = 30$) having pursued graduate or professional education beyond college, and 14.5% ($n = 14$) having attended some college or less. Regarding annual income, 19.6% ($n = 19$) reported earning more than $75,000, 17.5% ($n = 17$) reported earning between $50,000 and $75,000, 24.7% ($n =
24) reported earning between $25,000 and $50,000, 14.4% (n = 14) reported earning between $10,000 and $25,000, and 15.5% (n = 15) reported earning under $10,000.\textsuperscript{i}

\textit{II. Measures}

\textit{History of depression.} The SCID-I interview inquired about history of depressive episodes. The number of prior depressive experiences was assessed to determine whether the participant met criteria for past episodes of MDD. Most participants had four or fewer lifetime depressive episodes; the range of total lifetime episodes was 1 (n = 18) to “too many to count” (n = 15). Due to the low number of participants in each specific number category beyond five lifetime episodes, individuals with more than five episodes were collapsed across episode history categories. In order to maintain approximately equal numbers of individuals per category, those who reported six or more lifetime episodes were collapsed into three categories: 6-10 episodes, 11-36 episodes, and >36-“too many to count.”

The SCID-I has been found to have good test-retest and interrater reliability for diagnosis major depressive disorder, with coefficients ranging from .61 to .93 (Zanarini et al., 2000 ; Skre, Onstad, Torgersen, & Kringlin, 1991). Validity of the SCID-I has not been adequately assessed due to a lack of “gold standard” measure of diagnoses with which to compare SCID-I results.

\textit{Life stress and early adversity assessment.} Life stress was operationalized and assessed using The Life Events and Difficulties Schedule (LEDS; Brown & Harris, 1978). This semistructured interview covers all domains of the respondent’s life
(e.g., work, school, family, friends, etc.), using probes to stimulate recall of past experiences, including history of early adversity. The rating assigned to each event is based on extensive information about the circumstances surrounding the event as well as on the biographic circumstances of the individual, resulting in a contextual rating of the severity of the event. Ratings were made by a panel of trained LEDS raters who determined what qualified as an event or difficulty, and then determined the severity rating of each. Each rater first provided an independent rating of the major stress dimensions, and discrepancies were then resolved through group discussion and consensus meetings.

All raters had access to the LEDS training manual, a 520-page manual with thousands of case vignettes that offer anchoring examples to assist the rater in defining events and assigning threat ratings. Raters were blind to clinical considerations (e.g., person’s subjective response to stress, clinical status) and dependent variables (e.g., timing of depression onset, lifetime history of episodes). Any “dependent” events (those that could be considered direct consequences of depression, e.g., interpersonal problems due to irritability or poor concentration) were excluded from the analyses.

Brown and Harris (1978) defined severe events as events that are rated very highly on the long-term threat scale (i.e., a 1 or 2 on the 5-point scale), and directly affect the subject (i.e., subject or joint focused). These ratings are based on the degree of contextual threat, the unpleasantness associated with the situation, and the likelihood of prolonged consequences. These events are rated both contextually
(taking into account the circumstances surrounding the event and the individual’s biographic circumstances) and consensually. Examples of severe events include major discontinuity in the subject’s career or life pattern, a very serious fight with a spouse, and unexpected death of a spouse or child.

Following the LEDS assessment, a consensus meeting was held via teleconference with trained raters at the University of Oregon, Eugene. These raters were also blind to relevant dependent measures and clinical status and performed all ratings after discussion and consensus agreement. Interviews and rating sessions each required approximately 2 hours to complete.

The LEDS is generally considered the gold standard for life stress assessment and has significant psychometric advantages over standard life stress checklists. Previous research using the LEDS has demonstrated reliability of assessment of events and difficulties for at least 2 years prior to interview, and a high reliability for severe events for up to 10 years (Brown, 1989; Neilson, Brown, & Marmot, 1989). The current project demonstrated reliability for pair-wise comparisons in the range of .72 to .79 ($M = .76$; Cohen’s k, corrected for differences in the number of raters per event; Uebersax, 1982).

**III. Analyses.** Regression analyses were run to predict personal history of depression based on the presence or absence of early adverse events and current, severe life events. We hypothesized that early abuse and recent major events would be strongly associated with a personal history of depressive episodes. Variables were
entered hierarchically, with history of early adversity followed by presence of severe stress. In the final step, the interaction (as a cross product of early adversity and severe stress) was tested. Chi-square analyses were used to determine whether there was an association between a severe event and history of early life stress in general within the depressed sample. Here we were testing the stress sensitization hypothesis, expecting to find that individuals who experienced early life stress and were currently depressed would not have an association with recent major life events. An independent samples t-test was run to determine the amount of time between the severe event and onset in the group with a history of early life stress and in the group without a history of early life stress. Here we predicted that there will be a shorter interval to depression onset in the group with a history of early adversity. Finally, regression analyses and independent samples t-tests examined the relationship between early adversity and age of onset.

Data analysis also included descriptive statistics of all study variables, reporting mean, standard deviation, and range. Bivariate correlations and chi square tests were run to determine associations between variables.

\[ n’s \text{ vary slightly due to missing data} \]
RESULTS

The sample for these analyses (N=97) included 20 individuals with a history of physical (n=10) and sexual (n=14) abuse; four individuals experienced both physical and sexual abuse. Regarding severe events, 19 individuals experienced a severe event within 12 weeks of depressive onset; 25 individuals experienced a severe event within 26 weeks of onset. Most individuals experienced recurrent depression (77 reported more than one depressive episode).

I. Preliminary analyses.

Preliminary analyses were conducted to examine associations between the major demographic and clinical variables with primary study variables. The major demographic variables including age, gender, race, and income were examined with independent samples t-tests and chi square tests and had no significant effects on history of abuse or presence of a severe life event (Table 1). However the presence of physical abuse was significantly associated with the presence of sexual abuse $X^2\ (1, N=97)=5.90, p<.01$. 
TABLE 1

MEANS, STANDARD DEVIATIONS, AND CORRELATIONS OF MAJOR DEMOGRAPHIC AND CLINICAL VARIABLES

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<th>Variable</th>
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<td>1. Any Abuse Reported</td>
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<td>4. SLE within 26 weeks of onset †</td>
<td>.11</td>
<td>.09</td>
<td>.11</td>
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<td>.25</td>
<td>.44</td>
</tr>
<tr>
<td>5. SLE within 12 weeks of onset †</td>
<td>.07</td>
<td>.02</td>
<td>.09</td>
<td>.84**</td>
<td>-</td>
<td></td>
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<td></td>
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<td>.19</td>
<td>.39</td>
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<tr>
<td>6. Age</td>
<td>- .06</td>
<td>.02</td>
<td>- .06</td>
<td>-.16</td>
<td>-.09</td>
<td>-</td>
<td></td>
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<td>34.88</td>
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<td>7. Gender</td>
<td>.17</td>
<td>.17</td>
<td>.04</td>
<td>.13</td>
<td>.06</td>
<td>-.18</td>
<td>-</td>
<td></td>
<td></td>
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<td>.44</td>
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<td>.05</td>
<td>-.04</td>
<td>-.09</td>
<td>-.14</td>
<td>.36**</td>
<td>-.12</td>
<td>-</td>
<td></td>
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<td>9. Race</td>
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<td>.09</td>
<td>.08</td>
<td>.12</td>
<td>.06</td>
<td>.11</td>
<td>.09</td>
<td>-.13</td>
<td>-</td>
<td>4.69</td>
<td>1.51</td>
</tr>
</tbody>
</table>

† Severe Life Event (SLE)

* p < .05, two-tailed

** p < .01, two-tailed

II. Severe events and depression history

Owing to the lack of standardization of knowing when events are likely to cause depression, data from events that occurred 12 weeks prior to onset are nested in the data that occurred 26 weeks prior to onset. In this section, data from events that occurred within 12 weeks and 26 weeks of onset are presented separately.
Severe events 12 weeks prior to onset. Consistent with the majority of research on stress and depression (e.g., Brown & Harris, 1989; Hammen, 2005; Monroe & Harkness, 2005), the presence of at least one severe life event within 12 weeks of onset was related to history of depressive episodes. Mann-Whitney tests were used to determine whether there was an association between severe events and history of depression. Results revealed a significant effect of severe events occurring within 12 weeks of depressive onset ($Z=2.87, p < .01$), with participants reporting a severe event having a mean rank of 32.13 versus 52.68 for those who did not report a severe event. To determine if there was a significant difference between first episodes versus all recurrences collapsed, history of depression was dichotomized then compared to the presence or absence of severe events. The Pearson chi-square was significant for severe events occurring within 12 weeks of depressive onset $X^2 (1, N=97)=8.86, p < .01$.

Severe events 26 weeks prior to onset. Mann-Whitney test results again revealed a significant association between severe events occurring within 26 weeks of depressive onset and depressive history ($Z=2.52, p < .01$), with participants reporting a severe event having a mean rank of 33.36 versus 55.03 for those who did not report a severe event. The Pearson chi-square was significant for severe events occurring within 26 weeks of the dichotomized depressive onset, $X^2 (1, N=97)=6.96, p < .01$. 


III. Severe events and abuse history

Severe events 12 weeks prior to onset. With respect to the predictions involving abuse history and major life stress, analyses showed history of abuse was unrelated to the presence or absence of a severe life event within 12 weeks of onset $X^2(1, N=97)=.47$, n.s. Breaking the abuse history variable into reported physical and sexual abuse and repeating these analyses for each variable did not yield any significant associations.

Severe events 26 weeks prior to onset. Abuse history was also found to be unrelated to the presence of a severe event occurring within 26 weeks of onset $X^2(1, N=97)=1.12$, n.s. Again, breaking the abuse history variable into reported physical and sexual abuse and repeating these analyses did not yield any significant associations.

IV. Abuse history and depression history

In terms of abuse history and history of depressive episodes, Mann-Whitney tests revealed that history of abuse was unrelated to the number of lifetime depressive episodes, $Z=-1.52$, n.s., with individuals who reported abuse having a mean rank of 56.25 verses 45.80 for individuals not reporting abuse. Breaking the abuse history variable into reported physical and sexual abuse and performing the separate analyses did not yield any significant associations. Chi-square tests indicated that abuse history was unrelated to recurrent episodes of depression, however the cell containing data
for participants with a history of abuse and only one depressive episode was smaller than the expected count ($n=3$; minimum expected $n=3.80$).

**V. Abuse history, severe events, and depression history**

*Poisson regression.* In order to best understand the nature of the relationship between early abuse, depression history, and severe life events the data must fit a regression model without violating the assumptions of the model. Linear regression imposes the strictest assumptions on the data; the proportional odds model relaxes some of these assumptions, but because the assumptions of the proportional odds model were violated with these data, the Poisson regression model (Geweke, 1994) was selected. The Poisson regression model has more relaxed assumptions than the proportional odds model, but maintains the assumption that the dependent variable (i.e., history of depressive episodes) is ordered.

The Poisson regression model predicting history of depression from history of abuse and presence or absence of a severe life event within 12 weeks of onset was significant, with a likelihood ratio $X^2 (2, N=95) =18.47, p <.001$. The predictors, history of abuse and severe event within 12 weeks of onset, were each statistically significant. The Wald chi-square estimate for presence of abuse while controlling for severe life events within 12 weeks of onset, $X^2 (1, N=95) =3.96, <.05$, suggests that history of abuse increases the log odds of having a greater history of depression. The interaction between history of abuse and severe life events occurring within 12 weeks
of onset was not significant for any type of abuse. Goodness of fit criteria did not give
evidence of extreme over-dispersion $X^2 (91, N=95)=1.39$, suggesting that this model
fits the data.

The Poisson regression model predicting history of depression from history of
abuse and presence or absence of a severe life event within 26 weeks of onset was
significant with a likelihood ratio $X^2 (2, N=95) =22.58, p <.001$. The predictors,
history of abuse and severe event within 26 weeks of onset, were each statistically
significant. The Wald chi-square estimate for presence of abuse while controlling for
severe life events within 26 weeks of onset $X^2 (1, N=95) =5.77, <.05$, again suggests
that history of abuse increases the log odds of having a greater history of depression.
The interaction between history of abuse and severe life events occurring within 26
weeks of onset was not significant for any type of abuse.

Breaking down the history of abuse variable into reported physical or sexual
abuse and rerunning these analyses yielded significant findings only for individuals
with a history of sexual abuse. The Poisson regression model predicting history of
depression from history of sexual abuse and presence or absence of a severe life event
within 26 weeks of onset was significant with a likelihood ratio $X^2 (2, N=95)
=21.02, p <.001$. The predictors, history of sexual abuse and severe event within 26
weeks of onset, were each statistically significant. The Wald chi-square estimate for
presence of sexual abuse while controlling for severe life events within 26 weeks of
onset $X^2(1, N=95) = 4.62, <.05$, suggests that a history of sexual abuse is a significant predictor of a greater history of depression.\textsuperscript{iv} The model failed to reach significance for either physical or sexual abuse when severe events occurring within 12 weeks of onset was entered into the equation.

\textit{VI. Early abuse and time to depressive onset following severe events}

An independent samples t-test compared the interval between first lifetime severe event and onset of a depressive episode in the group with a history of early abuse and in the group without a history of early abuse. Although the results did not detect a significant difference between these groups, $t(23)=1.22, p=.095$, the trend suggested that those with a history of early abuse ($M=54.29, SD=41.33$) had fewer days between the severe event and onset than those without a history of early abuse ($M=107.06, SD=109.99$). Breaking down the history of abuse variable into its components and performing these analyses did not yield significant results. There was no significant difference in time from severe event to depressive onset between individuals with a history of sexual abuse ($M=69.20, SD=17.42$) and those without a history of sexual abuse ($M=98.05, SD=24.09$), $t(23) = .58$, n.s. There was no significant difference in time from severe event to depressive onset between individuals with a history of physical abuse ($M=48.25, SD=22.93$) and those without a history of physical abuse ($M=100.67, SD=22.60$), $t(23) = .34$, n.s. Again, although
these results were not significant, the general trend indicated that both types of abuse decrease the interval between severe event and onset of a depressive episode.

VII. Age of onset

The data did not fully support the prediction that individuals with a history of abuse would have an earlier age of onset than those without a history of abuse. An independent samples t-test found a marginally significant difference between individuals with a history of abuse (\(M=17.11, SD =1.71\)) and individuals without a history of abuse (\(M=21.46, SD =1.55\)) on age of onset \((t[78]=1.87, p=.07)\). The general trend suggests that individuals with a history of abuse are likely to have an earlier age of onset than individuals without abuse history. Breaking history of abuse into reported sexual abuse and physical abuse and performing these analyses again did not yield significant findings.

VIII. Summary of results

In sum, support was found for the hypothesis that history of abuse and presence of a severe life event would predict personal history of depression in opposite directions. That is, history of abuse was found to predict a greater history of depression, whereas presence of a severe life event was found to predict a lesser history of depressive episodes. The present analyses found support for the stress sensitization hypothesis with regard to abuse; currently depressed individuals who reported abuse did not have an association with recent major life events. Some
support was also found for this hypothesis with regard to stress; severe events were observed to become progressively less associated with recurrent depressive episodes. The prediction that individuals with a history of abuse would have a shorter interval between severe event and depressive onset was not statistically supported in this sample.

\[ii\] The Mann-Whitney test is a non-parametric analog to the independent samples t-test used when the nature of the dependent variable is ordinal.

\[iii\] Linear regression was not appropriate because the nature of the dependent variable is ordinal. The proportional odds model reached significance but violated the assumptions of the model.

\[iv\] Severe life events, occurring 12 and 26 weeks before onset of a depressive episode, were found to have significant, negative Wald chi-square estimates in the Poisson regression models, again confirming the inverse relationship between severe events and history of depression.
DISCUSSION

A vast amount of research has examined the effects of stress on the development of depressive disorders, and major life stress has been consistently found to precede the onset of major depression. However, stressful life events have been found to account for only 50-80% of depressive episode onsets, which suggests that other factors are involved with determining when major stress does and does not lead to depression (Brown & Harris, 1978). The present study sought to understand other essential factors that may contribute to the onset of depressive episodes, namely physical and sexual abuse. These factors were examined by themselves with regard to depression history, as well as in association with major life events. The results of this study suggest an independent contribution of early abuse to the likelihood of experiencing multiple depressive episodes over the life course, in addition to the independent contributions previously accounted for by severe life events.

In accordance with prior research (Mazure, 1998; Post, 1992), severe life events were found to be inversely associated with prior history of depression (i.e., fewer depressive episodes) in this sample. Severe events maintained some importance for episodes subsequent to the first lifetime episode, but the contribution of these events waned for participants with a higher number of lifetime episodes. These results are consistent with the results of Kendler, Thornton and Gardner (2000), who found that the odds ratio between severe events and depressive episodes steadily decreased over recurrent episodes. It is clear that other causal factors must come to play a larger role in the etiology of recurrent episodes, which helps to explain the diminishing
contribution of severe life events. It may be important to consider the role of minor stressors in the recurrence of depression, given that minor events (i.e., daily hassles) occur more frequently than do major events. It is possible that once an individual becomes sensitized to depression (through multiple depressive episodes) that minor events become sufficient to cause onset of a depressive episode and eclipse the major event, thus making it unlikely that the individual will experience a major event in a non-depressed state (Monroe & Harkness, 2005). Another possibility is that stress and depressive onsets become progressively disassociated over the course of recurrent depressive episodes. That is, recurrent episodes of depression begin to occur autonomously, independent of the presence or absence of major events, and thus major stressors appear to decrease in etiologic importance (Post & Weiss, 1998). Clearly, these are topics that require the attention of future research to more accurately parse out the contributing factors to recurrent depression.

At a general level, early adversity has been found to be a vulnerability factor for the development of major depressive disorders (Hammen, 2000; McCauley et al., 1997; Mullen et al., 1996). The current state of research on this topic has left considerable inconsistencies in the definition of early adversity. Some researchers have proposed that early loss of a parent is most deleterious (Brown & Harris, 1978), while others have focused on the issues of parental substance abuse or mental illness (Hammen, 2000) and physical or sexual abuse (Garnefski, van Egmond, & Straatman, 1989; Hammen, 2000). The present study focused on physical and sexual abuse as the early adversity factors that may render an individual susceptible to adulthood
depressive episodes. Upon initial analysis employing linear regression, early abuse did not appear to predict prior history of depression; further examination of the issue suggested that this may have been due to the nature of the dependent variable. Since there were relatively few participants in each specific number category beyond five lifetime episodes, individuals with more than five episodes had been collapsed across episode history categories. In order to maintain approximately equal numbers of individuals per category, those who reported 6 or more lifetime episodes were collapsed into three categories: 6-10 episodes, 11-36 episodes, and >36-“too many to count.” As a result, the dependent variable became an ordinal variable and linear regression was not the most appropriate test.

The proportional odds model, also known as ordinal logistical regression, does not assume that there are equal differences between levels of the dependent variable, making it more suitable to these ordinal data. Results from the proportional odds model found an increase in log-odds of becoming depressed for participants with a history of abuse, and a decrease in log-odds of becoming depressed for participants who reported a severe event. Model fitting values suggested that both history of abuse and severe events each account for a significant amount of variance in the history of depression variable.

Although statistical analysis using this model revealed significant results, further diagnostics also indicated that the assumption of parallel lines was violated. This assumption presupposes that the effect of the independent variable is the same for each level of the dependent variable. That is, this model assumes that the effect of
severe life events and abuse history would be the same across all levels of history of depression. However, the literature is quite clear with regard to life events, that this indeed is not the case; the kindling model (Post, 1992), which states that progressively lower levels of stress are needed to precipitate affective disorders over repeated episodes, has been widely accepted by researchers (Kendler et al., 2000; Monroe et al., 2007). Thus, the violation of assumptions in the ordered logistical regression is not surprising, but does suggest that there may be better ways to portray these data.

In order to ensure that the data are most suitably analyzed, the mixed logit model, was fitted to these data (also known as Poisson regression). Results indicated that this model provided the best fit and did not violate the assumptions of the model (which are more relaxed than the proportional odds model but not as lenient as the multinomial odds model). As originally hypothesized, a history of early adversity was found to predict a greater personal history of depression using the Poisson regression model; history of abuse significantly increased the log odds of having a history of more depressive episodes. Additionally, when abuse was broken down into physical and sexual abuse and analyzed separately, history of sexual abuse was found to increase the log-odds of having a greater history of depression, whereas history of physical abuse was not significant. These results point to early abuse as the more potent sensitizing factor for major depression and suggest that sexual abuse, specifically, may be an important contributor to the etiology of recurrent depression. These results are in accord with much of the current literature on childhood abuse,
which suggests that sexual abuse is the most deleterious to the individual (Bifulco et al., 2002; Gunnar & Quevedo, 2007; Gold, 1986). Using the Poisson regression model, it was also found as hypothesized that severe events, at both 12 and 26 weeks before onset, were associated with a decrease in the log-odds of having a greater history of depression, again indicating an inverse association of severe events with history of depression.

Individuals who experienced early adversity also were hypothesized to experience a depressive episode more quickly following a severe life event than those who did not experience early adversity. Support for this prediction was mixed; although the results were only marginally significant ($p=.95$), the general trend indicated that individuals with early adversity became depressed in nearly half as many days ($M=54.29$) following a severe event compared with those without a history of early adversity ($M=107.06$). When abuse was broken down, the results were not significant, but maintained the same general trend. That is, individuals who reported sexual abuse ($M=69.20$) had fewer days between severe event and onset than individuals who did not report sexual abuse ($M=98.05$). The same was true individuals who reported physical abuse ($M=48.25$) and those who did not report physical abuse ($M=100.67$). In this instance, with regard to timing of onset, both forms of abuse appeared to be approximately comparable in their effects. Again, if the distribution of individuals with and without a history of early adversity was more evenly distributed, the test may have had sufficient power to detect this difference.
Chi-square tests of association indicated that early adversity and current, severe events were unrelated in this sample. This means that support for the stress sensitization hypothesis was found independently with regard to history of abuse, since individuals who experienced early abuse and were currently depressed did not reveal any systematic association with recent, severe events. If an individual is currently depressed and has not experienced a recent severe event, there must be some other operative variable accounting for onset. Within a depressed sample, one hypothesis is that these major alternative risk indices are systematically associated with one another. That they were not suggests that they have, at least in part, independent associations with the person’s history of depression.

Of course, there still could be a higher order interaction between abuse and stress (e.g., they work synergistically, for example, with abuse and the lack of major stress being especially predictive of many lifetime episodes). The interaction between history of abuse and presence of severe life events, however, did not predict prior history of depression. That is, the combination of early abuse and current severe events was expected to result in fewer lifetime depressive episodes, but was not supported in this sample. This suggests that the two main effects of abuse history and presence of a severe event were not conditional on one another in relation to depression history.

Regarding the hypothesis that individuals who reported a history of abuse would have an earlier age of onset, an independent samples t-test revealed marginally significant results, which indicated a tendency for individuals with a history of abuse.
to experience earlier age of onset. The difference in these groups was nearly four years, finding that individuals who reported abuse experienced onset in the late teens whereas individuals who did not report abuse experienced onset in the early twenties. The breakdown analyses analysis did not find significant individual effects for physical or sexual abuse on age of onset, suggesting with regard to this clinical variable there may be more than one form of abuse that is relevant.

The major demographic variables (age, sex, race, and income) were not found to be associated with the primary clinical variables (history of abuse, history of depression, presence/absence of a severe life event). This lack of correlation suggests that the results obtained for the primary analyses are likely to be attributed to the independent variables and not to shared variance with demographic characteristics of the participants. The clinical variable of abuse history was found to be associated with its components in the expected relationship. That is, reported history of sexual abuse was significantly correlated with reported history of physical abuse (i.e. repeat victimization; Miller, 2006).

There were several strengths of this study. First, the Life Events and Difficulties Schedule (LEDS; Brown & Harris, 1978) was used to assess history of life events, and is considered the “gold standard” in the field of stress assessment. The LEDS is unique in that it considers personal as well as situational variables to generate a measure of contextual threat, and it is the rater, not the respondent, who defines the event ranking (Dohrenwend, 2006). In fact, intrapair agreement has been found to be as high as 91% for severe events in depressed samples (Brown, Sklair,
Harris, & Birley, 1973). Second, the clinical sample contained individuals with a wide range of reported past depressive episodes, making it ideal for studying the effects of the primary variables (i.e., abuse history and severe events) on history of depression. Additionally, most of the participants were not experiencing symptoms during the study period and were able to report a clear onset of depressive episodes, ensuring that participants did not suffer from chronic depression.

It is important to note, too, that the present study had some limitations. As previously mentioned, low numbers of participants in some groups greatly affected the power of the statistical tests, making it difficult to detect relationships between some variables that may have been more apparent in a larger sample size. More significant results may have been found, too, if the sample contained a higher proportion of individuals who had experienced early adversity. The possible underreporting of abuse and lack of strict definition for abuse (e.g., hitting versus slapping versus spanking) should also be taken into account. Finally, more intensive and detailed interview-based procedures to document past history of abuse may be of value to better capture the information, particularly for individuals who report very large numbers of past episodes.

These results offer statistical as well as descriptive information regarding the relationship between early abuse, severe events, and history of depression. The results suggest that early abuse, particularly sexual abuse, plays a role in the lifetime course of recurrent depression, forecasting a higher likelihood of experiencing repeated episodes. Results also point to possible effects of early abuse on age of onset and the
interval between a severe event and onset of a depressive episode. Future research can examine the extent of this contribution by increasing the number of participants who have experienced early physical and sexual abuse, thereby providing a more even distribution of the target variable. This might be achieved by targeting samples that may have a higher rate of likely abuse (e.g., samples with low SES, social isolation, drug and alcohol use) may increase the number of participants reporting abuse. Finally, it may also be informative to focus on sensitization factors for first episodes and examine the contribution of lower level events in precipitating onset for individuals who report early abuse.
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