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The role of chronic interpersonal stress and its interactions with episodic stressors and gender upon depression and anxiety in adolescents

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ABSTRACT

The role of chronic interpersonal stress and its interactions with episodic stressors and gender upon depression and anxiety in adolescents

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Although chronic stress has been shown to be significantly associated with depression, this relationship has not received adequate attention, particularly in adolescent samples. One gap lies in the examination of whether particular domains of chronic interpersonal stress are uniquely related to risk for depression. Furthermore, the degree to which chronic interpersonal stress is associated with increased risk for anxiety outcomes is largely unknown. The present study had four objectives. First, the project examined the associations between particular domains of chronic interpersonal stress (e.g., close friendships, social group, romantic relationships, and family relationships) with depression and anxiety outcomes. Second, this study examined whether positive relationships in one interpersonal domain buffered against the deleterious effects of negative relationships in another domain. Third, this study explored whether positive peer or family relationships buffered the negative impact of episodic stressors. Fourth, this project examined whether gender moderated the relationships between stress and depression and anxiety. A sample of 486 adolescents completed a life stress interview, psychiatric diagnostic assessment, and symptom questionnaires at two assessments approximately 1 year apart. Higher levels of chronic stress in close friendships, social group, and family relationships all prospectively predicted major depressive episodes during follow-up. Importantly, only social group was a significant predictor beyond baseline symptoms and the other domains of chronic

interpersonal stress. Lower quality of aggregated peer relationships prospectively predicted anxiety disorder onset, although no individual domain of chronic interpersonal stress was a significant predictor. In prospective analyses of depression and anxiety symptoms, social group contributed small, but significant variance. Cross-sectional analyses of depressive symptoms revealed that family relationships were a significant unique predictor of symptoms at follow-up beyond baseline symptom levels and other chronic stress domains. Interactions between chronic stress domains as well as between chronic and episodic stress yielded minimal evidence of buffering. Finally, although women were more likely to experience major depression and anxiety during follow-up, no significant gender differences in reactivity to stress were found. Methodological limitations that may have contributed to many of these null findings are discussed. Future studies should incorporate chronic stress into models of risk for psychopathology in adolescence.

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INTRODUCTION

Several reviews of the life stress literature over the past 20 years have noted the importance of exploring the relationship between chronic stress and depression (Coyne & Downey, 1991; Hammen, 2005; Mazure, 1998). Notwithstanding, relatively few studies have directly examined this relationship. The assessment of ongoing circumstances, particularly within interpersonal domains, would seem to be especially important during adolescence, a period marked by increased relationship intensity, initiation of dating, and more conflicts with parents (e.g., Arnett, 1999; LaGreca & Harrison, 2005). Indeed, some 20 years ago Compas (1987) noted "[i]t appears that chronic stressors, including daily hassles and characteristics of the social environment, hold greater promise than major life events in understanding the development of psychological distress during adolescence" (p. 293).

As reviewed below, chronic stress and its synonyms (chronic strain, ongoing stressors, difficulties) have been operationalized in various ways both in terms of their temporal parameters as well as in the domains that were examined as sources of chronic stress. One important distinction across studies is that chronic stress can be meaningfully differentiated from episodic stress. The former represents ongoing, temporally extended conditions, whereas the latter has a clear onset and offset and generally takes place over a short period of time, although the effects of an episodic event can be long lasting (e.g., death of a friend, loss of a job).

Only a small body of work has investigated the role of chronic stress in predicting the occurrence of major depressive disorder (MDD) in adolescent samples (e.g., Daley, Hammen & Rao, 2000; Eberhart & Hammen, 2006). There is a need to extend that preliminary work as well

as to examine whether particular domains of chronic stress are uniquely associated with depression. That is, it may be the case that specific domains of functioning (e.g., family, social group) are particularly influential on adolescents' mental health. A related area requiring further attention is exploration of the nature of interactions among different domains of chronic interpersonal stress. Of particular import is refining our understanding of whether positive relationships in one domain of interpersonal functioning buffer the effects of negative relationships in another domain (Brendgen, Wanner, Morin, & Vitaro, 2005). Utilizing a more nuanced approach will help establish whether predictive validity of chronic stress can be improved by examining specific types of relationships instead of aggregating across domains. Furthermore, if specific types of interpersonal relationships were uniquely associated with risk for depression, such findings could help guide prevention and intervention programs.

The relative lack of attention to chronic stress in studies of depression has also precluded clarification of the nature of interactions between chronic stress and episodic stress (Hammen, 2005). Acute stressors have been repeatedly demonstrated to predict depressive symptoms over time in adolescent samples (Grant, Compas, Thurm, McMahon, & Gipson, 2004). Further exploration of the interaction between chronic and episodic stress will help clarify whether chronic conditions moderate the relationship between episodic events and depression. In particular, it has not been firmly established whether positive functioning in family or peer domains may protect against the negative effects of stressful interpersonal life events.

Finally, it has been well established that there are substantial gender differences in rates of both depressive symptoms and unipolar mood disorders, beginning in adolescence (e.g., Ge et al., 1994; Hankin et al., 1998; Nolen-Hoeksema & Girgus, 1994). A strong research base has

also demonstrated that males and females may differ on certain psychosocial variables or sets of variables that are relevant to risk for depression (Hankin & Abramson, 2001). For these stated reasons as well as ones that are reviewed in more detail below, it is important to examine whether gender moderates the relationships between life stress variables and depression.

Comorbidity of Depression and Anxiety

Another objective of the present study was to examine whether chronic interpersonal stress was also associated with anxiety symptoms and disorders. This focus is due in large part to the considerable comorbidity and likely etiological overlap between anxiety and depression. Significant rates of comorbidity between anxiety and depression have been noted in adults (e.g., Mineka, Watson, & Clark, 1998), as well as in children and adolescents (e.g., Brady & Kendall, 1992; L. Seligman & Ollendick, 1998). For example, in their sample of community adolescents, Kashani et al. (1987) reported that 75% of participants meeting criteria for a depressive disorder also met criteria for an anxiety disorder. Even among adolescents who did not meet full criteria for diagnosis, but had depressive symptoms or depressed mood only, about 20% had an anxiety disorder. In addition, large-scale studies of adolescent samples have found that a prior history of anxiety disorder significantly increased the risk for subsequent depression and a history of depression increased the risk for anxiety disorders (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Lewinsohn et al., 1993).

One reason for the overlap between depression and anxiety may derive from shared etiological factors. One prominent vulnerability factor that has been shown to relate to both anxiety and depression is neuroticism or negative affectivity (Eysenck & Eysenck, 1975; Watson & Clark, 1984). Watson and Clark (1984) described negative affectivity (N/NA) as a trait

tendency to experience negative mood states. The tripartite model of anxiety and depression suggested three factors related to depression and anxiety, one specific to depression, one specific to anxiety, and the factor of N/NA which was associated with both anxiety and depression (Clark & Watson, 1991; Clark, Watson, & Mineka, 1994; Mineka et al., 1998; Watson, Clark, & Carey, 1988). Although this model was originally formulated based on adult samples, extensive support has been found for the shared negative affect component in youth samples (e.g., Chorpita, 2002; Turner & Barrett, 2003). Given the substantial overlap between depression and anxiety, the present investigation examined whether life stress variables hypothesized to be related to depression were also related to anxiety outcomes.

The Present Study

Thus, the present study had several goals: (1) examine the associations of specific domains of chronic interpersonal stress with depression and anxiety outcomes and test whether any particular domain is uniquely related to these outcomes; (2) examine the interaction between peer and family chronic stress in predicting anxiety and depression; (3) explore whether episodic and chronic stress interact to predict internalizing outcomes; and (4) identify whether any of these main effects and interaction effects are significantly moderated by gender.

These issues were addressed within the context of a larger 8-year longitudinal study of adolescents at risk for mood and anxiety disorders. The present study focused only on the baseline and 1-year follow-up assessments. Although a substantial number of vulnerability measures were included in the larger study, the present investigation focused on the important role of life stress alone. It was hoped that by carefully examining life stress, the present study

would help future research focus on the most crucial aspects of life stress that are relevant to adolescents' vulnerability to emotional symptoms and disorders.

Defining Chronic Stress

As noted by Hammen (2005), chronic stress has been defined in many different ways across studies. For example, some studies of chronic stress have explored specific populations undergoing particular forms of chronic stress, such as adult caregivers of spouses suffering from dementia (Dura, Stukenberg, & Kiecolt-Glaser, 1990), or mothers and siblings of disabled children (Breslau & Davis, 1986; Breslau & Prabucki, 1987). Several other studies have measured chronic difficulties or strains across a number of life domains (e.g., Brown, Bifulco, & Harris, 1987; Daley, Hammen, & Rao, 2000; Ormel & Wohlfarth, 1991). For example, in their sample of adults, Ormel and Wohlfarth (1991) examined chronic stress in interpersonal relationships, work, health, and housing domains. Using a late adolescent sample, Daley et al. (2000) examined chronic stress across close friendships, romantic relationships, family relationships, school, and work. Mazure (1998) concluded that this approach of examining chronic stress across multiple domains simultaneously was important for most accurately assessing an individual's psychosocial circumstances.

Another area of enormous variation across studies involves the time parameters for what constitutes chronic stress. George Brown and colleagues have conducted extensive work using the gold-standard Life Events and Difficulties Schedule (LEDS) and have defined "difficulties" as lasting at least 4 weeks (Brown, 1989), although "*major* difficulties" exceed 6-month or 2-year minimum durations (e.g., Brown et al., 1987; Brown & Harris, 1989). Similarly, Rojo-Moreno, Livianos-Aldana, Cervera-Martínez, Dominguez-Carabantes, and Reig-Cebrian (2003)

used the LEDS and required a major difficulty to last at least two years. Dougherty, Klein, and Davila (2004) used a different life stress interview and examined chronic stressors (e.g., interpersonal difficulties, health, financial problems) lasting at least 6 months. Using yet a different life stress interview, Ormel and Wohlfarth (1991) defined long-term difficulties as circumstances of at least 2 months duration.

Two other studies derived chronic stress from measures originally intended to assess episodic events (Avison & Turner, 1988; McGonagle & Kessler, 1990). Avison and Turner (1988) used a life events checklist that also asked participants to define the onset and offset of particular "events." The authors defined acute events as lasting 1-2 months whereas chronic strains needed to last 10-12 months (e.g., problems with children, financial worries, problems with spouse). McGonagle and Kessler (1990) defined events that began more than one year prior to the assessment interview as chronic (e.g., long lasting health, financial, or marital difficulties). Thus, different investigators of adult samples have defined the minimum duration for chronic stress very differently, ranging from 2 months to 2 years, making direct comparisons across studies quite difficult.

Studies in youth samples have also varied in their definitions of chronic stress. For example, in one study of about 260 adolescents, Timko, Moos, and Michelson (1993) used the Life Stressors and Social Resources Inventory-Youth Form (LISRES-Y; Moos & Moos, 1992) to explore five areas of interpersonal stressors between the participant and his or her mother, father, friends, sibling, and school (peer and teacher). The LISRES-Y does not require a minimum duration for chronic stress. Rather it asks the participant to indicate from *never* to *often* how frequently certain behaviors (e.g., arguments, the person is critical of you) occur. By contrast,

Williamson et al. (1998) used the LEDS in their study of depressed (n = 26) and control (n = 15) adolescents aged 13-18, and so major difficulties required at least 2 years duration. Finally, Towbes and Cohen (1996) developed a 54-item checklist–the College Chronic Life Stress Survey–and asked students to check off items that made them "feel stressed, upset, or worried, at least two to three times a week for the past one month" (p. 204).

Hammen and colleagues have conducted several studies of youth using an interview-based measure of chronic stress (e.g., Daley et al., 2000; Eberhart & Hammen, 2006; Shih, Eberhart, Hammen, & Brennan, 2006). The Life Stress Interview (LSI; Hammen, 2002) covers the previous 6-12 months and assesses quality of functioning across a number of domains (e.g., social group, family relationships, school, work, etc.). Importantly, the person's circumstances in any domain may rate anywhere between exceptionally good to severely adverse (Hammen, 2002). Thus, the top end of the scale represents positive functioning in that domain, rather than the absence of chronic stress. Therefore, the interview differs conceptually from measures that assess for the absence or presence (and severity) of chronic stress.

The present project utilized Hammen's (2002) LSI and focused on interpersonal stressors. There are several implications to the use of this interview. First, chronic interpersonal stress was operationalized as the quality of functioning in four domains covered by the interview: close friendships, social group, romantic relationships, and family relationships, over the course of the year prior to assessment. Second, as addressed more fully below, because ratings in each domain range from very favorable circumstances to highly adverse circumstances, it was possible to examine the interaction of positive circumstances in one interpersonal sphere with negative circumstances in another sphere. Importantly, some of the aspects of this measure of

"chronic stress" overlap with aspects of other measures examining social networks, peer support, and family relationships. It may be that be that these two somewhat different approaches, one focused on stress and the other on examining indices of support (and conflict) are meaningfully related. The measurement of chronic interpersonal stress in this study may be tapping the overall quality of relationships rather than solely focusing on stressful circumstances in these relationships. Thus, studies examining peer and family support/conflict are also reviewed to lend additional support to the associations found in the chronic stress literature.

Chronic Stress and Depression

Although the number of studies that have focused on chronic stress is much smaller than those examining episodic stressors, significant relationships between chronic stress and depressive outcomes have been found (e.g., Avison & Turner, 1988; McGonagle & Kessler, 1990; for reviews see Hammen, 2005; Mazure, 1998). For example, across 10 studies of community samples of adult women, a significant chronic stressor (LEDS major difficulty) was reported by 40% of individuals with a depression onset (Brown & Harris, 1989). Unfortunately, the number of studies examining this issue in adolescents is particularly small.

Some studies have examined the relationship between chronic stressors as measured by checklist and depressive symptoms. In a sample of Norwegian adolescents in 11th grade who were followed-up at 18 months, Ystgaard, Tambe, and Dalgard (1999) found that chronic stress ("adversities") during follow-up was related to a measure of anxiety and depressive symptoms at follow-up assessment. Towbes and Cohen (1996) conducted studies on two separate college samples using their chronic stress checklist. Assessing students at two time points 1 month apart, they found that after accounting for several demographic covariates and negative life

events, the relationship between Time 2 chronic stress and depressive symptoms approached significance. In a second study, Towbes and Cohen (1996) reported that chronic stress during a 1-month follow-up was significantly associated with depressive symptoms at follow-up after accounting for neuroticism. Prospective analyses regressing Time 2 depressive symptoms onto Time 1 chronic stress revealed that after entering Time 1 distress and neuroticism on an earlier step, Time 1 chronic stress approached statistical significance ($p < .10$) in predicting symptoms.

Several other studies have examined the relationship between interview-based chronic stress and depression. Williamson et al. (1998) compared the frequency of LEDS major difficulties in a 12-month period prior to onset of MDD in adolescents versus a 12 month "linked" period in control subjects (8-20 months before interview, so as to roughly equate the time frames between groups). Williamson et al. reported that major difficulties occurred in 27% of depressed participants but 0% of controls. Using Hammen's LSI to measure chronic stress, Rizzo, Daley, and Gunderson (2006) focused on the relationship between romantic stress and depressive symptoms and disorder in a sample of 10th grade women. They reported that baseline levels of chronic romantic stress prospectively predicted depressive symptoms 6-months later. In addition, Rizzo et al. (2006) examined non-romantic interpersonal chronic stress, forming a composite across close friendships, social life, and family relationships. The non-romantic chronic stress composite was also a significant prospective predictor of follow-up depressive symptoms. However, neither chronic romantic stress nor chronic non-romantic stress were significant predictors of diagnostic status over the follow-up period.

Also using the LSI, Daley et al. (2000) assessed chronic stress averaged across five domains (close friendships, romantic relationships, family relationships, work, and school) in a

sample of women followed-up annually for 5 years after high school. The chronic stress composite was significantly predictive of first onsets of depression but not recurrences over the five-year period.[1] Eberhart and Hammen (2006) followed a sample of 18-year old women for two years with LSI and diagnostic assessments conducted at baseline, 6 months, 12 months, and 24 months. They examined both baseline peer and family relationships as prospective predictors of the first onset of major depression during follow-up, as well as of depressive symptoms during the first 6 months of the follow-up period (all women with a history of depression were excluded from analyses). Of note, whereas quality of family relationships at baseline was a significant predictor of depressive symptoms and disorder, baseline peer relationships were only predictive of later symptoms.

Thus, some evidence suggests that chronic stress confers risk for later depressive symptoms and disorder. However, this research base is small: Only a few studies have taken a truly prospective approach, and many samples were wholly female. Furthermore, most studies have either examined a composite of chronic stress domains or have examined family and peer relationships separately, thus leaving questions of the relative predictive utility among domains unexamined.

Adolescents' Family and Peer Relationships and Depression

A larger body of research has examined the relationship between depression and enduring aspects of family and peer relationships such as support and conflict. Before reviewing some findings from the literature on peer and family relationships, it is important to first touch upon

[1] Daley et al. (2000) did not use a truly prospective approach. As the authors noted, although they attempted to measure chronic stress for the 3 months prior to MDD onset, the ratings of chronic stress from any particular assessment covered a one-year period. Thus, the ratings may include periods of time before, during, and after a depressive episode.

the issue of adolescent development within the family context. Clearly, adolescence involves a time of substantial psychosocial adjustments (see Buhrmester, 1990; Fuligni & Eccles, 1993, for brief discussions). In early adolescence, youth place increasing emphasis on peer relationships concomitant with decreased closeness with parents. However, as noted by Fuligni and Eccles (1993), the decrease in closeness with parents seems to be only temporary and the parent-child relationship is reformulated and renegotiated in middle and later adolescence. Further, in their review of studies of family relationships and adolescent depression, Sheeber, Hops, and Davis (2001) reported that family environment has actually been a more consistent predictor of adolescent depressive symptoms than peer relationships. Thus, we have good reason to believe that both family relationships as well as peer relationships are important factors in adolescents' adjustment.

Several studies have examined cross-sectional associations between adjustment and adolescents' peer and family relationships (e.g., Greenberger & Chen, 1996; Rubin et al., 1992). LaGreca and Harrison (2005) explored the incremental validity of several peer variables as correlates of depressive symptoms. They examined a sample of 421 high school students who completed questionnaires assessing social status (peer group affiliation), experiences of peer victimization, and quality of best friend and romantic relationships. In the full model, which included all of the peer relationship variables, experiences of relational victimization (e.g., rumor spreading, social isolation), negative aspects of the best friend relationship, and negative aspects of the romantic relationship were all unique correlates of depressive symptoms. Of note, however, is that positive aspects of best friend and romantic relationships were not significantly associated with lower levels of depressive symptoms.

However, cross-sectional designs do not rule out the possibility that youth depression produces family and peer problems. Importantly, a number of longitudinal studies have demonstrated that these psychosocial variables are predictive of depression over time. For example, Sheeber, Hops, Alpert, Davis, and Andrews (1997) examined a sample of adolescents aged 14-20 over the course of one year. The concurrent associations between depressive symptoms and family variables were substantial, and all variables were fairly stable over time. Notwithstanding, Sheeber et al. (1997) found that Time 1 family conflict and support variables were significant predictors of Time 2 depressive symptoms after accounting for Time 1 depression levels (the relationships were positive and negative, respectively). Regarding peer relationships, a methodologically strong study conducted by Nolan, Flynn, and Garber (2003) assessed a sample of 6th graders over 3 consecutive years. The child, child's mother, and child's teacher all filled out questionnaires assessing the child's experience of rejection. These scales contained items examining primarily peer rejection, but also contained items focused on rejection from teachers and parents as well. Using cross-lagged structural equation modeling, Nolan et al. (2003) reported that rejection *prospectively* predicted depressive symptoms, but that symptoms did not predict subsequent rejection.

Brendgen et al. (2005) followed a sample of adolescents at ages 11, 12, 13, and 14. The investigators first identified four profiles of symptom trajectories (e.g., consistently low, consistently moderate, consistently high, and increasing symptoms). Using a sociometric measure of popularity and self-reported parent-child relationship (both averaged across measures taken at ages 11-13), Brendgen et al. (2005) found that better relationships with parents were associated with increased likelihood of being in the low symptom group versus any of the other

three groups. Neither popularity with same nor other sex peers significantly predicted group membership in this early adolescent sample. Similarly, in a large high school sample (mean age = 16.6), Lewinsohn et al. (1994) reported that self-reported low family support and high conflict with parents were significantly related to increased risk of depressive disorder during a 1-year follow-up.

Further highlighting the importance of parent-adolescent relationships was a 7-year longitudinal study of a community sample of adolescents (mean age at Time 1 = 12.7) conducted by Rueter, Scaramella, Wallace, and Conger (1999). Using latent growth curve modeling procedures, Rueter et al. (1999) reported that increases in the levels of parent-adolescent disagreements over the first three years of the study was positively associated with increases in the levels of adolescent's internalizing symptoms during the first four years of the study. Importantly, changes in symptoms predicted the presence of mood and anxiety disorders during years 4-7 of the study, as assessed by structured clinical interview. Both the initial level of disagreements as well as changes in the amount of disagreements were indirect predictors (via increased symptoms) of internalizing disorder in late adolescence. Finally, Rueter et al. examined the direction of effects between internalizing symptoms and parent-adolescent disagreements, using cross-lagged structural equation modeling. They reported that disagreements measured at year 1 were significantly predictive of internalizing symptom levels at year 3, but that baseline symptom levels were not significantly predictive of later disagreements. Thus, whereas their overall growth curve models were largely based on symptoms and disagreements measured concurrently, these latter results are consistent with the hypothesis that interpersonal difficulties led to subsequent increases in symptoms.

In sum, substantial support from cross-sectional, longitudinal, and prospective analyses highlights the importance of both peer and family relationships in adolescent depression. These findings supplement the results of studies explicitly examining the relationship between chronic stress and depression in adolescents.

Interaction between Domains of Chronic Stress and Depression

In their discussion, Brendgen et al. (2005) highlighted the importance of examining interactions among adolescents' different social contexts. The authors hypothesized that one superior domain of functioning might compensate for deficits in another domain and protect against depressive outcomes. That type of interaction would be consistent with a *buffering* effect. Generally speaking, buffering means that a positive resource (e.g., social support) lessens or eliminates the negative consequences of a stressor (Cohen and Wills, 1985, Figure 2). Buffering is one specific example of a moderation effect where the relationship between one predictor and the outcome variable varies at different levels of a second predictor (e.g., Holmbeck, 1997). A competing model would hypothesize that the predictors have significant main effects, but do not significantly interact. For example, a good family relationship may be associated with a roughly equal decrease in depressive symptoms at different levels of peer functioning. Although this line of exploration is provocative, firm conclusions about buffering cannot yet be drawn because only a few studies have examined buffering effects among variables relevant to chronic interpersonal stress. Findings are also challenging to integrate because interactions between interpersonal domains have sometimes been found to interact with gender as well (e.g., Gore & Aseltine, 1995).

In their cross-sectional study of adolescents' relationships with their parents, Sheeber et al. (2007) examined whether parental support buffered against the detrimental effects of parental conflict. They found that the relationship between depression status (unipolar disorder, subclinical symptoms, or healthy controls) and conflict with one parent was not significantly moderated by levels of support by the same parent or by the other parent. LaGreca and Harrison (2005) examined whether the positive aspects of adolescents' best friendships or romantic relationships moderated the relationship between peer victimization and depressive symptoms in cross-sectional analyses. No support for buffering was found. A longitudinal study by Young, Berenson, Cohen, and Garcia (2005) found a significant interaction between peer and family support in predicting later depressive symptoms; however, results were not consistent with a buffering model. Specifically, higher levels of anticipated peer support were associated with increased symptoms of depression at follow-up for adolescents who had low parental support, whereas higher levels of anticipated peer support were associated with decreased symptoms for adolescents with high parental support. Diagnostic analyses also revealed a significant interaction: the level of anticipated peer support had a relatively larger impact on risk for depression when parental support was high than when it was low. One major limitation of this study was that the investigators dichotomized their anticipated peer support variable, based on a single "no" response to one of six questions assessing anticipated peer support. In sum, the interaction between chronic interpersonal stress variables has received little attention, though support has generally not been found for the buffering hypothesis. However, studies have either been cross-sectional or have other methodological limitations (e.g., operationalization of peer support) and so further study is warranted.

Chronic Stress and Anxiety

Although depression and anxiety have substantial overlap, few studies have examined the relationship between chronic (interpersonal) stress and anxiety outcomes. For example, Towbes and Cohen (1996) found that chronic stress measured across one-month follow-up was a significant correlate of anxiety symptoms at follow-up. Time 1 chronic stress approached statistical significance in prospective analyses predicting Time 2 symptoms. Using a different sample, Towbes and Cohen reported that Time 2 chronic stress was a significant correlate of Time 2 anxiety symptoms beyond the effects of Time 2 neuroticism, although prospective results were not significant.

Eley and Stevenson (2000) examined a sample of 90 same-sex MZ and DZ twin pairs ages 8-16: 61 pairs in which at least one twin had elevated depression or anxiety symptoms and 29 pairs in which neither twin had elevated symptoms. In an examination of chronic stress over the prior 12 months, elevated family and friendship problems were found in depressed probands as compared to controls, but not in anxious probands as compared to controls. Although the level of friendship problems was not significantly worse for anxious probands than controls, the effect size was .23, indicating a small effect. Two limitations of this study include its cross-sectional design, and its use of clinical cutoff scores to determine elevated symptoms, rather than analyzing continuous symptom scores or using formal diagnostic categories. Another retrospective study by Goodyer, Wright, and Altham (1990) examined a clinic-referred sample, ages 7-16, labeled as having anxiety-dominant disorders (n = 68) or depressive-dominant disorders (n = 32). As compared with controls (n =100), both anxious-dominant and depressive-dominant disordered children were more likely to report stressful life events and/or poorer

friendships prior to onset, but no differences were found between anxious versus depressed participants in terms of the presence or combination of those factors. Unfortunately, because the onset of illness across clinical cases ranged from 1-12 months before study entry, and because cases were interviewed about the period 12 months prior to disorder onset, they needed to recall information from up to 13-24 months prior to the interview.

In their cross-sectional investigation of a large sample of high school students, LaGreca and Harrison (2005) reported a number of interpersonal correlates of social anxiety symptoms. First, association with a social group, regardless of the group's status (i.e., popular/jock or burnout/alternative) was significantly associated with fewer social anxiety symptoms. Second, whereas positive aspects of adolescents' best friendships were significantly associated with fewer social anxiety symptoms, experiences of relational victimization and negative aspects of the best friendship were significantly associated with higher levels of social anxiety symptoms. Finally, Rueter et al. (1999) found a significant relationship between parent-child disagreements and later internalizing disorders, an outcome that included both depressive and anxiety disorders. The authors reported that their results were similar when examining depressive and anxiety disorders separately as well as together, although they only formally reported results for the combined group. Overall, there is some evidence supporting a connection between anxiety and chronic interpersonal stress. However, the evidence is limited and substantial methodological problems such as the use of cross-sectional designs are all too common.

Chronic Stress Summary

Substantial evidence has been reviewed linking chronic stress, in particular interpersonal chronic stress, with depressive outcomes. However, most conclusions that can be drawn from

the existing literature are at best tentative. Many studies have utilized cross-sectional designs and little attention has been paid to deconstructing peer relationships into more specific domains (e.g., best friend, romantic relationships, and social group). Few studies have examined both peer and family relationships and further exploration of the interplay between peer and family relationships is needed. Finally, very little work has examined anxiety outcomes in relation to chronic stress. The present project sought to address each of these concerns.

Episodic Stress and Depression

In contrast to the relatively limited amount of work on chronic life stress and depression, a large body of work has examined the relationship between episodic stress and depression (e.g., Hammen, 2005; Kessler, 1997; Mazure, 1998; Paykel, 2003; Tennant, 2002). However, as reviewed below, few studies have examined the relationship between life events and depression within the context of chronic interpersonal stress. A potentially fruitful line of inquiry involves examining whether interpersonal relationships moderate (i.e., buffer) the negative effects of stressful life events on depression. First, a general review of the literature focusing on the main effects of stressful life events is presented followed by discussion of studies that have examined the interaction of chronic and episodic stress.

Although less attention has been paid to studying life stress and depression in child and adolescent samples than in adults, a substantial body of work has focused on youth samples. Across broadly defined psychopathology, Grant et al. (2004) reported finding approximately 500 studies examining life events and adjustment in youth samples. Over 60 of those studies were longitudinal, in which stressors predicted symptoms at follow-up after entering initial symptom levels as a covariate. Grant et al. reported that 53 of the 60 studies, some of which examined

depressive symptoms, yielded a significant relationship between events and symptoms; the average effect size was about 4% of unique variance (Grant et al., 2004). Thus, there is a consistent association between life events and depressive symptoms; however, the overall size of this effect is small.

Supplementing the longitudinal studies involving a single follow-up assessment of life events and symptoms reviewed by Grant et al. (2004), are studies that have examined the life events-symptoms relationship over multiple time points. For example, Ge, Natsuaki, and Conger (2006) examined the trajectories of life events and depressive symptoms in a combination of two separate samples that were assessed 7-8 times from ages 11-23. Although the average number of stressful life events decreased over development, there was a significant longitudinal relationship between increases in depressive symptoms and both relationship events (e.g., having an argument with a boy/girlfriend, having a friend move away, having an argument with a friend) as well as "personal events" (e.g., being a victim of a violent crime, getting seriously ill or injured, getting fired or laid off).

Other studies have examined life events and diagnoses of depression. For example, Williamson et al. (1998) found that whereas 46% of the cases of MDD in their adolescent sample had a severe life event in the 12 months prior to onset, only 20% of controls had such an event during a comparable 12 month period. Goodyer, Herbert, Tamplin, and Altham (2000) investigated a "high-risk" sample of adolescents[2] and found that 60% of cases who developed MDD during one-year follow-up had an event of moderate to severe impact in the month before

[2] Goodyer et al. (2000) defined high risk as either having a parent with a history of psychiatric disorder or having two of the following: at least two undesirable life events of moderate or greater severity in the 12 months prior to initial assessment, baseline inter-parental discord or past dissolution, a score above the 80th percentile on a measure of emotionality, or the occurrence of two or more major exit events (deaths/separations) in the adolescent's lifetime.

onset versus 22% of control participants who had such an event in the month before follow-up interview. The types of events that were significantly associated with MDD onset during follow-up involved loss or disappointment. Finally, in their study of a sample of late adolescent women, Daley et al. (2000) reported that episodic stress was elevated in the 3 months prior to the onset of MDD as compared to a matched 3-month period for control participants.

An important study conducted by Monroe, Rohde, Seeley and Lewinsohn (1999), focused on the impact of romantic relationship break-ups in a large sample of over 1500 adolescents. Using a prospective approach, a self-reported break-up in the year prior to initial assessment significantly increased the odds for a major depressive episode (MDE) during follow-up, although the effect was only significant for first onsets of depression. Notably, this predictive relationship remained significant beyond the effects of other life events between Time 1 and Time 2. Thus, break-ups may be particularly important events for adolescents, especially as precipitants to initial onset of depression.

In sum, substantial research has documented an association between life events and increased risk for depression. Further, several studies have highlighted the importance of interpersonal and loss events (e.g., Ge et al., 2006; Goodyer et al., 2000; Monroe et al., 1999) in precipitating depressive outcomes.

Episodic Stress and Anxiety

Across all ages, relatively few studies have examined the relationship between life events and anxiety outcomes.[3] In one of the soundest investigations conducted to date, Finlay-Jones

[3] Not included in this statement are studies of the traumatic events leading to Post Traumatic Stress Disorder (PTSD) or the conditioning of specific phobias. Those areas are well researched. Rather it is the relationship between more generically defined life events (e.g., job loss, break-up of a relationship, death of a family member) and other anxiety disorders that have not been carefully explored across many studies.

and Brown (1981) examined a sample of women (ages 16-40) who met criteria for a recent onset of depression, anxiety, or both classes of disorder, and also a comparison group that included healthy women as well as those with chronic or subthreshold cases of disorder. Finlay-Jones and Brown reported that women with recent onset depression had a higher rate of loss events prior to disorder than anxiety or non-cases, whereas severe danger[4] events occurred more frequently in anxiety cases than in depression and non-cases (also Brown, 1993, for a close replication). Kendler, Hettema, Butera, Gardner, and Prescott (2003) found that loss events were related to increased risk within the month of occurrence for MDD, generalized anxiety syndrome (GAS),[5] and the combination of the two. Danger events were associated with increased risk for GAS within the same month and 3 months after the event. This line of research is geared toward establishing whether particular types of events might be more relevant to depression versus anxiety outcomes. Kendler et al. (2003) reported that four of seven studies, including their own, have demonstrated moderate specificity wherein some event types were specifically associated with disorder type and some (e.g., loss) were not.

A few cross-sectional studies have examined stress-anxiety associations in youth samples. As noted above, Goodyer et al. (1990) reported that poor friendships and life events alone or in combination were more common prior to onset in anxious-dominant youth than in control participants. In their sample of 90 twin pairs, Eley and Stevenson (2000) found that

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[4] As described by Finlay-Jones (1989), danger is contained in events that increase the likelihood of future stressors; however, this likelihood is neither improbable nor inevitable. Examples of danger events include having a biopsy on a lump in one's breast or having a parent diagnosed with cancer wherein one physician is optimistic about the effectiveness of treatment, but another doctor tells the parent to get his affairs in order (see Finlay-Jones, 1989, p. 103).

[5] Kendler et al. (2003) defined Generalized Anxiety Syndrome as at least two weeks of disturbance meeting six or more *DSM-III-R* (American Psychiatric Association, 1987) D criteria for Generalized Anxiety Disorder. *DSM-IV-TR* (American Psychiatric Association, 2000) requires a minimum disturbance of 6 months.

probands with elevated anxiety symptoms had a higher number of threat events in the prior year than did nonanxious participants, with an effect size of .29. Loss events were not significantly elevated in anxious participants versus controls, but yielded a small effect size of .21.

Two studies of college students examined the association between life events and anxiety using longitudinal designs. Monroe, Imhoff, Wise, and Harris (1983) followed college students at two time points about 6 weeks apart. The zero-order correlation between Time 1 events (totaled over the prior year) and anxiety symptoms at Time 2 was significant. However, in regression analyses in which general distress levels at Time 1 were entered, the relationship between events and anxiety symptoms was no longer significant. Further, the total number of events and the undesirability rating of events were more strongly related to depressive symptoms than to anxiety symptoms. Two limitations to the study include its timing: students' symptoms were measured under stressful circumstances (just before the period of final examinations), and its measure of psychopathology (only symptoms were measured). Hankin, Abramson, Miller, and Haeffel (2004) examined associations between life events and both anxiety symptoms and anxiety disorders. In two studies, one with a 5-week follow-up and one with a 2-year follow-up, Hankin et al. (2004) reported that life events during follow-up were significantly associated with anxiety symptoms at follow-up. However, life events during follow-up were not significant predictors of anxiety disorders in the 2-year study. Limitations to the Hankin et al. study include the checklist measure of life events and the high rates of anxiety disorder (26% incidence over 2 years) in an unselected college student sample, and so results should be viewed with some caution.

In sum, several studies have demonstrated a significant association between life events and anxiety symptoms. Unfortunately, only limited attention has been paid to adolescent samples. Further, few studies have examined life stress as a predictor of anxiety disorders.

Interaction of Chronic and Episodic Stress

Two related, but distinct, approaches have been taken to examining the interaction of chronic and episodic stress. One framework has focused on buffering and examines whether positive relationships moderate the association between stressful life events and depression. A second framework focuses on the impact of negative life events within the context of negative chronic circumstances. Within this second framework, two types of interactions have been described. A saturation (dampening) effect suggests that the effects of episodic stress are muted when overlaid on chronic stress, whereas a sensitization (exacerbation) effect predicts that the effects of episodic stress are amplified by the presence of chronic stress. Hammen (2005) summarized that support has been found for both sensitization and saturation effects. Both the buffering and sensitization/saturation approaches are described below, although the buffering framework is more germane to the goals of the present study and is discussed second.

McGonagle and Kessler (1990) conducted a large cross-sectional study of over 1700 adults that examined the effect of chronic and episodic stress occurring within the same domain (e.g., marriage, interpersonal) on depressive symptom levels. Using an interview-based assessment of life stress, the authors found that the matching of chronic and severe episodic stress in the same domain yielded a negative interaction in 7 of 10 analyses (the interaction was statistically significant in two analyses). A negative interaction signifies that the combined effects of chronic and episodic stressors on symptoms were less than those expected by an

additive model of their independent effects. The authors suggested that this negative interaction may indicate that chronic stress facilitates resiliency, and/or that chronic stress leads to quicker coping, and/or that episodic stress resolves a chronic stressor (e.g., a divorce ending a troubled marriage). The first two possibilities are consistent with the *saturation* effect. Additional support for a saturation effect was found by Cairney, Boyle, Offord, and Racine (2003). In their cross-sectional study of nearly 3,000 adult women, the authors found that self-reported episodic stressors had a larger effect on married versus single mothers in predicting depressive disorder. Given that single mothers tended to have higher levels of self-reported chronic stress than married mothers, the authors interpreted their results to suggest that the impact of episodic stress was blunted by chronic stress.

However, other studies have found support for a synergistic interaction between chronic and episodic stressors consistent with the *sensitization* effect. For example, using the LEDS interview in a sample of adult women, Brown et al. (1987) found that the risk for depressive disorder was elevated when a severe event matched an ongoing difficulty: the rate of major depression in these instances was 46%.[6] By contrast, when the domain of the difficulty and the stressful life event did not match, the risk for depression was 14%. In a study of older adults, which also used the LEDS interview, Ormel, Oldehinkel, and Brilman (2001) found that risk for depression was elevated for participants who had a chronic difficulty of moderate severity and had a stressful life event of at least mild severity, relative to what would be expected by purely additive effects (no domain matching was conducted). As noted by Hammen (2005), whether

[6] The matching of an event with an ongoing difficulty was based on whether the event could be seen as "linked" to the difficulty. For example, Brown et al. (1987) noted that if a pregnancy was rated as having severe long term threat because of housing problems, then the event would be considered "linked" to the chronic housing difficulty.

and under what circumstances the relationship between chronic and episodic stressors fits a model of saturation or sensitization requires further study.

As noted above, buffering is defined as the extent to which positive relationship qualities reduce or eliminate the negative effects of life stress. A major difference between the two lines of research, buffering versus saturation/sensitization, is that the former is focused on moderation of one risk variable (stress) by one protective attribute (e.g., support), whereas the latter is focused on the interaction of two risk variables. Given that Hammen's (2002) LSI ranges from negative circumstances to positive circumstances, the present study has the potential to examine buffering effects.

Several studies have examined the buffering hypothesis using depressive symptoms as the outcome variable. Notably, buffering effects of positive ongoing circumstances on acute stressors have been frequently reported to be moderated by gender. For example, in their cross-sectional study of high school students, Rubin et al. (1992) found that whereas family support buffered girls from episodic stressors, good peer relationships buffered boys from episodic life stress. In two longitudinal studies examining high school samples, both Gore and Aseltine (1995) and Ystgaard et al. (1999) reported that only boys were buffered by support measures. In the former study, the effects of personal events on symptoms of general distress were buffered by family support, whereas in the latter study, the effects of total life events on depressive symptoms were buffered by peer relationships. Finally, in their multiwave, growth curve analysis of life events and depressive symptoms during adolescence, Ge et al. (1994) reported that maternal warmth and support significantly moderated the relationship between life events and depression for girls but not for boys. Specifically, whereas the initial levels of stressful life

events were significantly associated with the trajectory of depressive symptoms over time for girls whose mothers were low on warmth and support, neither the initial levels nor the change in levels of life events over time were significantly associated with depressive symptoms in girls whose mothers were high on warmth and support.

Although some studies have found support for stress buffering, Burton, Stice, and Seeley (2004) examined this hypothesis in a large sample of adolescent women over 2-year follow-up and concluded that the vast majority of *prospective* analyses, including their own, have yielded non-significant findings. The study by Burton et al. (2004) had some limitations: exclusive reliance on self-report of life events and support, and an all female sample. It also had several strengths: reliable measures of parent and peer support, symptoms and diagnoses of depression, prospective methodology, and adequate power to detect interaction effects. Burton et al. (2004) noted that most support for stress-buffering has been found in cross-sectional studies.

Related to the critiques raised by Burton et al. (2004), it is important to note that both prospective and cross-sectional analytical approaches have merit. Prospective analyses clearly demonstrate that the predictor precedes the outcome because it is measured at an earlier assessment. Cross-sectional studies, as well as longitudinal studies that measure stress concurrently with the dependent variable (and are thus cross-sectional) have the advantage of more closely approximating the level of support during the development of symptoms or disorder. However, because there is no way to determine the temporal precedence of the predictor when it is measured at the same time as the outcome, reciprocal or reverse causation cannot be ruled out (e.g., Singer & Willett, 2003).

In summary, the nature of the interplay between chronic and acute stressors remains unclear, and scant information is available on this interaction in adolescent samples. Some studies focusing on buffering have found that favorable circumstances can protect against the negative effects of life events. However, prospective studies have generally not found support for the buffering hypothesis. Finally, virtually no attention has been focused on examining these issues with anxiety outcomes.

Chronic Stress, Episodic Stress, and Recurrence of Depression

One additional consideration when examining the relationship between life stress variables and depression is that the nature of these relationships may differ between first onsets and recurrences of depression, although the reasons for these changes are not entirely clear (Monroe & Harkness, 2005). Post (1992) suggested that initial episodes of depression are more likely to be associated with severe life events than later episodes. This "kindling" theory hypothesizes that stressful events and particularly prior depressive episodes alter the body's biochemistry and thereby confer increased vulnerability to subsequent episodes. These subsequent episodes may require lower intensity stressors or possibly no stress to trigger them (Monroe & Harkness, 2005). Studies have shown that later episodes of depression are less likely to be preceded by major life events than are first onsets of depression (e.g., Kendler, Thornton, & Gardner, 2000). For example, in their large adolescent sample, Lewinsohn, Allen, Seeley, and Gotlib (1999) reported that number of stressful life events was a significant predictor of first onsets of MDD but not recurrences. Similarly, Monroe et al. (1999) reported that romantic break-ups were a significant predictor of first onsets but not recurrences of MDD.

Using a relatively novel approach, Monroe, Slavich, Torres, and Gotlib (2007) examined the relationship between severe life events, LEDS major difficulties, and the number of lifetime episodes of depression in an adult sample. They found that whereas severe events were negatively associated with the number of lifetime episodes, the presence of a major difficulty was positively associated with the number of lifetime episodes. Using Hammen's LSI, Daley et al. (2000) also examined chronic stress, stressful life events, and depressive disorder, though they dichotomized depression history. They reported that life events were a significant predictor of both first onsets and recurrences of depression, although chronic stress was significantly predictive of first onsets only. Monroe et al. (2007) suggested that methodological differences between the studies may partially account for differences in findings, including Monroe et al.'s use of an adult sample of males and females, versus Daley et al.'s use of a late adolescent female sample. In sum, there is sound theoretical and empirical evidence to examine whether depression history moderates the relationship between life stress variables (chronic or episodic) and MDD.

Relationship between Gender, Life Stress, and Depression

As noted above, rates of depression are significantly higher in women than men, a difference that begins in early-mid adolescence (Hankin et al., 1998; Nolen-Hoeksema & Girgus, 1994). Putative risk factors contributing to this difference have been examined in a variety of domains: genetic, hormonal, personality, social role, coping style, and life stress (Hankin & Abramson, 2001; Kuehner, 2003). Studies focusing on gender differences in life stress have tended to examine one of two frameworks: stress exposure and stress reactivity (Rudolph, 2002).

The stress exposure model is a mediational model which hypothesizes that the gender difference in depression may be partially accounted for due to women's increased exposure to life stress, usually operationalized as life events (Hankin & Abramson, 2001; Hankin, Mermelstein, & Roesch, 2007). The stress reactivity model is a moderational model that hypothesizes that women are more likely than men to experience depression when confronted with a particular life stressor (Rudolph, 2002). As noted by Hankin et al. (2007), these models are not mutually exclusive. The present study focused on the stress-reactivity model to examine whether gender moderated the association between life stress (chronic and episodic) and depression (or anxiety). The reactivity model is of greater interest here because the present investigation was not focused on attempting to account for gender differences in depression, but rather to examine whether particular types of life stress were more detrimental to women or men.

Several theoretical and empirical accounts suggest that men and women may be differentially affected by disruptions in particular types of relationships. Using a developmental framework, Maccoby (1990) cited how childhood play for girls typically involves close, intimate friendships based on trust, whereas boys tend to have larger groups and a more controlling, competitive interactive style. As children progress into adolescence, these different styles continue, women being more enabling and supporting of intimate relationships, and men being more likely to develop groups based on roles and dominance (Maccoby, 1990). Gardner and colleagues have suggested that while both men and women have belongingness needs, those needs may be met in different ways (e.g., Gabriel & Gardner, 1999; Gardner & Gabriel, 2004). Across five studies examining emotional, behavioral, and cognitive outcomes, Gabriel and Gardner (1999) found that women were more focused on intimate relationships, whereas men

had a stronger focus on collective (group) membership. Importantly, this line of research suggests that a gender difference lies not in the importance of connectedness, but on the *focus* of that connectedness.

Taylor et al. (2000) offered an evolutionary account for gender differences in stress response. Incorporating across animal and human studies, they suggested that whereas *fight-or-flight* is the conventionally expected reaction to stress, females tend to display a different response, labeled "tend and befriend." Taylor et al. suggested that this alternative response promotes the protection and care of offspring, and facilitates building of social groups which may ward off danger. Importantly, they noted that women were more likely than men to seek out support when stressed, particularly from other women, and that support was more beneficial when it was provided by a woman. Relatedly, Cyranowski, Frank, Young, and Shear (2000) highlighted the importance of affiliative needs in women. Affiliative needs are believed to strengthen during adolescence and refer to a desire for "close emotional communication, intimacy, and responsiveness within interpersonal relationships" (Cyranowski et al., 2000, p.22). All together these theoretical accounts suggest that women are particularly attuned to and affected by intimate, dyadic relationships, whereas men are more focused on larger group membership.

A few cross-sectional studies have explicitly explored gender differences in reactivity to chronic stress. In a cross-sectional investigation of a large Australian sample of 15 year-olds, Shih et al. (2006) examined the interaction between gender and the four domains of chronic interpersonal stress also used in the present study. They found that social group significantly

interacted with gender in the prediction of depressive disorder.[7] Whereas girls had relatively high and stable rates of depressive disorder regardless of social group quality, boys with poorer social group functioning had substantially higher rates of depressive disorder than boys with good social relationships. Rudolph (2002) examined the relationship between peer stress and symptoms of depression and anxiety in a sample of 460 5th-8th graders. The stress measure used in the study (Connor-Smith, Compas, Wadsworth, Thomsen, & Saltzman, 2000) appeared to tap aspects of both episodic stress (e.g., asking someone out and being turned down, having someone stop being your friend) and chronic stress (e.g., being teased or hassled by other kids, not having as many friends as you want). Across four regressions, gender interacted significantly with close friendships and peer stress in the prediction of depression and anxiety symptoms. In all analyses, gender differences in symptoms were small at low stress levels, but girls had notably higher levels of symptoms under conditions of high relationship stress.

Related studies have focused on gender difference in reactivity to the quality of family and peer relationships. Sheeber et al. (2001) concluded in their review that although there has been much suggestion that girls are more affected than boys by their family environments, empirical support for this contention has not been consistent. For example, Sheeber et al. (1997) found that baseline levels of family conflict and support were prospectively related to depressive symptoms at follow-up, but this relationship did not differ by gender (see also Sheeber et al., 2007). Ystgaard et al. (1999) found that Time 2 family support buffered the effects of Time 2 overall adversities for boys only. In a sample of approximately 1000 adolescents followed up

[7] Notably, Shih et al. (2006) used a liberal approach to defining disorder. Participants who had clinical or subclinical major depressive disorder or dysthymia were included in analyses. The authors reported that additional analyses demonstrated that findings were similar using only clinically significant depression, and that those effects were generally somewhat larger than for the subclinical participants.

after 1-year, Gore and Aseltine (1995) reported that the negative impact of Time 2 friendship problems (rejection by or loss of a friend during the year)[8] on depressive symptoms were buffered by both Time 2 friend and family support for boys only. Although these results are intriguing, further research is needed to clarify the impact of gender on the relationship between depression and chronic stress.

A relatively larger body of work has examined gender differences in reactivity to episodic stressors and findings have also been inconsistent (Hankin et al., 2007; Rudolph, 2002). For example, a longitudinal study by Ge et al. (1994) found that over the course of 4 years, the trajectories of girls' depressive symptoms were affected by changes in the number of stressful life events, whereas boys' symptoms were not significantly related to changes in life event levels. In contrast, Monroe et al. (1999) reported that there was not a significant interaction between romantic break-up and gender in the prospective prediction of MDD during one-year follow-up.[9]

Two other studies have examined gender differences in reactivity to specific types of episodic stress. In their cross-sectional study, Shih et al. (2006) reported a significant interaction between gender and interpersonal stress in which boys were largely unaffected by high levels of interpersonal episodic stress whereas girls who had high levels of interpersonal episodic stress had higher rates of depressive disorder than girls with low levels of interpersonal episodic stress. The interaction between gender and non-interpersonal episodic stress was not statistically significant. Hankin et al. (2007) assessed stressors and depressive symptoms at baseline, 6-

[8] It would also be reasonable to view these difficulties in peer functioning as more episodic than chronic. However, given their assessment of events, it is not clear whether the rejection or loss of a friend was acute or more gradual.
[9] Monroe et al. (1999) used a forward entry procedure for the interaction terms in their logistic regression. Whereas the interaction of break-up and gender did not meet criteria for entry, the coefficient for this particular interaction term was not reported.

month, and 12-month follow-up in a large sample of 538 adolescents. At each of the three time points, they examined stressors across several domains (e.g., peer, family, romantic, school, and athletic) using a relatively novel approach in which participants reported the most negative event of the day for 7 days using a diary method. The events were then rated for long-term contextual threat by independent raters to ensure that the events were are least "minimally threatening." Women were more reactive than men to overall stressors, achievement stressors, peer stressors, and independent (fateful) stressors. The interaction terms for interpersonal stressors and dependent stressors (events the participant played some role in creating) with gender approached significance ($p < .10$). No significant differences in reactivity were found for romantic, family, school, or athletic stressors. Although this study had several methodological strengths, one limitation was that the assessment of life events tended to elicit daily hassles rather than major life events.

Stroud, Salovey, and Epel (2002) examined gender differences in stress reactivity by assessing cortisol responses to social versus achievement stressors in a sample of healthy volunteers ranging from ages 17-23. Laboratory stressors included a social rejection challenge involving exclusion of the participant by two confederates, as well as two achievement stressors: one mathematical challenge, requiring the participant to solve very difficult problems using a novel numbering system, and one verbal challenge, requiring memorization and recitation of a passage. Although men and women did not differ in self-reported negative or positive affect following the challenges, men had a relatively stronger cortisol response during and after the achievement stressor whereas women demonstrated higher cortisol levels after the interpersonal stressors. Although this study was conducted with healthy adults using a laboratory paradigm,

the results are suggestive of gender differences in hormonal responses to different types of stressors.

In sum, findings have generally been suggestive of gender differences in the impact of chronic and episodic stressors on depressive outcomes. With regard to episodic stressors, it appears that women may be more reactive than men to interpersonal events. Studies examining gender differences in reactivity to chronic stress are more limited, but suggest that men may be more impacted by disruptions in social group functioning. Given the limited research base, it is difficult to draw any clear conclusions regarding gender as a moderator of the interaction between domains of chronic stress. Finally, gender differences in stress reactivity have scarcely been examined in the prediction of anxiety outcomes.

The Current Study

There were several aims of the current project:

(1) The present project examined the relationship between four domains of chronic interpersonal stress and anxiety and depressive outcomes at the symptom and disorder levels. Several studies have found that family and peer variables are significantly associated with depression (e.g., Brendgen et al., 2005; Eberhart & Hammen, 2006; Nolan et al., 2003, Rizzo et al., 2006; Rueter et al., 1999). Thus, it was predicted that quality of both peer and family relationships would have significant associations with both depressive symptoms and disorder. Although the research base is smaller, positive relationships have been found between family and peer variables and anxiety (LaGreca & Harrison, 2005; Rueter et al., 1999). It was predicted that each of the four domains would be significantly predictive of anxiety outcomes.

Little research has directly addressed the predictive utility of different interpersonal relationships (cf. LaGreca & Harrison, 2005). No firm predictions were made for these analyses, which were considered exploratory. To the degree that chronic interpersonal stress is significantly related to depression and anxiety outcomes, the predictive utility analyses may find support for one of two models. A unique effects model would demonstrate that one or more domains independently contribute to the prediction of depression or anxiety. A shared effects model would demonstrate that the shared variance among chronic stress domains is of primary importance and would suggest that aggregating across domains is a preferable approach to modeling the effects of chronic stress on internalizing disorders.

(2) The interaction among domains of chronic stress was explored to test for buffering. One advantage to the current methodology is that different types of peer relationships were assessed separately. Thus, the close friendships, social life, and romantic relationship domains could be analyzed individually, separated into dyadic versus group variables, or aggregated into a single construct. If support was found for the buffering hypothesis, then one domain of good functioning would moderate the relationship between outcome and the other chronic stress variable. Alternatively, support may be found for a main effects model, in which high functioning in one domain would be associated with better outcome overall, but high functioning would not moderate the relationship between chronic stress in another domain and outcome.

(3) Buffering was also examined within the context of the interaction between episodic and chronic stress. The current project focused exclusively on interpersonal events because several studies have highlighted the importance of interpersonal and loss events on depression outcomes (e.g., Ge et al., 2006; Goodyer et al., 2000; Monroe et al., 1999). The present project

examined whether and under what circumstances family and peer relationships buffered life events (e.g., Gore & Aseltine, 1995; McGonagle & Kessler, 1990). Specifically, within-domain interactions (e.g., peer stressors X peer relationships) and cross-domain interactions (e.g., peer stressors X family relationships) were explored. Few studies have carefully examined these relationships. Interestingly, although Gore and Aseltine (1995) speculated that cross-domain "matching" would not be demonstrated, they found that boys were significantly buffered from friendship rejection/losing a friend by both peer and family support. If episodic events are buffered by ongoing interpersonal circumstances, we would expect less impact of episodic stressors on psychopathology for an adolescent with good interpersonal relationships than for an adolescent with stressful relationships. A competing main effects model predicts significant main effects for interpersonal life events and peer/family relationships, but no significant interaction. Empirical study in this area has been too limited to make firm predictions.

(4) A substantial body of theoretical and empirical work has suggested that gender may moderate the relationship between stress and depression. In particular, there is some indication that boys may be particularly affected by the quality of their larger social group functioning (e.g., Gabriel & Gardner, 1999; Shih et al., 2006), whereas women may be significantly more oriented towards and impacted by intimate relationships (e.g., Cyranowski et al., 2000). Although prior empirical research has not provided uniform support for gender differences in reactivity to chronic stress, it was predicted that women would be more reactive to chronic stress in romantic and close friendship relationships, whereas men would be more reactive to chronic stress in their social group (Rudolph, 2002; Shih et al., 2006). Theoretical accounts are not as clearly informative for making predictions regarding gender differences in reactivity to chronic family

stress. Furthermore, there is limited empirical support for gender differences in reactivity to family stress (e.g., Sheeber et al., 1997, 2007). Thus, those analyses were considered exploratory.

Finally, it was predicted that gender would significantly moderate the interaction between chronic stress domains. Whereas women may be more likely to seek out and use support to help cope with stress (Taylor et al. 2000), they were expected to be more likely to benefit from the buffering effects of good close relationships. Given that men tend to focus more on group membership (Gabriel & Gardner, 1999), they were expected to be more likely to benefit from the buffering effects of good social group relationships.

METHOD

Participants

The data analyzed in this project have been collected as part of a two-site, 8-year longitudinal study. At the time of study recruitment, all participants were in their junior year at two large public high schools, one in suburban Chicago and one in suburban Los Angeles. Recruitment was conducted in the falls of 2002, 2003, and 2004. After receiving parental consent and student assent, participants filled out a 23-item screening questionnaire: the Neuroticism subscale of the Eysenck Personality Questionnaire-Revised (EPQ-R; Eysenck, Eysenck, & Barrett, 1985).

In total, 1976 students (n = 1111 [56.2%] female)[10] filled out the screening questionnaire. Students scoring in the upper 33% on the neuroticism inventory were overrecruited relative to participants scoring in the middle and lower-thirds. Of the 1976 students who completed the EPQ-R, 1269 (797 female, [63%]) were invited to participate in the study, and 627 students received parental consent (and assented themselves) to participate in wave 1 of the data collection across both sites (N = 305 at Northwestern University; N = 322 at UCLA). On the basis of the neuroticism scores from the EPQ-R, students were classified as low-risk (n = 114, 18.2%), medium-risk (n = 145, 23.1%) or high-risk (n = 368, 58.7%). At the Time 1 assessment, participants had a mean age of 16.91 (SD = .39) and were predominantly female: 432 (69%) women, 195 men. This gender difference was due in part to women being more likely to complete the screening questionnaire and to enroll in the study if invited, as well as because

[10] Gender information was missing for 12 cases who completed the screening questionnaire and for three cases that were invited to participate in the study. Gender information was available for all 627 participants who entered the study.

women scored more highly on the screening instrument (as expected based on prior research e.g., Costa, Terracciano, & McCrae, 2001). The ethnic composition of the final sample was as follows: African American, 13.1%; Asian American or Pacific Islander, 4.9%; Caucasian, 48.2%; Hispanic or Latin American, 15.3%; other or mixed ethnicity, 18.5%. Of the 627 participants who completed the interview component of the Time 1 assessment, 607 (96.8%) filled out the questionnaire packet containing the symptom measures.

Procedure

At Time 1, participants were administered a semi-structured life stress interview (LSI; Hammen, 2002) and the Structured Clinical Interview for DSM-IV-Lifetime Version (SCID; First, Spitzer, Gibbon, & Williams, 2002). Either at the same session as the interviews or at a session shortly thereafter, participants filled out a battery of questionnaires, which included the three measures of depressive symptomatology and five measures of anxiety sympotmatology described below. The initial assessment lasted approximately 3 hours. For the purposes of assessing reliability of life stress interviews, all interviews were audiotaped with the participant's permission. Reliability of diagnostic interviews was assessed by having two interviewers present during selected interviews. Participants were financially compensated for completion of the interviews and the questionnaires ($40 at Time 1; $45 at Time 3).

Participants were followed-up at an intermediate assessment approximately 5-8 months after Time 1 for administration of symptom and self-report life stress measures (Time 2). At Time 3 assessment, the LSI, a modified SCID covering the follow-up period only, and the full questionnaire packet were re-administered.

At Time 3, 497 (79.2%) participants completed the LSI and 496 participants completed the SCID (mean T3-T1 interval = 402 days, SD = 64; range: 183-644).[11] There were no significant differences between the 497 participants interviewed at Time 3 and the 130 participants who were not interviewed on gender, ethnicity, history of depression, or history of anxiety. There was a significant difference between groups on distribution of risk status, with higher percentages of low-risk and high-risk participants in the drop-out group, and thus a higher percentage of medium-risk participants in the follow-up group ($\chi^2(2) = 10.07$, $p < .01$). Across the eight measures of depression or anxiety symptoms, only one measure differed significantly between groups, and it was the follow-up group who scored more highly on that scale at Time 1. The final sample used in the present study consisted of 486 participants. The exclusion of 11 participants was based on incomplete diagnostic assessment in full or in part (two cases), chronic Major Depressive Disorder across Time 1 and Time 3 (two cases), or diagnosis of Bipolar Disorder I or II or possible psychotic disorder (seven cases). Of the 486 eligible participants with diagnostic and life stress interviews at both time points, 479 completed questionnaires at Time 1 and 442 completed questionnaires at Time 3, although only 436 of those cases completed enough symptom measures to be eligible for analyses predicting follow-up symptom levels.

Interview-Based Measures

Life Stress Interview. Assessment of both episodic and chronic life stress was based on the UCLA Life Stress Interview (LSI; Hammen, 2002; also Daley et al., 2000; Hammen et al., 1987). This semistructured interview assesses chronic stress across 10 domains: close friendships, social group, romantic relationships, family relationships, academic performance,

[11] Two additional participants completed only the questionnaire measures at Time 3.

neighborhood, finances, work, and health (self and family). Episodic stressors are assessed at the end of each domain module; participants are asked if there were any particular events in the previous year that had occurred within that specific domain. The LSI covers the previous 12 months; therefore the Time 1 assessment reflected chronic and episodic stress throughout the year before Time 1. The Time 3 LSI covered the period between Time 1 and Time 3. When the LSI assessments were conducted more than 12 months apart, there was some inconsistency in the period of time covered by the Time 3 LSI. In some cases only the most recent 12 months were covered, in other cases the full length of follow-up was covered. Thus, for some participants, there is the possibility of small gaps of time for which no chronic stress data are available.

Regarding *chronic interpersonal stress*, 4 of the 10 chronic domains were the focus of this project: close friendships, social group, romantic relationships, and family relationships. Within each of these interpersonal domains, several aspects of the relationship were probed and utilized to form a single rating. Interviewers rated each domain on a 1-5 scale with a rating of 1 designating exceptionally favorable circumstances and a rating of 5 representing the most stressful circumstances. Of note, because the rating of 1 was reserved for exceptional circumstances, it was rarely used. Furthermore, whereas the interview covered a 12-month period, if there were substantial changes in an individual's circumstances (e.g., two separate committed relationships), then the ratings for each period were prorated to form a single score for the year (Hammen, 2002).

The anchors on which ratings in each domain were made are briefly described. For example, a close friendship would receive a rating of 1 if it contained the following characteristics: "(m)utually satisfying, reciprocal, good conflict resolution, mutual disclosure in

many areas." A rating of 3 connotes either a "close, confiding friendship (that) may be unstable at times" or the "presence of only a moderately close friendship that is fairly stable." A rating of 5 represents the "(a)bsence of a close, confiding friendship where there is no one they feel close to or confide in."

For social life, an exceptional social life (1) includes "many good friends, very popular and engages in frequent social activities (at least every weekend)…gets along well with others, no conflict." A rating of 3 entails "some activities on weekends (but not every weekend), some conflicts with peers or difficulty making and keeping friends" and a 5 indicates "(s)evere social problems with no friends…rejected by peers."

For romantic relationships, participants were rated either on the quality of their committed relationship and/or the quality of their casual dating relationships and/or their satisfaction with being single. If a participant was in a committed relationship for only part of the time period covered by the interview, then ratings were prorated to represent the relative contributions of the relationship and non-relationship periods (and likewise for two different relationships). A rating of 1 indicates either an exceptional relationship "on all quality factors" (e.g., closeness, support, conflict, conflict resolution) or, if dating, frequent dating of partner(s) who have "excellent potential for future relationship" or, if not dating, being "completely satisfied without partner…has other life plans for now and adequate social life." A rating of 3 indicates a committed relationship that "has some significant problems,…but basic strong foundation is present," or the person engages in some dating with partners of limited potential for future relationships, or the participant is "looking for someone and spends time thinking about how to find someone…occasional distress about not having a partner." Finally a 5 indicates

either an abusive relationship, being mistreated on dates, or "ongoing concern about never having a partner."

In the family relationships section, the quality of relationships with each parent (and step-parent) as well as siblings is evaluated. The rating is more strongly weighted by the participant's relationships with caregivers than with siblings. Based on factors such as frequency of contact, closeness, understanding, and conflict, a rating of 1 indicates an excellent relationship with family members that is "exceptional on all quality factors, good conflict resolution." A rating of 3 indicates a "(g)ood quality relationship with one parent, some problems with other parent (e.g., lack of communication, trust, availability, etc.)." Finally, a 5 indicates significant and enduring problems such as abuse or neglect. Particularly favorable or unfavorable relationships with siblings could lead to small adjustments to the overall rating.

In the present study, 44 cases were selected for reliability from the first cohort at the first assessment: 38 cases were rated between-sites and 38 cases were rated within-sites (i.e., most of these cases contributed to both within- and cross-site reliability). Cross-site reliability estimates across the four interpersonal domains ranged from intraclass correlations of .66-.76, and within-site reliability ranged from .63-.80. In previous work, Hammen and colleagues have aggregated scores across domains to create a summary score of chronic stress (e.g. Daley et al., 2000). For a small number of analyses, a chronic interpersonal stress composite was formed by averaging ratings from the close friendship, social group, romantic relationship, and family relationship domains (α = .64 at Time 1; α = .57 at Time 3).

As noted above, the occurrence of *episodic stressors* was elicited from general probes within the LSI. As described in Hammen (1991), the interviewer probed the details of each

event and ascertained the degree and duration of its consequences, the participant's prior experience with the event, and the availability of social support.

Information on episodic events was then later presented by the interviewer to an independent team of at least two raters who evaluated the event on its contextual threat. The contextual threat methodology (Brown & Harris, 1978) utilizes the circumstances surrounding an event in rating "objectively" how much impact a particular event would have for a similar person in similar circumstances. Information about how the particular participant felt/reacted to the event was not presented. Ratings were made on a scale of 1-5 with 1 representing minimal or no negative impact, 2 indicating mild impact, 3 representing moderate impact, 4 indicating marked impact with many consequences, and 5 signifying severe and catastrophic negative impact (Hammen, 2002). A consensus rating was reached by the team for the severity of each event. In rare instances where individual ratings were greater than one point apart, the episode was presented to an additional rater, and consensus was then reached.

Given that the present project focused on interpersonal events, only a subset of life events were examined. The selected categories of events involved either loss or conflict within one's peer or family networks. The focus of events was determined by using the event codes given by the rating team to categorize events. Peer events included: death of a close friend, end of a dating relationship or engagement, serious argument or difficulties with romantic partner, serious argument or problem with a friend, end of a friendship, separation from a significant friend,

pregnancy, and abortion/miscarriage. Family events included death of a family member, serious argument or problem with a family member, and separation from a family member.[12]

For many events, participants were unable to state the exact date on which the event occurred. To facilitate analyses, a protocol was developed to translate the descriptors given by participants (e.g., "mid-December", "two weeks ago") into specific dates (see Appendix A). This methodology approximated the date of the event, and was useful in estimating whether events fell within a specified time frame relative to the outcome measures.

Reliability analyses have been conducted on 79 events (not necessarily interpersonal) from 17 cases assessed at the UCLA site at Time 1 for the first cohort. These events were rated by the Northwestern site and yielded an intraclass correlation of .66 for the objective threat ratings.[13]

Structured Clinical Interview for DSM-IV-Lifetime Version. The SCID-IV Lifetime Version (First et al., 2002) was administered to participants at the initial interview. This semistructured interview is used to derive diagnoses consistent with *DSM-IV-TR* (American Psychiatric Association, 2000) criteria across a range of disorders. The present project focused only on mood and anxiety disorders. Zanarini et al. (2000) reported that the SCID-IV yielded excellent agreement for depressive disorders (median $\kappa = .80$ for Major Depression, .76 for dysthymia) and fair to good agreement for several anxiety disorders (median $\kappa = .65$ for panic

[12] The event codes used in the LSI do not distinguish between the death of a close friend or a family member, or specify from whom a separation occurred. Those events were recoded through examination of the hardcopy of the LSI and identification of a target person for the event.

[13] These reliability data are notably broader than would be ideal in that all kinds of events were rated. It is assumed that the level of reliability would not vary across events types, such as those that are the focus of the present study. As part of the larger study, additional reliability data on the episodic threat ratings are in the process of being gathered.

disorder, .63 for social phobia, .57 for obsessive-compulsive disorder, and .63 for generalized anxiety disorder) (see also Williams et al., 1992).

At Time 1, participants were queried about both current and lifetime diagnoses of mood and anxiety disorders (as well as other disorders not discussed here). Interviewers also made a severity rating of the participant's level of impairment or distress using a 1-8 clinician severity rating (CSR; Di Nardo & Barlow, 1988). Ratings from 1-2 indicated subthreshold disturbances, 3 represented borderline disturbance, and 4-8 were gradations of clinically significant impairment and distress. For current diagnoses, the CSR refers to the severity of disturbance within the past month. For past diagnoses, the CSR ratings were condensed into three categories because of the difficulties of acquiring detailed information about past impairment or distress. CSR ratings were categorized as either No, denoting subclinical psychopathology; ?, denoting borderline disturbance; or Yes, indicating clinical levels of disturbance (mild to severe). Diagnoses and severity ratings were presented by the interviewer at weekly supervision meetings with a doctoral-level supervisor and consensus diagnoses using *DSM-IV-TR* criteria were reached. At Time 3 the SCID protocol was modified to assess diagnoses only during the follow-up period.

As part of the larger study, 69 cases were selected for reliability analyses at Time 1. Two interviewers attended the same interview. One interviewer conducted the diagnostic interview, and at the conclusion of his or her evaluation, the second interviewer was able to ask additional questions, without the first interviewer present. Reliability was based on agreement on both the diagnostic category and whether the diagnosis was clinically significant (CSR of 4 or higher). Reliability for cases of MDD ($\kappa = .83$) and social phobia ($\kappa = .65$) were good. Several other

diagnoses were fairly rare among these 69 cases chosen for reliability (fewer than 5 cases by either interviewer). For those diagnoses, interrater agreement ranged from excellent (e.g., generalized anxiety disorder $\kappa = .85$, obsessive-compulsive disorder $\kappa = .85$) to poor (specific phobia $\kappa = .38$).[14]

Measures of Depressive and Anxiety Symptomatology

Mood and Anxiety Symptom Questionnaire (MASQ; Watson, Clark, et al., 1995). The MASQ consists of 90 items that the participant rates on a 1-5 scale from "not at all" to "extremely." The MASQ contains five subscales: General Distress: Mixed (15 items); General Distress: Anxiety (11 items); General Distress: Depression (12 items); Anxious Arousal (17 items); and Anhedonic Depression (22 items comprised of 8 loss of interest and 14 reverse scored high positive affect items). Each of the scales has demonstrated strong internal consistency ($\alpha > .8$) in student, adult, and patient samples (Watson, Weber, et al., 1995). Furthermore, the specific symptom scales, Anhedonic Depression and Anxious Arousal, have demonstrated excellent convergent validity with the General Distress: Depression and General Distress: Anxiety scales, respectively. The specific symptom scales also demonstrate discriminant validity, with each related more strongly with construct specific symptoms (Watson, Weber, et al., 1995). In the present investigation, the two depression and two anxiety symptom scales had good internal consistency ($\alpha > .84$ at Time 1 and Time 3).

Inventory to Diagnose Depression (IDD). The IDD (Zimmerman, Coryell, Corenthal, & Wilson, 1986) is a 21-item self-report measure that can be used to diagnose MDD, although in

[14] A total of 4 cases were rated by both interviewers as meeting diagnostic criteria A, B, C, and D for Specific Phobia. However, in only one of the four cases did both interviewers agree that the distress or impairment was clinically significant.

this study it was used as a symptom measure rather than a diagnostic one. Items are scored on a 0 to 4 scale with participants endorsing the item that best describes how they felt over the past week. Zimmerman et al. (1986) reported convergent validity with other interview and self-report measures and high internal consistency (α = .92) in their sample of depressed and nondepressed participants. Because the present study examined both depression and anxiety outcomes, two items related to anxiety were deleted from the IDD. Coefficient alpha for the 19-item IDD in this sample was good (α = .87 at Time 1, α =.85 at Time 3).[15]

Social Phobia Scale (SPS). This study used the 13-item version of the SPS (Mattick & Clarke, 1998), which assesses symptoms of social phobia that have a self-consciousness focus. Items are scored on a 0 to 4 scale ranging from "not at all typical of me" to "extremely typical of me." Zinbarg and Barlow (1996) reported that this 13-item version has strong internal consistency (α = .92) and construct validity, and is more factorially homogeneous than the full SPS. Internal consistency for this scale in the present study was good (α = .88 at Time 1; α = .89 at Time 3).

Situational Fears Questionnaire (SFQ). A 22-item measure assessing fears was adapted from the Albany Panic and Phobia Questionnaire (Rapee, Craske, & Barlow, 1995). The scale contains 11 items that measure interoceptive fears and 11 items related to agoraphobic situations. These two scales correlate highly (Zinbarg & Barlow, 1996) and were combined to yield a single total score for the scale. Item scores range from 0, representing "no fear," to 8 which signifies

[15] Correlations between the 19-item and 21-item versions of the IDD were extremely high (*r* = .99 at Times 1 and 3).

"extreme fear." In the current study, internal consistency was good (α = .89 at Time 1, α = .91 at Time 3).[16]

Fear Survey Schedule (FSS). A 10-item scale was derived from the larger Fear Survey Schedule-II (Geer, 1965). Items from three subscales identified by Zinbarg and Barlow (1996) were included: blood/injury, heights, and animals. Items are scored from 0 to 6, ranging from fearfulness levels of "none" to "terror." Because the present project sought to operationalize fear on a continuum rather than for diagnostic purposes, the 10 items were summed to form a single score (α = .81 at Time 1, α = .82 at Time 3).

Preparation for Analyses

Diagnostic Groups. Major Depressive Disorder cases were defined by the onset of a depressive episode during follow-up with a CSR of at least 4. Thirty-eight cases (33 females, 5 males) were included in this group.[17] Both first onsets (n = 19) and recurrences (n = 19) were analyzed together. An interaction term containing depression history was used to determine whether separate analyses were needed for those with and without a prior history of depression.

Onset of an anxiety disorder was defined as meeting criteria for Panic Disorder, Obsessive Compulsive Disorder, Generalized Anxiety Disorder, Specific Phobia, or Social Phobia during follow-up. Twenty participants (17 females, 3 males) were included in this group. Notably, it was permitted for the participant to have symptoms of the disorder at Time 1, so long

[16] For all other questionnaires, a minimum of 80% of answers had to be present. For 38 cases of the SFQ at Time 1, the last six items were missing, apparently because participants neglected to fill out the reverse side of the questionnaire. For these cases, the total scale score was based on the average score from the first 16 items. In all 38 cases, no other items were missing. Further, for the 412 participants who completed all items, the correlation between the total score from the first 16 items and the 22 item total score was $r = .97$.

[17] Included in this group were two cases that were given a diagnosis of Bipolar Disorder Not Otherwise Specified. In both cases, the level of hypomanic symptoms was subthreshold.

as the disorder was not clinically significant.[18] All cases, both first onsets (n = 10) and cases with a prior history of anxiety disorder (n = 10), were analyzed together. An interaction term including anxiety history was used to determine whether separate analyses for those with and without a prior history of anxiety were needed.

The comparison cases (n = 430; 289 females, 181 males) consisted of participants who did not meet criteria for inclusion in either the depression or anxiety case groups. Thus, the comparison group was identical for analyses predicting MDD or anxiety disorders. Importantly, the comparison group was not a "healthy control" group. Although most comparison participants did not experience significant depression or anxiety during follow-up, some participants did. In addition to the 349 participants who did not experience significant anxiety or depression during follow-up, the comparison group contained 81 (18.8%) participants who fell into one or more of the following categories: met symptom criteria for MDD during follow-up but had a CSR of less than 4 (n = 3); met diagnostic criteria for dysthymia with a CSR of 4 of higher at Time 3 (n = 3);[19] met criteria for a Depressive Disorder Not Otherwise Specified during follow-up with a CSR of 4 or higher (n = 2); met criteria for an Anxiety Disorder at Time 1 and Time 3 with a CSR of 4 or higher (n = 11); met symptom criteria for an Anxiety Disorder during follow-up but had a CSR less than 4 (n = 61); or met criteria for an Anxiety Disorder Not Otherwise Specified, with a CSR of 4 or higher (n =8). Although the presence of such psychopathology may have made comparisons with the depression and anxiety case groups more conservative, these cases comprised fewer than 20% of the comparison group.

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[18] One case who met criteria for Panic Disorder during follow-up was excluded from the anxiety disorder analyses because the Time 1 CSR was missing from the database. Thus, it was not possible to determine whether this disorder was present at clinically significant levels at Time 1.

[19] Two of the three cases also met diagnostic criteria for dysthymia at Time 1.

Symptom Measures. Several symptom outcome measures were formed. A depression symptom composite was derived from the IDD, MASQ-General Depression, and MASQ-Anhedonic Depression scales. Average-item scores from each scale were standardized and averaged (α = .84 at Time 1 and Time 3). An anxiety symptom composite was derived from the MASQ-General Anxiety, MASQ-Anxious Arousal, SPS, SFQ, and FSS (α = .78 at Time 1 and Time 3). Finally, for analyses predicting changes in symptom levels (rather than regressed change), a composite of change scores was formed. For depressive symptoms, each of the three symptom scales was standardized, Time 1 levels were subtracted from Time 3 levels, and the individual scale change scores were averaged (α = .81). Similarly for anxiety symptoms, each of the five symptom scales was standardized, Time 1 levels were subtracted from Time 3 levels, and the individual scale change scores were averaged (α = .70). [20]

Life Events. Several episodic life stress measures were created. For analyses in which MDD onset was the dependent variable, the presence/absence of an event of at least mild severity ($\geq$ 2.0) was measured during the period 3 months prior to onset for MDD cases and during the period 3 months prior to interview for comparison cases. The presence/absence of events was coded separately for family events and peer events. Using the presence or absence of a major stressor is a very common operationalization of episodic stress (Hammen, 2005), and has been used in adolescent samples (e.g., Williamson et al., 1998).

[20] In several cases, one of the individual measures that formed the composite was missing, and the composite was formed from the remaining two (for depression) or four (for anxiety) scales. This occurred in 10 cases for the Time 1 Depression Composite, 4 cases for the Time 3 Depression Composite, 12 cases for the Depression Symptom Change Score, 10 cases for the Time 1 Anxiety Composite, 3 cases for the Time 3 Anxiety Composite, and 11 cases for the Anxiety Symptom Change Score. For reasons that are unclear, 24 participants did not complete the SPS and SFQ at Time 3 although they had completed the other anxiety scales. Those cases were excluded from the composite because a threshold was set that a maximum of one scale could be missing from the composite.

For MDD cases whose depressive episode onset less than 3 months into the follow-up period, events from the Time 1 assessment were used if they fell within the 3-month window. The 3 month window was chosen for several reasons. First, Hammen (2005) summarized that most studies of life events and depressive disorder onset have used a 3-6 month time frame. Second, work by Hammen and colleagues (Daley et al., 2000) has found that episodic stress was elevated in the 3 months prior to disorder onset. Livianos-Aldana et al. (1999) reported that increased episodic stress levels were found in cases with depressive disorder relative to controls beginning about 26 weeks prior to onset, with a pronounced elevation 7 weeks before onset.[21] Thus, there is reason to examine stressors relatively close in time to disorder onset.

The presence/absence of family and peer events was also calculated for the period 12 months prior to onset for MDD cases and 12 months prior to interview for comparison cases.[22] Focusing solely on life events that occurred close in time to the onset of MDD may overestimate the effects of stress on depression (R.E. Zinbarg, personal communication). By examining a longer period of pre-onset time, it is possible to assess for the occurrence of life events in both the MDD and comparison cases over a time period that is not yoked to the occurrence of disorder. This analytic approach is more closely, although not exactly, geared towards assessing the *impact* of life events, rather than the *frequency* of life events (Monroe & Harkness, 2005). The former refers to the likelihood of developing depression given the occurrence of a life event, whereas the latter focuses on the percentage of depressed cases that had a recent life stressor prior to onset (Monroe & Harkness, 2005). Onset dates for depressive episodes were estimated

[21] That study used a decay model in which an event was considered to have a certain level of impact at the time of its occurrence and continued to have decreasing effects over time. The authors thus combined all of the residual stress effects for each week over the 52 weeks preceding the onset of depressive disorder in cases versus 52 weeks prior to interview for the control participants.

[22] Technically, the 3-month period was 90 days long and the 12-month period was 360 days long.

employing the same rubric used for estimating the dates of stressful events (Appendix A).[23] An estimated onset date could be calculated for 36 of 38 cases.

Two additional criteria were utilized for determining eligibility for episodic analyses of MDD. First, the targeted period for assessing events occasionally coincided with a gap in the follow-up life stress assessment as mentioned above. If any portion of the targeted period was not covered by the LSI, the case was excluded from those analyses. Second, care was taken to ensure that the targeted period for assessing life events did not overlap with a prior depressive episode. A minimum of one month was required between the offset of a prior depressive episode and the beginning of the time period during which events were assessed. Although many major depressive episodes were given an onset and offset date, many participants who were in the midst of a current depressive episode at Time 1 were not assigned an offset date for that episode at the follow-up interview. For those cases, participants were only included in analyses if the time window for assessing events ended at least 1 month after the Time 1 assessment. Given these two criteria used for determining eligibility for episodic analyses, several MDD cases were excluded: only 32 of 38 cases were eligible for the 3-month analyses and 22 of 38 cases were eligible for the 12-month analyses.

For analyses in which anxiety disorders were the outcome variable, onset dates for disorder were generally not available, likely due to the chronicity of the symptoms for most of the anxiety disorders. For most new cases of anxiety disorder, the participant provided an estimated date for the onset of the *symptoms* of the disorder, which often preceded the Time 1

[23] For one MDD case this protocol was adjusted. A participant acknowledged that an event preceded her depressive episode and so the estimated date of that event was moved to ensure that it occurred prior to episode onset. Rigid adherence to the dating rubric would have placed the depression as occurring *before* the event.

interview, even though the participant did not meet full diagnostic criteria for that disorder at Time 1. At most 2 of the 12 anxiety disorder cases at the Northwestern site provided an onset date that occurred during the follow-up interval. Given this lack of precision in dating the onset of most anxiety disorders, life event analyses were not run.

For analyses examining symptoms as the outcome variables, the presence or absence of an event of at least mild severity (≥ 2.0) during the 3 months prior to the administration of the follow-up questionnaires was calculated for both peer and family events. In order to minimize the possibility that stressful events might have occurred between the life stress assessment and the completion of the questionnaires, cases were excluded if the questionnaires were filled out more than 15 days after the life stress interview was administered. A total of 409 cases were eligible for analyses involving episodic stress and symptom level outcomes.

Extreme Values. Predictor and outcome variables were screened for univariate outliers using visual inspection of histograms, examination of graphic plots indicating values outside Tukey's outer fences (Velleman & Hoaglin, 1981), and z-score values greater than 3.29 (Tabachnick & Fidell, 2007) as guides. In order to decrease the possible influence of extreme values and/or improve skewness/kurtosis of distributions, some values were recoded. The following variables had some recoded values: Time 3 Peer Chronic Stress (1 case), Time 3 Dyadic Chronic Stress (5 cases), Time 3 Chronic Interpersonal Stress Composite (2 cases), Time 1 Depression Composite (1 case), Time 3 Depression Composite (3 cases), Depression Symptom Change Score (4 cases), Time 1 Anxiety Composite (3 cases), Time 3 Anxiety Composite (2 cases), and Anxiety Symptom Change Score (4 cases).

Plan for Analyses

All analyses examining MDD onset were run three ways. Because Time 1 depression symptoms might plausibly serve as the mediator of the prospective relationship between Time 1 chronic stress and MDD onset, analyses were run first without the Time 1 depression symptom composite entered as a covariate. A second analysis then included the Time 1 symptoms in the model. Finally, a third model included baseline depression symptoms and excluded all cases with a current Time 1 mood disorder (dysthymia or major depressive episode) to safeguard against effects driven largely by state-dependent reporting of life stress.

For anxiety disorder outcomes, only a history of anxiety disorder term was entered as a covariate, but no symptom measure was included. This decision was based on the fact that the anxiety disorder group was comprised of a range of disorders rather than a single disorder. Thus, the use of the anxiety symptom composite seemed less appropriate for gauging participants' level of symptomatology of the particular disorder they ultimately developed.

Symptom level analyses were run two different ways. One set of analyses examined regressed change, that is, the degree to which predictors accounted for variance in Time 3 symptom levels beyond the Time 1 symptom levels. A second set of analyses examined the relationship between stress and actual change in levels of depression or anxiety symptoms (i.e., the dependent variable was a change score).

To protect against type I error, partial regression coefficients were only interpreted if the full step of entry made a statistically significant addition to the overall model at $p < .05$.

1.) The first objective of the study was to examine the relationship between individual domains of chronic interpersonal stress with depression and anxiety outcomes. Three sets of analyses addressed this first objective.

A. The first set of analyses examined individual domains of chronic stress as predictors of symptom and disorder level outcomes. Each of the four domains of chronic stress (close friendships, social group, romantic relationships, and family relationships) was examined in separate analyses. For diagnostic analyses the chronic domain and appropriate covariates were entered on step 1, and the interaction of domain x disorder history was entered on step 2. As discussed below in objective 4, gender was not entered into diagnostic analyses. For symptom level analyses, the chronic domain, gender, and baseline symptoms (when appropriate) were entered on step 1, and the interaction of gender x domain was entered on step 2. *Time 1* measures of chronic stress were used in the prediction of both diagnostic and symptom level outcomes. However, given that there was a substantial time gap between the Time 1 and Time 3 assessments, there were competing concerns between ensuring that stress preceded psychopathology and assessing chronic life stress close in time to the outcomes. Therefore, symptom level analyses were also conducted using Time 3 measures of chronic stress.

B. The second set of analyses addressing the study's first objective examined the unique relationship between domains of chronic stress and depression and anxiety outcomes. All four domains of chronic stress (close friendships, social group, romantic relationships, and family relationships) were entered *simultaneously* in regression equations. For analyses predicting MDD, the history and symptom covariates (when appropriate) were entered on step 1 and the four chronic stress domains were entered on step 2. For analyses predicting anxiety disorder, lifetime history of anxiety disorder was entered on step 1 and the four domains of chronic stress were entered on step 2. Finally, chronic domains were examined as predictors of both regressed and actual change in depression and anxiety symptoms levels from Time 1 to Time 3. For

regressed change analyses, the Time 1 symptom levels were entered on step 1 and the chronic domains were entered on step 2. This sequence of entry across analyses was chosen because the focus of these analyses was to examine the incremental contribution of the group of interpersonal domains and establish whether any of the domains was uniquely significant.

C.) The third set of analyses addressing the study's first objective examined the chronic interpersonal stress composite. These analyses examined the utility of using a single measure averaging across all four interpersonal domains.

2) The second objective of the study was to examine the interactions among different domains of chronic stress. Three specific interactions were explored: (1) family relationships X dyadic relationships (averaged across close friendship and romantic relationship domains); (2) family relationships X social group; and (3) family relationships X peer relationships (averaged across close friendship, social group, and romantic relationship domains). For example, in the prediction of MDD, one regression analysis included dyadic relationships, family relationships, and depression history on step 1 and the interaction of dyadic relationships and family relationships on step 2. Symptom level analyses were initially run using *Time 1* measures of chronic stress, and then run again using *Time 3* measures of chronic stress.

3) Third, this study examined the interaction between chronic stress and episodic stress. For these analyses, episodic events were divided into peer events and family events. In order to ensure that the chronic conditions were present at the time of episodic stressors, the *Time 3* chronic interpersonal stress domains were used. An important consequence of using Time 3 chronic stress is that for analyses predicting MDD, the chronic stress variables may cover periods before, during, and after the episode of major depression.

In the "matching analyses," the interaction between chronic peer stress and peer events as well as between chronic family stress and family events were examined. In the "cross-domain" analyses, the interaction of chronic peer stress and family episodic events (and vice versa) was explored. For the prediction of MDD onset, depression history, Time 1 symptoms (when appropriate), chronic (peer or family) stress, and episodic (peer or family) stress were entered on step 1; the chronic x episodic interaction was entered on step 2. For anxiety disorder onset, no analyses were run given that the vast majority of cases did not have a disorder onset date during follow-up. For symptom level outcomes, baseline symptoms were entered on step 1 (where appropriate), stress main effects were entered on step 2, and the interaction of chronic and episodic stress was entered on step 3.

4) The fourth objective of this study was to examine the main effects and possible moderating effects of gender on the relationship between stress and internalizing psychopathology. Although this study had intended to examine whether gender moderated chronic life stress in the prediction of diagnostic outcomes, the gender distribution of disorders (33 females vs. 5 males for MDD; 17 females vs. 3 males for Anxiety Disorders) made those proposed analyses less feasible. For example, in the initial analyses predicting MDD from the close friendship domain, regression diagnostics suggested that one case (a male participant) may have had undue influence on the overall model. Removal of that case led to the appearance of another influential case (also male) and so on, until parameter estimates were unable to be interpreted (i.e., had huge standard errors and/or confidence intervals). Unfortunately, it appeared prudent to exclude gender from diagnostic analyses in order to increase interpretability of other partial regression coefficients.

Gender was included in several analyses predicting symptom level outcomes. Gender was entered as a main effect and in interaction with the individual chronic domains (analyses described in objective 1A.). Gender was also included in analyses examining the interaction between chronic domains (objective 2). Gender was not included in episodic event analyses (objective 3) because the frequency of recent peer or family events was already fairly rare in the symptom analyses (< 10% of participants) and inclusion of gender interactions led to a sequence of influential cases on the partial regression coefficients for interaction terms.

RESULTS

Descriptive Statistics

At the Time 1 diagnostic assessment, 100 of 486 (20.6%) participants met criteria for past or current Major Depressive Disorder and 86 (17.7%) participants met criteria for a current or past anxiety disorder. Twenty-three (4.7%) participants were experiencing a clinically significant mood disorder (MDD or dysthymia) at the Time 1 assessment and 67 (13.8%) participants were experiencing a clinically significant anxiety disorder.

Means and standard deviations for each of the symptom scales at both time points are presented in Table 1. Paired samples t-tests revealed that participants scored significantly lower on all symptom measures at Time 3 than they did at Time 1 assessment.

Gender differences were explored for MDD onset, anxiety disorder onset, depression history, and anxiety history. Gender was significantly associated with MDD onset $\chi^2(1) = 6.27$, $p < .05$. Examination of the 468 cases eligible for MDD diagnostic analyses revealed that 5 (3.4%) out of 146 males experienced a depressive episode whereas 33 (10.2%) out of 322 females experienced a depressive episode during follow-up. Gender approached significance as a predictor of anxiety disorder onset $\chi^2(1) = 2.78$, $p < .10$; rates of onset were 2.1% (3/144) for males and 5.6% (17/306) for females. Gender differences in history of psychopathology were conducted using the full sample of 486 participants. Gender differences did not reach statistical significance for history of (past or current) anxiety disorder at Time 1 (18.1% males vs. 17.6% females $\chi^2(1) = .02$, ns), but approached significance for history of MDD (22.8% females vs. 15.4% males $\chi^2(1) = 3.47$, $p < .10$).

Comparisons between genders on chronic interpersonal domains and symptom outcomes are presented in Table 2. Gender differences were found on only two variables. Women had significantly better close friendships at Time 1: $t(484) = 2.59$, $p < .01$, and this difference approached statistical significance at Time 3: $t(484) = 1.80$, $p < .10$. Women also experienced significantly higher levels of anxiety symptoms at Time 1: $t(473) = -2.38$, $p < .05$, a difference which approached statistical significance at Time 3: $t(411) = -1.81$, $p < .10$.

Correlations among Time 1 chronic interpersonal stress variables and outcomes are presented in Table 3. Chronic life stress variables (rows 6-12 in Table 3) generally had small, but significant relationships with MDD onset during follow-up (*r*'s =.09-.21). Associations between chronic stress and onset of anxiety disorder during follow-up were even smaller in size (*r*'s = .03-.13). Chronic life stress variables had small, but significant correlations with Time 3 depression symptom levels and smaller, but generally significant relationships with Time 3 anxiety symptoms. These results suggest that higher scores on the chronic interpersonal stress variables, indicating poorer relationships, were associated with higher levels of psychopathology. Associations between chronic interpersonal stress and depression and anxiety symptom change scores were negative, such that worse circumstances at Time 1 were associated with decreases in symptoms over follow-up. As discussed more at length below, the most plausible explanation for these relationships is a regression to the mean effect, i.e., participants with higher symptom scores at Time 1 (and higher levels of chronic interpersonal stress) tended to have lower symptom scores at Time 3.

Individual Domains of Chronic Interpersonal Stress

Logistic regression analyses predicting MDD during follow-up from each domain of chronic interpersonal stress at Time 1 are presented in Table 4.[24] Coefficients are presented only for the life stress domain because the step of entry for main effects was always significant and the juxtaposition of coefficients for each of the three analytical approaches was meant to facilitate interpretation. Chronic stress in close friendships, social group, and family relationships (but not romantic relationships) were all significant predictors of increased risk for MDD during follow-up beyond the depression history term (Odds ratios: 1.38, 1.74, 1.58, respectively, $p < .05$). Only social group was a significant predictor of MDD onset beyond depression history and the Time 1 depression symptom composite (Odds Ratio (OR) = 1.52, 95% Confidence Interval (CI) = 1.11-2.09).[25] Only romantic relationships interacted significantly with depression history: step $\chi^2(1) = 4.05$, $p < .05$. Separate analyses were run for participants with no history of depression and participants with a history of depression. In neither analysis was the main effect of romantic relationships statistically significant; however, the direction of the relationship differed such that romantic relationship stress was associated with increased risk of MDD for first onsets, but not recurrences.

Table 5 displays the relationships between individual domains of chronic interpersonal stress at Time 1 and anxiety disorder onset during follow-up. No domain of chronic interpersonal stress was significantly associated with the onset of anxiety disorders. Further, there were no significant interactions between history of anxiety and any domain.

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[24] As a general rule, results are presented using all cases. Instances where deletion of a case affected the statistical significance of a step or coefficient will be noted. Particularly for logistic regression analyses, conservative exclusion procedures were used because regression diagnostics were developed for Ordinary Least Squares analyses, not logistic regression (see Cohen, Cohen, West, & Aiken, 2003, who recommend taking an extra measure of caution when deleting cases in logistic regression based on regression diagnostics).

[25] Analyses excluding two potentially influential cases led to a significant main effect for family relationships beyond depression history and Time 1 depressive symptoms.

Multiple linear regression analyses examined the relationship between individual domains and depressive symptoms beyond the effects of gender and Time 1 symptom levels (Table 6). Depression symptoms were moderately stable across assessments ($r = .52$, $p < .01$). Across domains, only social group made a small, but statistically significant contribution to depressive symptom outcome ($\beta = .09$, $p < .05$). Participants with good social group functioning tended to have lower levels of depressive symptoms at follow-up than participants with poorer social group relationships. Gender was not uniquely associated with symptom levels nor were any significant interactions found between gender and chronic stress domains.

Results were notably different when examining the depression symptom change score. Whereas social group and close friendships were not significantly related to the depression change score, romantic relationships ($\beta = -.19$, $p < .01$) and family relationships ($\beta = -.16$, $p < .01$) were significant predictors. In separate analyses romantic relationships accounted for 3.4% of unique variance in symptoms and family relationships accounted for 2.5% of unique variance. Further investigation of these effects revealed that participants with poor relationships at Time 1 (e.g., scores of 3.5 or 4) tended to show decreases in symptoms over follow-up, whereas participants with good functioning in those domains tended to show small increases in symptoms over follow-up.

Individual domains were also investigated as predictors of regressed change in anxiety symptoms (Table 7). Anxiety symptoms were relatively stable across time ($r = .65$). Across all four domains, only social group accounted for small, but significant variance in anxiety symptoms beyond gender and baseline anxiety symptoms ($\beta = .08$, $p < .05$). Participants with good social group functioning at Time 1 had lower mean levels of anxiety symptoms at Time 3

than participants with poor social group relationships. Gender was not a significant unique predictor of anxiety symptoms, and no interactions between gender and individual domains were significant.

For regressions predicting the anxiety symptom change score, neither main effects nor interactions steps were significant for any of the four domains. Romantic relationships did have a significant negative relationship with change in anxiety symptom scores, but as noted above, individual coefficients were only interpreted if the full step of entry was significant.

In summary, MDD during follow-up was prospectively predicted by several domains of chronic interpersonal stress: close friendships, social group, and family functioning. However, only social group was predictive of MDD beyond the effects of baseline depressive symptom levels. Social group was also the only domain predictive of Time 3 symptom levels of both depression and anxiety, beyond baseline symptom levels. Analyses of the depression symptom change score revealed a different pattern of results in that poorer romantic relationships and family functioning were associated with symptom decreases over time, likely consistent with a regression to the mean effect. Finally, no individual domain of chronic stress was significantly predictive of Anxiety Disorder onset.

Full Models of Chronic Stress

Logistic regressions examining all four domains of chronic interpersonal stress predicting MDD simultaneously are displayed in Table 8. The set of four domains accounted for a significant improvement in model fit beyond depression history ($\chi^2(4) = 15.57$, $p < .01$). Among the four domains, only social group was uniquely predictive of MDD during follow-up (OR = 1.62, 95% CI: 1.14-2.30). After entering depression history and baseline depressive symptom

levels, the step of entry containing the four domains was not statistically significant and so no individual coefficients were interpreted. However, significant results were found *after* excluding the participants with a major mood disorder at Time 1 from analyses. In this subsample (409 comparison cases, 32 MDD cases) the set of four domains did contribute significantly to model fit beyond depression history and baseline depressive symptoms ($\chi^2(4) = 9.77$, $p < .05$). Similar to the results above, only social group made a significant unique contribution to the prediction of risk for MDD during follow-up (OR = 1.56, 95% CI: 1.07-2.28). Thus, across analyses of individual domains as well as in simultaneous analyses of all four domains, social group was the most consistent predictor of risk for MDD during follow-up.

Because prior studies have generally used a composite of chronic stress domains, additional analyses were run examining the relationship between the chronic interpersonal stress composite and MDD during follow-up.[26] The chronic interpersonal stress composite contributed significantly to the model fit beyond depression history ($\chi^2(1) = 10.83$, $p < .01$), beyond depression history and baseline depressive symptoms ($\chi^2(1) = 3.85$, $p < .05$), and beyond depression history and baseline symptoms when excluding participants with current mood disorder at Time 1 ($\chi^2(1) = 4.50$, $p < .05$). The composite was thus a somewhat more consistent predictor of MDD onset than the individual domains. A one unit increase in the chronic interpersonal stress composite was associated with about twice the risk for MDD onset during follow-up across the three analyses (respectively: ORs = 2.34, 1.75, and 1.90).

[26] It should be noted that there is no accepted equivalent of R^2 in logistic regression (Cohen et al. 2003; Tabachnick & Fidell, 2007). Values such as the Nagelkerke R^2 are considered "pseudo-R^2s" (see Cohen et al. 2003, p. 502) in that they are not equivalent to the "variance accounted for" R^2 used in linear regression. Thus, no direct comparisons are made between the sizes of the change in Nagelkerke R^2 for the models using all four individual domains versus models using only the composite.

In the prediction of anxiety disorder onset during follow-up, the step containing all four chronic domains did not significantly improve model fit beyond anxiety history (see Table 9). This result was expected in light of the findings from analyses of the individual domains. Furthermore, the chronic interpersonal stress composite was not a significant predictor of anxiety outcome beyond anxiety history (step χ^2 (1) = 2.59, $p > .10$).

In the prediction of Time 3 depressive symptoms (Table 10), the full step of entry containing all Time 1 four domains did not account for significant variance beyond baseline symptom levels ($\Delta R^2 = .01$, $p > .10$). Similarly, the Time 1 chronic interpersonal stress composite also did not contribute significantly beyond baseline symptom levels, although the effect approached statistical significance ($\Delta R^2 = .01$, $p < .10$).

Examination of the depression symptom change score yielded results similar to what might be expected from the analyses examining individual domains. The four domains as a group accounted for approximately 5% of the variance in the depression change score. Unique contributions were made by romantic relationships ($\beta = -.16$, $p < .01$) and family relationships ($\beta = -.12$, $p < .05$), which contributed 2% and 1% of unique variance, respectively. As noted above, the direction of the relationship was consistent with a regression to the mean effect. In theory, an alternative possibility is that poorer romantic relationships and family relationships are truly predictive of better outcomes. However, the positive correlations between the chronic stress domains and depressive symptoms at *both* Time 1 and Time 3 are not consistent with this latter explanation. Finally, the chronic interpersonal composite accounted for approximately 3% of the variance in the depression change score ($\beta = -.16$, $p < .01$).

As shown in Table 11, the combined effects of the four domains of chronic stress did not account for significant variance beyond the Time 1 composite of anxiety symptoms ($\Delta R^2 = .01$, $p > .10$). Although social group made a significant unique contribution, this coefficient was not interpreted because the overall step of entry was not significant. Additional analyses revealed that the chronic interpersonal composite did not explain a significant proportion of the variance in Time 3 anxiety symptoms beyond Time 1 symptom levels.

Analyses in which the four chronic domains predicted the anxiety symptom change score revealed a significant overall effect ($\Delta R^2 = .03$, $p < .05$). Unique contributions to outcome were made by social group ($\beta = .12$, $p < .05$) and romantic relationships ($\beta = -.12$, $p < .05$) with each contributing 1% of unique variance to the outcome. Thus, after accounting for the effects of baseline symptoms, family relationships, and close friendships, poorer social group functioning was associated with increased symptoms over time whereas poorer romantic relationships were associated with a decrease in symptoms. Finally, the chronic interpersonal stress composite did not contribute significantly to the prediction of the anxiety change score.

In sum, in the prospective prediction of MDD during follow-up, social group appeared to be a unique indicator of increased risk for depression, although the full step of entry was significant in only two of three models. The overall effect suggested that holding covariates and other chronic stress domains constant, a one unit increase in social group stress was associated with 1.5 times the risk for MDD during follow-up. The four chronic domains as a group did not contribute significantly to the prediction of Anxiety Disorder onset during follow-up. In analyses examining regressed change, the four chronic stress domains did not account for significant variance in depression or anxiety symptoms at Time 3 beyond the Time 1 symptom

levels. Analyses examining symptom change scores revealed small but significant effects for several Time 1 domains. Poorer romantic relationships were associated with decreasing symptoms of both anxiety and depression, poorer family relationships were associated with decreasing symptoms of depression, and poorer social relationships were associated with increasing symptoms of anxiety. Several of these effects are more consistent with a regression to the mean phenomenon than a salubrious effect of poorer relationships. Finally, the chronic interpersonal composite was a significant predictor of MDD during follow-up, but was not significantly associated with prediction of anxiety disorder or regressed change in symptom measures.

Individual Domains and Full Models Using Time 3 Chronic Domains

To supplement the prospective results presented above, analyses predicting symptom outcomes were re-analyzed using Time 3 chronic stress domains as predictors. The objective of these analyses was to examine chronic stress levels closer in time to the assessment of follow-up symptoms. Analyses of individual domains revealed that Time 3 close friendships, social group, and family relationships were all significant predictors of Time 3 symptoms beyond the effects of gender and baseline symptom levels (see Table 12). Effects were small, with each domain accounting for between 1-2% of unique variance. For each of these three domains, mean levels of Time 3 depressive symptoms were lower for participants with very good relationships than for participants with poor relationships. Romantic relationships approached statistical significance as predictors of Time 3 depressive symptoms ($\beta = .08$, $p < .06$). Gender was not a significant unique predictor of symptoms in any analyses, nor were any gender x domain interactions statistically significant. Analyses examining the relationships between individual domains and

the depression symptom change score did not yield any significant main effects or interactions between gender and chronic stress domains.

In analyses examining all chronic domains simultaneously, the results using Time 3 predictors differed from the results using Time 1 predictors (see Table 13). First, the overall step of entry containing the chronic stress domains was significant, accounting for an additional 3.5% of variance beyond baseline symptoms and gender. Second, family relationships contributed significant unique variance beyond the effects of Time 1 symptoms and the other chronic interpersonal domains ($\beta = .11$, $p < .01$). Third, neither the four domains nor the chronic interpersonal composite predicted the depressive symptom change score.

In a separate analysis, the Time 3 chronic interpersonal composite was a significant predictor of follow-up depressive symptoms beyond Time 1 depressive symptom levels ($\beta = .19$, $p < .01$), accounting for 3.3% of symptom variance. Although the composite was a significant predictor of symptoms, the results from analyses of all four domains suggest that examining individual domains of chronic stress is also important. The unique predictive utility of family relationships would not be illuminated through sole reliance on a composite.

Analyses examining the relationship between Time 3 chronic stress domains and anxiety symptoms are presented in Table 14. Across all individual domains, the only significant predictor of Time 3 anxiety was Time 1 anxiety, which accounted for about 40% of the variance in outcome. Gender, individual chronic stress domains, and the interaction of gender and the domains were not significant predictors of outcome. Similarly, no significant effects were found when examining the anxiety symptom change score. Models including all four domains of chronic interpersonal stress or the interpersonal composite did not contribute significant variance

to anxiety symptom outcomes. These results differ somewhat from the prospective models of individual domains in which Time 1 social group was a significant predictor of follow-up anxiety symptoms beyond the effects of baseline levels of anxiety. A second difference is that whereas no Time 3 domains were significant predictors of the anxiety change score, romantic relationships (negatively) and social group (positively) were significant predictors in the Time 1 analyses.

In sum, Time 3 measures of chronic stress tended to account for larger amounts of variance in depressive symptoms than their Time 1 counterparts. Furthermore, Time 3 family relationships were a significant unique predictor of Time 3 depressive symptoms beyond the other chronic domains and baseline symptom levels. Unlike Time 1 analyses, no Time 3 chronic stress variables were significant predictors of anxiety symptoms. Neither main effects nor interactions involving gender were significant in any analyses.

Interactions Between Time 1 Domains of Chronic Interpersonal Stress

Analyses examining the interactions between domains of chronic stress focused on three separate sets of peer X family interactions: dyadic relationships (close friendships and romantic relationships) X family relationships, social group X family relationships, and overall peer relationships (close friendships, social group, and romantic relationships) X family relationships. The major focus of these analyses was on the statistical significance of the interaction term rather than the main effects of chronic stress which had been largely examined above. Across all analyses predicting MDD onset during follow-up, not one interaction contributed significantly to overall model fit. Illustrative results from the set of analyses using MDD history and baseline symptoms as covariates are presented are presented in Table 15.

Additional logistic regressions were conducted to examine the relationship between the three sets of interactions of chronic interpersonal domains and anxiety disorder onset. Whereas the analyses of individual domains (Table 5) did not reveal that any domains were significantly associated with anxiety disorder onset, some significant relationships were found in these analyses (see Table 16). In separate analyses, both the dyadic and peer composites were significantly associated with increased risk for anxiety disorder onset beyond anxiety history and family relationships (Dyadic: OR= 1.84, 95% CI: 1.03-3.29, $p < .05$; Peer: OR = 1.89, 95% CI: 1.03-3.48, $p < .05$). Thus, there is some suggestion that chronic peer stress may be relevant to subsequent anxiety disorder. Of note, the significance of these main effects was not maintained when a few potentially influential cases were removed, suggesting that these results be interpreted somewhat cautiously.[27] Most relevant to the primary objective of these particular analyses, the interaction between chronic domains did not contribute significantly to the prediction model in any analysis.

Multiple linear regressions examined the relationship between the interactions of chronic domains and Time 3 depressive symptoms. As shown in Table 17, no significant effects were found for gender, chronic domains, nor the two and three-way interaction terms. Results were somewhat different for analyses predicting the depression symptom change score. When dyadic relationships and family relationships were analyzed together, both were significantly associated with the depression symptom change score (Dyadic: $\beta = -.10$, $p < .05$; Family: $\beta = -.12$, $p < .05$). Specifically, higher levels of dyadic peer stress and family stress were associated with decreases

[27] However, as mentioned above, Cohen et al. (2003) recommend caution in using regression diagnostics to eliminate cases in logistic regression.

in depressive symptoms from Time 1 to Time 3. Across all analyses predicting the depression symptom change score, no two-way or three interactions were significant.

The final set of analyses examining the interactions between chronic domains focused on anxiety symptoms (Table 18). Across analyses no significant effects were found for the step of entry for chronic stress domains (step 2) nor for any interactions among domains (steps 3 and 4). Similarly, no step of entry was statistically significant in the prediction of the anxiety symptom change score.

To summarize, although some main effects for peer chronic stress were found in the prediction of MDD and anxiety disorder, not one interaction between chronic domains or between gender and the chronic domains was statistically significant across any analyses at the diagnostic or symptom level.

Interactions Between Time 3 Domains of Chronic Interpersonal Stress

The interaction between Time 3 measures of chronic interpersonal stress in the prediction of symptom level outcomes was also investigated. As shown in Table 19, the main effects step containing chronic interpersonal stress variables was significant in all analyses, accounting for about 3% of the variance in Time 3 depressive symptoms beyond the effects of gender and baseline symptoms. Each peer and family variable was uniquely significant, but across all three analyses, no step containing interaction terms made a significant contribution to the overall model.

In analyses predicting the depression change score, no main effects for chronic domains were found. However, in the analyses examining the interaction of dyadic and family variables, a significant three-way interaction with gender was found ($\Delta R^2 = .01$, $p < .05$). The dyadic x

family interaction was examined in separate regressions for males and females and a significant interaction effect was found for females (β = .12, $p < .05$), but not males (β = -.09, $p > .10$). Following the recommendations of Cohen et al. (2003), the relationship between dyadic functioning and the depression change score was plotted at values of family functioning equal to z-scores of -1, 0, and 1 representing the mean, plus values one standard deviation above and below the mean (see Figure 1). Analyses of the simple slopes (Cohen et al., 2003) revealed that quality of family relationships was significantly related to depressive symptoms when dyadic relationships were poor in quality ($t(495) = 1.98$, $p < .05$) but not when they were good ($t(495) = -.85$, ns). Thus, there is some support for buffering in that better family relationships were associated with better outcomes under poor (or stressful) dyadic circumstances. Of note, peer relationships were not significantly related to depressive symptoms when family relationships were poor in quality; thus, there was no support for buffering of family relationships by peer relationships ($t(495) = .75$, ns). Overall then the practical significance of this lone significant interaction out of the dozens of analyses run heretofore is to be viewed cautiously.

Two final sets of regression analyses examined the relationship between the interaction of Time 3 chronic domains and anxiety symptom outcomes. Across analyses predicting regressed change in anxiety as well as analyses predicting the anxiety symptom change score, neither the main effects step containing chronic stress variables nor any interaction steps were statistically significant.

Interactions Between Chronic Stress and Episodic Stress: MDD

Correlations between predictors and outcomes for analyses examining chronic and episodic variables are presented in Table 20. Before conducting logistic regression analyses,

some initial exploration of the relationship between episodic events and Major Depressive Disorder onset and history was conducted. Recent family events (occurring within 90 days of onset for MDD cases and within 90 days of Time 3 interview for comparison cases) were uncommon in the comparison group where 30 out of 430 participants had such an event (7.0%), and even more rare in the MDD case group where only 1 out of 32 eligible cases (3.1%) had such an event. Given that only a single MDD case had a recent family event, no logistic regressions examining the recent family events were conducted. With regard to recent peer events, 38 of 430 comparison cases had such an event (8.8%), whereas 8 of 32 MDD cases (25%) had an event ($\chi^2(1) = 8.68$, Fisher's Exact $p < .05$). Although the history of MDD at Time 1 was not examined as a moderator of the chronic x episodic interactions, it is notable that the rate of recent peer events in MDD cases with no history of depression (18.8%; 3 of 16 cases) was not higher than the rate in cases with a history of depression (31.3%; 5 of 16 cases). [28]

The relationship between peer chronic stress, recent peer events, and their interaction in the prediction of MDD is presented in Table 21. Peer events were significantly related to increased risk for MDD onset beyond the effects of MDD history and the Time 1 depression symptom composite (OR = 3.01, 95% CI: 1.16-7.84). However this effect only approached statistical significance when cases with Time 1 current mood disorder were excluded from analyses.[29] Similarly, in analyses examining chronic family stress, recent peer events, and their interaction predicting MDD (Table 22), peer events were a significant predictor of MDD beyond history of depression and baseline symptoms (OR = 3.34, 95% CI 1.30-8.61), but only

[28] As was noted above with regard to interactions involving gender in diagnostic analyses, the inclusion of depression history in interactions terms in these analyses led to particularly influential cases and thus made interpretation of the models more tenuous.

[29] The main effect of peer events did reach statistical significance in this logistic regression when one potentially influential case was removed.

approached statistical significance once cases with current Time 1 mood disorder were excluded. Thus, overall these results suggest an important role for recent peer events in risk for MDD. Notably, chronic stress in the peer or family domains did not significantly interact with recent peer events.

Supplemental analyses were run using stressful life events from 360 days before MDD onset for cases and before the interview for comparison participants in order to examine the effects of including more distal events. For the proportion of the sample eligible for these analyses 10 of 22 (45.5%) MDD cases had a stressful peer event and 105 of 423 (24.8%) comparison cases had a peer event ($\chi^2(1) = 4.65$, $p < .05$). Across analyses examining the interactions between peer events with either chronic family or chronic peer stress, the main effect for peer events was not significant beyond history of MDD and baseline symptom levels. Furthermore, there were no significant interactions between peer events and chronic stress in either the peer or family domains.

MDD cases (36.4%; 8 of 22) were also more likely than comparison cases (16.3%; 69 of 423) to have had a stressful family event during the 360 day period ($\chi^2(1) = 5.88$, Fisher's Exact $p < .05$). Family events from the prior year were significantly associated with increased risk for MDD in several analyses. When family events and chronic peer stress were examined, family events were significant beyond MDD history and baseline symptoms, only when participants with Time 1 mood disorder were excluded: (OR = 2.83; 95% CI: 1.07-7.51). In analyses examining family events and chronic family stress, family events were significant predictors of MDD beyond MDD history and baseline symptoms regardless of whether Time 1 current mood disorder cases were excluded, with odds ratios around 3 ($p < .05$). However, in none of the

analyses was the interaction of family events and chronic stress significant. Further, when analyses were run excluding one or two potentially influential cases, the effect of family events dropped below statistical significance.

Thus, there is some suggestion that recent peer events and more distal family events are associated with increased risk for MDD. However, there was no evidence supporting a buffering role for positive ongoing relationships on the impact of peer or family events.

Interactions Between Chronic Stress and Episodic Stress: Symptom Outcomes

Chronic stress and stressful life events were also examined as predictors of symptom level outcomes. Gender was not examined in these analyses because very few participants had recent events and regression diagnostics suggested that the inclusion of gender in interaction terms led to cases with concerning levels of influence on the regression coefficients. Out of 409 participants, 28 (5.8%) had a stressful family event and 35 (7.2%) had a stressful peer event in the 90 days prior to the Time 3 questionnaires. In the prediction of Time 3 depressive symptoms, significant main effects were found for peer and family chronic stress beyond the effects of baseline symptoms, but no main effects were found for recent life events in either the peer or family domains (Table 23). Furthermore, there were no significant interactions between chronic and episodic life stress in the prediction of Time 3 depressive symptoms. With regards to prediction of the depression symptom change score, neither main effects steps nor interaction steps were significant in any analyses.

Analyses examining the relationship between chronic and episodic stress in the prediction of follow-up anxiety symptoms are presented in Table 24. After entering baseline symptom levels on step 1, no steps containing main effects or interaction effects accounted for significant

increments in variance. Similarly, in analyses predicting the anxiety change score, no main effect or interaction steps were statistically significant.

DISCUSSION

This longitudinal study used both prospective and cross-sectional analyses to examine the relationships between chronic interpersonal stress and depression and anxiety outcomes. Further, the interactions between domains of chronic stress as well as between chronic and episodic stress were explored to ascertain whether positive relationships in one interpersonal domain might buffer against the negative effects of stress.

Individual Domains of Chronic Stress and Depressive Disorder

This study adds to the literature by documenting significant prospective associations between interview-based peer and family functioning with major depressive disorder in adolescents. In separate analyses, Time 1 close friendships, social group, and family relationships were all significant prospective predictors of MDD during follow-up, beyond prior history of depression. Using a more conservative analytical approach which covaried baseline depressive symptoms, only social group remained a significant predictor of increased risk for MDD. Whereas chronic stress variables were measured over the year prior to Time 1, it is certainly plausible that negative relationships could have led to increased levels of depressive symptoms at baseline which consequently increased risk for MDD during follow-up. Therefore, it may be prudent to consider negative relationships in any of those three domains as conferring risk for MDD, although results were most compelling for social group.

The most significant contribution of the present study was its simultaneous examination of four domains of chronic interpersonal stress. This author is unaware of another study that has prospectively examined the predictive utility of these four domains on internalizing outcomes.

Social group remained a significant predictor of increased risk for MDD during follow-up beyond the other domains. Importantly, although the chronic interpersonal stress composite was a significant predictor of MDD, deconstructing the composite highlighted the incremental validity of social group to overall risk.

It is important in interpreting these results to be cognizant of what was assessed by the social group variable. Unlike studies that have focused on peer rejection (e.g., Nolan et al., 2003) or peer victimization and peer group status (LaGreca & Harrison, 2005), the LSI focuses on the frequency of contact, size, harmony, and closeness of an adolescent's group of friends. Thus, this variable focuses on social group quality rather than on peer relationships in the larger school environment. It may be that examination of peer functioning in the larger school milieu may also be an important interpersonal variable to measure in predicting adjustment. Notwithstanding, the results of this study underscore the unique importance of social group quality on risk for depression in mid-late adolescence even after incorporating other interpersonal relationships into the model.

Individual Domains of Chronic Stress and Anxiety Disorder

Examination of individual domains of chronic interpersonal stress did not yield any significant findings for predicting anxiety disorders during follow-up. Very little prior work has examined the longitudinal relationship between chronic stress and anxiety disorders, although Rueter et al. (1999) found a significant relationship between parent-adolescent arguments and anxiety disorder onset. However, that study differed in important ways from the present study. First, Rueter et al. focused solely on parent-adolescent disagreements, whereas arguments are only one of many factors measured on the LSI. Second, their outcome was restricted to first

onsets of anxiety disorders, whereas in the present study 50% of the anxiety onset cases had a history of an anxiety disorder. Third, Rueter et al. used multiwave data, examining disagreements from years 1-3 as predictors of anxiety disorders during years 4-7 of the study. A fourth difference involves the make-up of the anxiety disorder groups in each study. In the Rueter et al. study, 26 (out of 303) participants developed an anxiety disorder. Including cases with more than one anxiety disorder, the frequency of diagnoses was as follows: social phobia: 15 participants (58%); specific phobia: 8 participants (31%); agoraphobia: 5 participants (19%); and panic attacks: 4 participants (15%). In the present study the distribution of anxiety disorders for the 20 cases was: agoraphobia without panic: 1 participant (5%); panic disorder: 1 participant (5%); OCD: 2 participants (10%); social phobia: 9 participants (45%); and specific phobia: 9 participants (45%).

These differences highlight two avenues for future study. First, it may be the case that the negative aspects of family (or other) relationships are uniquely relevant to anxiety disorder onset and thus aggregating across positive and negative aspects of relationships may not be advisable. Second, it will be important to examine whether particular domains of chronic interpersonal stress are related to specific anxiety disorders. For example, LaGreca and Harrison (2005) found that several interpersonal variables, such as being in a romantic relationship, belonging to a social group, experiences of peer victimization, and negative qualities of the best friend relationship, were all uniquely related to social anxiety symptoms.

Although no individual domain of Time 1 chronic interpersonal stress was significantly related to anxiety disorder onset during follow-up, analyses in which peer relationships were aggregated revealed some significant findings. Both the dyadic and peer composites were

prospective predictors of increased risk for anxiety disorder onset during follow-up beyond the effects of history of anxiety disorder and family relationships. These results supplement cross-sectional studies that have demonstrated significant associations between peer relationships and anxiety disorders (e.g., Goodyer et al., 1990).

Individual Domains of Chronic Interpersonal Stress and Symptoms

Prospective analyses revealed that social group functioning contributed small but significant variance to later symptoms of both anxiety and depression after accounting for baseline symptoms. Surprisingly, family relationships were not significantly related to Time 3 depressive symptoms beyond baseline symptom levels, which differs from prior longitudinal studies that have documented significant relationships between aspects of adolescents' relationships with parents and depressive symptoms (Rueter et al., 1999; Sheeber et al., 1997). However, Eberhart and Hammen (2006) reported that whereas family and peer relationships were significantly predictive of depressive symptoms at 6 month follow-up, effects were not significant at 1 and 2 year follow-ups. In the present study, the combination of the duration of follow-up (mean = 13 months) as well as the use of a global score for each interpersonal domain may have contributed to the absence of significant findings for family relationships.

Using a longitudinal design with cross-sectional analyses of individual domains of chronic stress, Time 3 close friendships, social group, family relationships, and romantic relationships ($p < .06$) accounted for small but significant variance in follow-up depressive symptoms. Examination of the predictive utility of the domains revealed that Time 3 family relationships were significantly associated with Time 3 depressive symptoms beyond baseline symptoms and the three peer domains. These findings add to the literature suggesting that

current/recent family functioning is importantly related to depressive symptomatology in adolescents (e.g., Sheeber et al., 2007). It is also important to note that the size of the effects of family relationships, even when analyzed individually (2% of variance), was small.

Across prospective and cross-sectional analyses, only social group made a small, but significant contribution to predicting Time 3 anxiety symptoms. One factor that may have worked against finding significant effects was using a composite of anxiety symptom measures that assessed social concerns, specific fears, agoraphobic fears, and preoccupation with bodily symptoms. It may be that particular domains of chronic interpersonal stress are more relevant to particular classes of symptoms, which is an area that merits further exploration. A second factor that may have worked against finding significant results was the stability of the anxiety symptoms across follow-up (r = .65). Although significant bivariate correlations were found between several domains of chronic interpersonal stress and Time 3 anxiety symptoms, these effects were non-significant after factoring in baseline symptom levels.

Buffering of Chronic Domains

Across all analyses examining the interaction of family and peer relationships, minimal support was found for buffering. That is, positive family (or peer) relationships did not significantly moderate the impact of negative peer (or family) relationships on depression or anxiety outcomes. The present study did find some limited support for a main effects model in that both Time 3 family and peer relationships were significantly associated with Time 3 depressive symptoms beyond Time 1 symptom levels. Thus, although there was no significant interaction between domains, the presence of good relationships in one area would be beneficial to the adolescent.

Several methodological factors may have played a role in the null findings. McClelland and Judd (1993) presented a comprehensive discussion of issues relevant to the difficulties of detecting moderating effects like those proposed by the buffering model. First, they noted that the effect sizes for interaction terms, even those that are statistically significant, are generally quite small. Second, the power to detect interaction effects is limited in non-experimental research relative to in experimental studies. Two aspects of the data that facilitate detecting an interaction effect are the presence of extreme values on each variable, and the co-occurrence of extreme values. Examining the frequency distributions across domains at Time 1 and Time 3, stress ratings at the top and bottom of the scales were relatively uncommon. For example, the top rating of 1 occurred in fewer than 2.5% of cases across all variables, and was especially rare for social group and family ratings where it occurred in less than .5% of cases. On the bottom extreme, fewer than 5% of cases were given a rating of 4, 4.5, or 5 on close friendships, social group, or romantic relationships. Although about 10% of cases received a rating of 4 or higher on family relationships, only 1% received a score of 4.5 or 5. Thus, the paucity of values rated at the top and bottom ends of the scale may have impaired the ability to detect interaction effects.

Another factor relevant to the absence of significant interaction effects may be the reliability of the predictors. Cohen, Cohen, West and Aiken (2003) noted how the unreliability of predictors attenuates their relationship with outcome variables; this attenuation is compounded in interaction terms which multiply the two predictors. This study used either a single domain score (e.g., family relationships) or a composite of domains (e.g., close friendships and romantic relationships) in interaction terms. For the dyadic and peer composite variables, internal consistency coefficients were relatively low ($\alpha < .60$). Measures of reliability for individual

domains within and across sites ranged from fair to good. Thus, the interaction terms were comprised of only moderately reliable variables, which substantially affected power to detect interaction effects.

A related point is that the LSI combines a number of factors into a single domain score. For example, social group incorporates frequency of contact, range of activities, size of social group, closeness of social group, and amount of conflict into a single score. Typically, studies have either focused only on negative aspects of interpersonal relationships (e.g., Nolan et al., 2003; Rueter et al., 1999) or have separated positive and negative aspects of relationships, rather than generating global scores of relationship quality (e.g., LaGreca & Harrison, 2005; Sheeber et al., 2007). It might be that the LSI is more appropriate for examining main effects of relationship quality, than for addressing buffering since a single domain rating involves aggregating across several positive and negative aspects of a relationship.

Future studies investigating the nature of the interactions between different interpersonal relationships should attend to several methodological and theoretical considerations. First, interaction effects, if present, are very likely to be small (McClelland & Judd, 1993). Thus, using highly reliable measures will greatly facilitate the detection of moderation effects. Second, if interaction effects are found, it will be important to fully examine the moderating effects of both predictors. It may be the case that one domain (e.g., good peer relationships) buffers the effects of another domain (e.g., poor family relationships), but not the reverse (as found in the present study). Third, the present study only examined the interaction between family and peer domains. However, it would be informative for future studies to examine the interactions between different types of peer relationships, such as between dyadic relationships and larger

social group relationships. Finally, it is important for researchers to consider how they think buffering between (chronic) interpersonal domains might occur. If buffering effects between interpersonal domains are found, do we believe they are facilitated by frequency of social contact, by availability of support, by actual *provision* of regular support, or even by resources that help the participant actively cope with chronic stressors in the other domain? Focusing on potential *causal* mechanisms by which buffering might possibly work (e.g., actual provision of support) will greatly aid study design and applicability of results from future endeavors in this area of research.

The Buffering Hypothesis: Chronic and Episodic Stress

Results from this study demonstrated that recent peer episodic events and more distal family episodic events were associated with increased risk for Major Depressive Disorder during follow-up. These findings contribute to the relatively small literature that has used interview-based assessment of life events in longitudinal assessment of depression in adolescents (Grant et al., 2004). Notwithstanding, a major objective of this study was to examine whether there was any evidence to support the stress-buffering hypothesis (e.g., Cohen & Wills, 1985). Across all analyses, chronic stress did not significantly moderate the relationship between life events and depression or anxiety.

Several factors may have mitigated against finding significant interactions in this study. First, as noted above, the reliability of the chronic stress variables was moderate at best, thus contributing to unreliability of the interaction term and decreasing power to detect significant interactions. Second, because only a subset of life events was examined in each analysis, they were rare occurrences. Whereas only about 10% of the sample had a recent family or peer event,

very few cases were relevant for examining possible moderation effects of relationship quality. Third, this study used a cutoff of mild severity for events. It may be the case that social support is more relevant as a stress buffer after the occurrence of more severe events that tax the individual's internal coping resources. The role of event severity as a relevant variable in examination of buffering is an empirical issue that merits further study. Finally, the infrequency of events led to exclusion of gender from buffering analyses which may be an important moderator of buffering effects.

In their review of the literature on the social support buffering hypothesis, Cohen and Wills (1985) noted that studies which found support for the buffering hypothesis tended to focus on perceived availability of support. This seemingly important variable was not explicitly measured in this study. As part of the assessment of episodic stressors done here, participants were asked if support was available during the time of an event. However, rather than factoring in the availability of support into the contextual threat rating, it would be better utilized as a separate measure (Kessler, 1997).

Gender Effects

As expected, women had higher rates of MDD and anxiety disorder during follow-up. However, main effects of gender on symptom outcomes were not significant. Furthermore, no support was found for gender differences in reactivity to chronic interpersonal stress. That is, women were not more likely than men to experience depression or anxiety given similar levels of chronic stress. These results differ from the cross-sectional results of Shih et al. (2006) who found that boys were more reactive to social group stress, and from Rudolph (2002) who found that girls were more reactive to overall peer stress. The findings from the present study

contribute to the small number of studies that have examined gender reactivity in the context of chronic interpersonal stress in an adolescent sample.

A major methodological factor that may have influenced gender findings in this study involves the selected sample. It is important to remember that participants scoring in the top third on the neuroticism questionnaire were over-recruited into the study. In the screening sample, the high-risk cutoff score fell at the 75th percentile for men and the 59th percentile for women. Thus, slightly over 50% of the males in this study were sampled from the top 25% of the male distribution on neuroticism. Neuroticism was associated with higher levels of depression and anxiety symptoms at both Time 1 and Time 3 for both genders. At Time 3, the high risk men had even higher levels of depressive symptoms than the high N women. Gender differences that may be expected in community samples may not have been replicated in our sample because it contained a higher proportion of high N men than would be contained in an unselected sample.

Although other studies have also not found significant gender x domain interactions for best friendship and romantic relationships (LaGreca & Harrison, 2005) or family relationships (Sheeber et al., 2007), it was predicted that men would be more reactive to disturbances in their social group than women (Shih et al., 2006). Close inspection of the LSI suggests that the social group rating may not appropriately get at the different interdependent foci prioritized by men (collective) and women (relational; Gabriel & Gardner, 1999). For example, a participant who has a large group of friends, frequent social activities and little conflict, might get a similar rating to a participant with a smaller group of several (3-4) intimate friends who also see each other regularly and do not have substantial conflict. Both group size and closeness are factored into

the rating and so the global social score does not clearly distinguish close bonds from larger group membership. This distinction is likely to be an important one, and future studies may benefit from assessment measures that can make such a differentiation.

General Methodological Concerns

Several other issues and limitations of the present study deserve mention. First, an overarching goal of this study was to take a nuanced examination of life stress variables. This exclusive focus on stress variables revealed findings that, even when statistically significant, were small. Crucial to any comprehensive model of psychopathology is to look at stress in the context of pre-existing diatheses (Monroe & Simons, 1991). For example, Rizzo et al. (2006) found that chronic romantic stress and episodic romantic stress each significantly interacted with a measure of interpersonal sensitivity to predict depression diagnostic status over 6 month follow-up. Prinstein and Aikins (2004) found a significant three-way interaction between gender, social preference (peer acceptance/rejection measured by peer nomination), and self-reported importance of peer acceptance. The interaction revealed that social preference was significantly associated with depressive symptoms in women when they had high investment in being popular.

Second, this study only examined a one directional relationship between stress and anxiety and depression. However, it is likely that the relationship between stress and depression, and possibly anxiety, is bidirectional and transactional over time. Support has been found for stress generation models (Hammen, 1991) in which depression is associated with later stressful events (e.g., Cole, Nolen-Hoeksema, Girgus, & Paul, 2006; Hankin et al., 2007).

Third, this study focused only on interpersonal stress. Although there were sound empirical and theoretical reasons for examining interpersonal stress, additional areas of the adolescent experience may be useful to explore. For example, successes or disappointments related to academic or athletic achievement may well be relevant to adolescent adjustment (e.g., Hankin et al., 2007).

Fourth, fewer significant effects were found between stress and anxiety outcomes. It may be the case that life stress plays a less important role in precipitating anxiety outcomes than it does for depression. However, it may also be the case that the types of events or domains of chronic stress measured in the present study did not adequately sample the types of life stress that may be most relevant to anxiety outcomes. For example, danger events have been shown to be associated with anxiety outcomes (Finlay-Jones & Brown, 1981; Kendler et al., 2003). Furthermore, anxiety disorders and symptoms were looked at as a group. It would be advisable for future studies to examine whether particular classes of symptoms and disorders are related to particular types of chronic and episodic stress.

Finally, symptoms of anxiety and depression were moderately stable over follow-up. Using a relatively novel statistical approach called trait-state-error model (TSE model; Kenny & Zautra, 1995), Cole et al. (2006) examined the relationship between life events and depressive symptoms in two samples, one with six waves of data collection and one with 12 waves of data collection. The TSE approach allows a construct like depressive symptoms to be broken into trait and state components, the latter of which would be expected to change over time (Cole et al, 2006). The most relevant finding of Cole et al. to the present discussion was that effect sizes for life events were larger when focusing on state components of depressive symptoms. Although

the present study had only two waves of data and thus could not use such a methodology, it may be a viable approach for subsequent studies using this sample.

Summary and Conclusions

This study focused on the relationships between interpersonal domains of chronic and episodic stress with depression and anxiety. In prospective analyses examining unique predictive validity, social group at Time 1 was a significant predictor of risk for MDD during follow-up even after accounting for the effects of other important interpersonal relationships. In cross-sectional analyses, Time 3 family relationships were uniquely related to Time 3 depressive symptoms. These unique contributions suggest that future studies should continue to examine specific types of interpersonal relationships rather than relying solely on a composite of chronic interpersonal stress. Such a nuanced approach may help elucidate key targets for psychosocial interventions.

Relatively fewer significant associations were found between chronic interpersonal stress and anxiety, although composites of peer relationships were significant prospective predictors of risk for anxiety disorders. Future studies should examine different anxiety disorders (and symptoms) separately to establish whether interpersonal relationships are more relevant as risk factors for certain types of anxiety.

Several methodological limitations may have contributed to the null findings involving stress-buffering and stress-reactivity. Further explorations of both of these areas with methodologies more appropriate to address those questions are merited. The goal of such endeavors would be to attempt to identify particular interpersonal domains, for one or both genders, that could be important targets for intervention.

Results of this study support the continued exploration of chronic interpersonal stress as a risk factor for depression (and anxiety) in adolescents. Future studies of chronic stress should examine chronic stress in conjunction with both episodic stressors and diatheses (e.g., neuroticism, parental depression, rumination, cognitive style). That multi-faceted approach may provide a richer picture of the relationship between interpersonal functioning and adjustment in adolescents.

Table 1

Means, Standard Deviations, and Comparison of Symptom Measures at Time 1 and Time 3

Measure	Time 1 Mean (SD)	Time 3 Mean (SD)	T-test
IDD	.53 (.42)	.38 (.35)	$t(421) = 7.98$**
MASQ-Gen Dep	2.12 (.86)	1.89 (.76)	$t(430) = 5.84$**
MASQ-Anhed Dep	2.69 (.65)	2.57 (.63)	$t(431) = 4.02$**
MASQ-Gen Anx	1.81 (.64)	1.67 (.60)	$t(431) = 5.03$**
MASQ-Anx Aro	1.49 (.54)	1.38 (.47)	$t(430) = 4.61$**
SPS	.98 (.71)	.89 (.71)	$t(407) = 2.98$**
SFQ	.98 (.85)	.88 (.86)	$t(403) = 2.44$*
FSS	1.84 (1.02)	1.54 (1.02)	$t(428) = 7.28$**

Note. Mean values represent mean item scores. IDD = Inventory to Diagnose Depression; MASQ-Gen Dep = Mood and Anxiety Symptom Questionnaire General Depression; MASQ-Anhed Dep = Anhedonic Depression; MASQ-Gen Anx = General Anxiety; MASQ-Anx Aro = Anxious Arousal; SPS = Social Phobia Scale; SFQ = Situational Fears Questionnaire; FSS = Fear Survey Schedule.
* $p < .05$. ** $p < .01$.

Table 2

Means, Standard Deviations, and Comparison of Chronic Interpersonal Stress and Symptom Measures Between Male and Female Participants

Measure	Males Mean (SD)	Females Mean (SD)	T-test
T1 Close Friendship	2.21 (.60)	2.05 (.63)	$t(484) = 2.59^{**}$
T1 Social Group	2.30 (.73)	2.33 (.62)	$t(484) = -.47$
T1 Romantic	2.29 (.56)	2.39 (.62)	$t(484) = -1.58$
T1 Family	2.63 (.68)	2.73 (.77)	$t(484) = -1.24$[a]
T3 Close Friendship	2.13 (.76)	2.01 (.71)	$t(484) = 1.80^{t}$
T3 Social Group	2.26 (.68)	2.27 (.61)	$t(484) = -.19$
T3 Romantic	2.30 (.53)	2.26 (.57)	$t(484) = .66$
T3 Family	2.59 (.71)	2.62 (.70)	$t(484) = -.42$
T1 Depression Comp	-.03 (.87)	.02 (.87)	$t(477) = -.55$
T3 Depression Comp	.06 (.91)	-.03 (.84)	$t(433) = .98$
T1 Anxiety Comp	-.12 (.78)	.05 (.69)	$t(473) = -2.38^{*}$
T3 Anxiety Comp	-.09 (.76)	.05 (.71)	$t(411) = -1.81^{t}$

[a] The two groups in this comparison did not have equal variances. Equal variances not assumed did not substantially alter these findings, so results are presented without adjusted degrees of freedom.

$^{t}p < .10$. $^{*}p < .05$. $^{**}p < .01$.

Table 3

Correlations Among Time 1 Chronic Interpersonal Stress Measures and Depression and Anxiety Outcomes

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1. Gender																		
2. MDD Hx	.08																	
3. Anx Hx	-.01	.25																
4. MDD onset	.12	.22	.09															
5. Anxiety onset	.08	.07	.19	-														
6. T1 Close Friend	-.12	.15	.09	.12	.09													
7. T1 Social Group	.02	.18	.11	.21	.08	.48												
8. T1 Romantic	.07	.26	.20	.09	.11	.23	.28											
9. T1 Family	.06	.33	.20	.17	.03	.24	.31	.33										
10. T1 Dyadic	-.03	.26	.18	.13	.13	.79	.49	.79	.36									
11. T1 Peer	-.01	.27	.18	.19	.13	.77	.79	.68	.39	.92								
12. T1 LSI Composite	.01	.33	.21	.21	.11	.70	.74	.66	.67	.87	.94							
13. T1 Dep Comp	.03	.31	.39	.22	.15	.17	.25	.35	.37	.33	.34	.41						
14. T3 Dep Comp	-.05	.17	.26	.27	.26	.15	.20	.20	.22	.22	.25	.28	.52					
15. Dep Change Score	-.06	-.18	-.17	.04	.06	-.04	-.05	-.19	-.16	-.14	-.13	-.16	-.58	.40				
16. T1 Anx Comp	.11	.20	.35	.15	.13	.05	.11	.27	.20	.20	.19	.22	.62	.39	-.31			
17. T3 Anx Comp	.09	.14	.24	.11	.25	.05	.13	.15	.13	.12	.15	.16	.41	.65	.17	.65		
18. Anx Change Score	.01	-.13	-.13	.00	.12	-.01	.06	-.11	-.07	-.08	-.03	-.05	-.29	.26	.56	-.47	.37	
N	486	486	485	468	450	486	486	486	486	486	486	486	479	435	432	475	413	407

Note. N represents the number of valid values for that variable. Gender coded as 0 = male 1= female, MDD Hx = History of Current or Past Major Depressive Disorder at Time 1 interview, Anx Hx = History of Current or Past Anxiety Disorder at Time 1 interview, T1 Dep Comp = Time 1 Depression Symptom Composite, T3 Dep Comp = Time 3 Depression Symptom Composite, Dep Change Score = Change in Depressive Symptoms (Time 3 – Time 1), T1 Anx Comp = Time 1 Anxiety Symptom Composite, Time 3 Anx Comp = Time 3 Anxiety Symptom Composite, Anx Change Score = Change in Anxiety Symptoms (Time 3 – Time 1).

All correlation coefficients $r > |.09|$ are significant at the $p < .05$ level; $r > |.15|$ are significant at the $p < .001$ level.

Table 4

Individual Domains at Time 1 Predicting Major Depressive Disorder

Time 1 Close Friendships

Type of Analysis	B	S.E.	Wald	Exp(B)	95% C.I
1. Dep History Term Only	.32	.16	4.06	1.38*	1.01-.1.89
2. Dep History and Time 1 Dep Comp	.21	.17	1.64	1.24	.89-1.72
3. Dep History, Time 1 Dep Comp, Excluding Current MDD	.26	.18	2.01	1.29	.91-1.84

Note. Coefficient information refers only to the life stress domain. Analyses were run three different ways.
* $p < .05$.

Time 1 Social Group

Type of Analysis	B	S.E.	Wald	Exp(B)	95% C.I
1. Dep History Term Only	.55	.15	13.18	1.74**	1.29-2.35
2. Dep History and Time 1 Dep Comp	.42	.16	6.64	1.52**	1.11-2.09
3. Dep History, Time 1 Dep Comp, Excluding Current MDD	.48	.17	7.95	1.62**	1.16-2.27

** $p < .01$.

Time 1 Romantic Relationships

Type of Analysis	B	S.E.	Wald	Exp(B)	95% C.I.
1. Dep History Term Only	.12	.17	.52	1.13	.81-1.59
2a. Dep History and Time 1 Dep Comp: First Onsets[a]	.28	.26	1.20	1.33	.80-2.19
2b. Recurrences[a]	-.37	.28	1.78	.69	.40-1.19
3. Dep History, Time 1 Dep Comp, excluding Current MDD[b]	-.07	.20	.13	.93	.63-1.38

[a] Interaction of romantic stress x depression history was statistically significant in the full model.
[b]Interaction step for this analysis was significant. However, further analyses were not run because the interaction was investigated in prior analyses (2a. and 2b.).

Time 1 Family Relationships

Type of Analysis	B	S.E.	Wald	Exp(B)	95% C.I.
1. Dep History Term Only	.46	.19	5.88	1.58*	1.09-2.29
2. Dep History and Time 1 Dep Comp	.28	.20	1.90	1.32	.89-1.97
3. Dep History, Time 1 Dep Comp, excluding Current MDD	.32	.21	2.29	1.38	.91-2.10

* $p < .05$.

Table 5

Individual Domains at Time 1 Predicting Anxiety Disorder

Time 1 Close Friendships

	Step χ^2	B	S.E.	Wald	Exp(B)	95% C.I.
1. Anxiety History	14.05**	1.64	.47	12.12	5.16**	2.05-12.98
Close Friendships		.27	.20	1.90	1.32	.89-1.94
2. Interaction	.19	-.17	.40	.19	.84	.39-1.83

Note. Chi-square refers to the increment in model improvement by the full step.
**$p < .01$.

Time 1 Social Group

	Step χ^2	B	S.E.	Wald	Exp(B)	95% C.I.
1. Anxiety History	13.77**	1.64	.47	12.01	5.14**	2.04-12.96
Social Group		.26	.21	1.58	1.30	.87-1.94
2. Interaction	1.38	-.48	.41	1.37	.62	.28-1.38

**$p < .01$.

Time 1 Romantic Relationships

	Step χ^2	B	S.E.	Wald	Exp(B)	95% C.I.
1. Anxiety History	14.88**	1.54	.48	10.42	4.67**	1.83-11.91
Romantic Relationships		.37	.22	2.69	1.44	.93-2.24
2. Interaction	.06	-.11	.45	.06	.90	.37-2.16

**$p < .01$.

Time 1 Family Relationships

	Step χ^2	B	S.E.	Wald	Exp(B)	95% C.I.
1. Anxiety History	12.29**	1.71	.48	12.63	5.52**	2.15-14.15
Family Relationships		.00	.25	.00	1.00	.62-1.62
2. Interaction	.10	-.16	.50	.10	.85	.32-2.28

**$p < .01$.

Table 6

Individual Domains at Time 1 Predicting Depression Symptoms: Regressed Change

Time 1 Close Friendships

	Step Δ R^2	β	pr^2
1.Gender	.28**	-.05	.00
Time 1 Depression Composite		.51**	.26
Close Friendships		.05	.00
2. Gender X Close Friendship	.00	.08	.00

**$p < .01$.

Time 1 Social Group

	Step Δ R^2	β	pr^2
1.Gender	.28**	-.06	.00
Time 1 Depression Composite		.50**	.24
Social Group		.09*	.01
2. Gender X Social Group	.00	.01	.00

* $p < .05$. **$p < .01$.

Time 1 Romantic Relationships

	Step Δ R^2	β	pr^2
1.Gender	.27**	-.06	.00
Time 1 Depression Composite		.51**	.23
Romantic Relationships		.02	.00
2. Gender X Romantic Relationship	.00	.12	.00

**$p < .01$.

Time 1 Family Relationships

	Step Δ R^2	β	pr^2
1.Gender	.28**	-.06	.00
Time 1 Depression Composite		.50**	.22
Family Relationships		.05	.00
2. Gender X Family Relationships	.00	.02	.00

**$p < .01$.

Table 7

Individual Domains at Time 1 Predicting Anxiety Symptoms: Regressed Change

Time 1 Close Friendships

	Step Δ R^2	β	pr^2
1.Gender	.42**	.03	.00
Time 1 Anxiety Composite		.64**	.41
Close Friendships		.01	.00
2. Gender X Close Friendships	.00	-.01	.00

**p < .01.

Time 1 Social Group

	Step Δ R^2	β	pr^2
1.Gender	.43**	.03	.00
Time 1 Anxiety Composite		.64**	.40
Social Group		.08*	.01
2. Gender X Social Group	.00	-.07	.00

* p < .05. **p < .01.

Time 1 Romantic Relationships

	Step Δ R^2	β	pr^2
1.Gender	.42**	.03	.00
Time 1 Anxiety Composite		.64**	.39
Romantic Relationships		.00	.00
2. Gender X Romantic Relationships	.00	.06	.00

**p < .01.

Time 1 Family Relationships

	Step Δ R^2	β	pr^2
1.Gender	.42**	.03	.00
Time 1 Anxiety Composite		.64**	.39
Family Relationships		.01	.00
2. Gender X Family Relationships	.00	-.03	.00

**p < .01.

Table 8

Models of all Time 1 Chronic Domains Predicting Major Depressive Disorder

Order of Entry	Step χ^2	Step R^2	B	S.E.	Wald	Exp(B)	95% C.I.
1. Depression History	18.78**	.09	1.56	.35	19.91	4.73**	2.39-9.37
2. Close Friendships	15.57**	.07	.07	.18	.14	1.07	.75-1.54
Social Group			.48	.18	7.25	1.62**	1.14-2.30
Romantic Relationships			-.08	.18	.19	.92	.64-1.32
Family Relationships			.33	.20	2.84	1.40^t	.95-2.06

Note. Chi-square refers to the increment in model improvement by the full step. Step R^2 refers to change in Nagelkerke R^2 values for that step.
$^t p < .10$. * $p < .05$. ** $p < .01$.

Order of Entry	Step χ^2	Step R^2	B	S.E.	Wald	Exp(B)	95% C.I.
1. Depression History	31.07**	.15	1.24	.38	10.82	3.46**	1.65-7.24
T1 Dep Composite			.64	.20	10.70	1.90**	1.29-2.79
2. Close Friendships	8.02^t	.04	.04	.19	.05	1.04	.72-1.51
Social Group			.39	.18	4.57	1.48*	1.03-2.12
Romantic Relationships			-.16	.19	.69	.85	.58-1.25
Family Relationships			.23	.21	1.22	1.26	.84-1.90

$^t p < .10$. * $p < .05$. ** $p < .01$.

Current T1 MDD excluded

Order of Entry	Step χ^2	Step R^2	B	S.E.	Wald	Exp(B)	95% C.I.
1. Depression History	27.13**	.15	1.14	.40	8.04	3.12**	1.42-6.90
T1 Dep Composite			.76	.21	13.14	2.14**	1.42-3.23
2. Close Friendships	9.77*	.05	.07	.20	.12	1.07	.72-1.60
Social Group			.45	.19	5.28	1.56*	1.07-2.28
Romantic Relationships			-.20	.21	.93	.82	.54-1.23
Family Relationships			.27	.22	1.52	1.31	.85-2.02

* $p < .05$. ** $p < .01$.

Table 9

Model of all Time 1 Chronic Domains Predicting Anxiety Disorder

Order of Entry	Step χ^2	Step R^2	B	S.E.	Wald	Exp(B)	95% C.I.
1. Anxiety History	12.29**	.09	1.71	.47	13.38	5.52**	2.21.13.77
2. Close Friendships	4.46	.03	.20	.23	.72	1.22	.77-1.93
Social Group			.14	.25	.30	1.15	.71-1.86
Romantic Relationships			.35	.24	2.16	1.42	.89-2.26
Family Relationships			-.17	.26	.43	.84	.50-1.41

Note. Chi-square refers to the increment in model improvement by the full step. Step R^2 refers to change in Nagelkerke R^2 values for that step.
**$p < .01$.

Table 10

Models of all Time 1 Chronic Domains Predicting Depression Symptoms: Regressed Change

Four Domains of Chronic Stress

	Step ΔR^2	β	pr^2
1.Time 1 Depression Composite	.27**	.52**	.27
2. Close Friendships	.01	.03	.00
Social Group		.07	.00
Romantic Relationships		-.01	.00
Family Relationships		.03	.00

**$p < .01$.

Chronic Interpersonal Composite

	Step ΔR^2	β	pr^2
1.Time 1 Depression Composite	.27**	.52**	.27
2. Interpersonal Chronic Composite	$.01^t$	$.08^t$	.01

$^t p < .10$. **$p < .01$.

Table 11

Models of all Time 1 Chronic Domains Predicting Anxiety Symptoms: Regressed Change

Four Domains of Chronic Stress

	Step ΔR^2	β	pr^2
1.Time 1 Anxiety Composite	.42**	.65**	.42
2. Close Friendships	.01	-.04	.00
Social Group		.11*	.01
Romantic Relationships		-.02	.00
Family Relationships		-.01	.00

* $p < .05$. ** $p < .01$.

Chronic Interpersonal Composite

	Step ΔR^2	β	pr^2
1.Time 1 Anxiety Composite	.42**	.65**	.42
2.Time 1 Chronic Composite	.00	.04	.00

** $p < .01$.

Table 12

Individual Domains at Time 3 Predicting Depression Symptoms: Regressed Change

Time 3 Close Friendships

	Step ΔR^2	β	pr^2
1.Gender	.29**	-.05	.00
Time 1 Depression Composite		.50**	.24
Close Friendships		.12**	.01
2. Gender X Close Friendship	.00	.00	.00

**p < .01.

Time 3 Social Group

	Step ΔR^2	β	pr^2
1.Gender	.29**	-.06	.00
Time 1 Depression Composite		.49**	.23
Social Group		.12**	.01
2. Gender X Social Group	.00	-.04	.00

**p < .01.

Time 3 Romantic Relationships

	Step ΔR^2	β	pr^2
1.Gender	.28**	-.05	.00
Time 1 Depression Composite		.50	.24
Romantic Relationships		.08^t	.01
2. Gender X Romantic Relationships	.00	-.06	.00

$^t p < .10$. **p < .01.

Time 3 Family Relationships

	Step ΔR^2	β	pr^2
1.Gender	.29**	-.06	.00
Time 1 Depression Composite		.48**	.22
Family Relationships		.15**	.02
2. Gender X Family Relationships	.00	.05	.00

**p < .01.

Table 13

Models of all Time 3 Chronic Domains Predicting Depression Symptoms: Regressed Change

Four Domains of Chronic Stress

	Step ΔR^2	β	pr^2
1.Time 1 Depression Composite	.27**	.52**	.27
2. Time 3 Close Friendships	.03**	.08[t]	.01
Time 3 Social Group		.06	.00
Time 3 Romantic Relationships		.05	.00
Time 3 Family Relationships		.11**	.01

[t] $p < .10$. ** $p < .01$.

Time 3 Chronic Interpersonal Composite

	Step ΔR^2	β	pr^2
1.Time 1 Depression Composite	.27**	.52**	.27
2. Chronic Composite	.03**	.19**	.03

** $p < .01$.

Table 14

Individual Domains at Time 3 Predicting Anxiety Symptoms: Regressed Change

Time 3 Close Friendships

	Step Δ R^2	β	pr^2
1.Gender	.42**	.04	.00
Time 1 Anxiety Composite		.64**	.40
Close Friendship		.04	.00
2. Gender X Close Friendships	.00	-.01	.00

**p < .01.

Time 3 Social Group

	Step Δ R^2	β	pr^2
1.Gender	.42**	.03	.00
Time 1 Anxiety Composite		.64**	.39
Social Group		.01	.00
2. Gender X Social Group	.00	-.08	.00

**p < .01.

Time 3 Romantic Relationships

	Step Δ R^2	β	pr^2
1.Gender	.42**	.03	.00
Time 1 Anxiety Composite		.64**	.40
Romantic Relationships		.03	.00
2. Gender X Romantic Relationships	.00	-.05	.00

**p < .01.

Time 3 Family Relationships

	Step Δ R^2	β	pr^2
1.Gender	.42**	.03	.00
Time 1 Anxiety Composite		.64**	.39
Family Relationships		.01	.00
2. Gender X Family Relationships	.00	.00	.00

**p < .01.

Table 15

Interactions Between Time 1 Chronic Domains Predicting Major Depressive Disorder

Time 1 Dyadic Composite and Family Relationships

Order of Entry	Step χ^2	Step R^2	B	S.E.	Wald	Exp(B)	95% C.I.
1. Depression History	33.15**	.16	1.08	.39	7.62	2.95**	1.37-6.36
T1 Dep Composite			.53	.21	6.44	1.71*	1.13-2.58
Dyadic Composite			.10	.24	.16	1.10	.69-1.77
Family Relationships			.26	.21	1.58	1.30	.86-1.96
2. Dyadic X Family	.79	.00	.20	.22	.80	1.22	.79-1.88

Note. Chi-square refers to the increment in model improvement by the full step. Step R^2 refers to change in Nagelkerke R^2 values for that step.
* $p < .05$. ** $p < .01$.

Time 1 Social Group and Family Relationships

Order of Entry	Step χ^2	Step R^2	B	S.E.	Wald	Exp(B)	95% C.I.
1. Depression History	38.37**	.19	1.07	.40	7.28	2.91**	1.34-6.31
T1 Dep Composite			.45	.21	4.49	1.57*	1.03-2.38
Social Group			.39	.17	5.66	1.48*	1.07-2.05
Family Relationships			.20	.21	.95	1.22	.82-1.84
2. Social X Family	.56	.00	.12	.16	.55	1.13	.83-1.53

* $p < .05$. ** $p < .01$.

Time 1 Peer Composite and Family Relationships

Order of Entry	Step χ^2	Step R^2	B	S.E.	Wald	Exp(B)	95% C.I.
1. Depression History	35.04**	.17	1.04	.39	6.96	2.82**	1.31-6.09
T1 Dep Composite			.48	.21	4.98	1.61*	1.06-2.45
Peer Composite			.37	.25	2.10	1.45	.88-2.38
Family Relationships			.21	.21	.98	1.23	.81-1.87
2. Peer X Family	.84	.00	.21	.23	.84	1.23	.79-1.92

* $p < .05$. ** $p < .01$.

Table 16

Interactions Between Time 1 Chronic Domains Predicting Anxiety Disorder

Time 1 Dyadic Composite and Family Relationships

Order of Entry	Step χ^2	Step R^2	B	S.E.	Wald	Exp(B)	95% C.I.
1. Anxiety History	16.35**	.12	1.57	.48	10.44	4.79**	1.85-12.39
Dyadic Composite			.61	.30	4.19	1.84*	1.03-3.29
Family			-.15	.26	.31	.86	.52-1.44
2. Dyadic X Family	.61	.00	.22	.27	.64	1.24	.73-2.12

Note. Chi-square refers to the increment in model improvement by the full step. Step R^2 refers to change in Nagelkerke R^2 values for that step.
* $p < .05$. ** $p < .01$.

Time 1 Social Group and Family Relationships

Order of Entry	Step χ^2	Step R^2	B	S.E.	Wald	Exp(B)	95% C.I.
1. Anxiety History	13.84**	.10	1.66	.48	11.85	5.27**	2.05-13.58
Social Group			.27	.21	1.66	1.31	.87-1.98
Family Relationships			-.07	.25	.07	.94	.57-1.52
2. Social X Family	.27	.00	.10	.19	.27	1.10	.76-1.61

** $p < .01$.

Time 1 Peer Composite and Family Relationships

Order of Entry	Step χ^2	Step R^2	B	S.E.	Wald	Exp(B)	95% C.I.
1. Anxiety History	16.33**	.12	1.57	.49	10.41	4.80**	1.85-12.45
Peer Composite			.64	.31	4.16	1.89*	1.03-3.48
Family Relationships			-.16	.26	.36	.86	.52-1.42
2. Peer X Family	.45	.00	.18	.26	.46	1.20	.72-2.00

* $p < .05$. ** $p < .01$.

Table 17

Interactions Between Time 1 Chronic Domains Predicting Depression Symptoms: Regressed Change

Time 1 Dyadic Composite and Family Relationships

	Step ΔR^2	β	pr^2
1. Time 1 Depression Composite	.27**	.52**	.27
Gender		-.06	.00
2. Dyadic Composite	.00	.04	.00
Family Relationships		.04	.00
3. Dyadic X Gender	.01	.14[t]	.01
Family X Gender		-.02	.00
Dyadic X Family		-.06	.00
4. Dyadic X Family X Gender	.00	-.04	.00

[t] $p < .10$. ** $p < .01$.

Time 1 Social Group and Family Relationships

	Step ΔR^2	β	pr^2
1. Time 1 Depression Composite	.27**	.52**	.27
Gender		-.06	.00
2. Social Group	.01	.08[t]	.01
Family Relationships		.03	.00
3. Social X Gender	.00	.01	.00
Family X Gender		.03	.00
Social X Family		-.06	.00
4. Social X Family X Gender	.00	.11	.00

[t] $p < .10$. ** $p < .01$.

Time 1 Peer Composite and Family Relationships

	Step ΔR^2	β	pr^2
1. Time 1 Depression Composite	.27**	.52**	.27
Gender		-.06	.00
2. Peer Composite	.01	.07	.00
Family Relationships		.03	.00
3. Peer X Gender	.01	.10	.00
Family X Gender		-.01	.00
Peer X Family		-.07	.00
4. Peer X Family X Gender	.00	.02	.00

** $p < .01$.

Table 18

Interactions Between Time 1 Chronic Domains Predicting Anxiety Symptoms: Regressed Change

Time 1 Dyadic Composite and Family Relationships

	Step Δ R^2	β	pr^2
1. Time 1 Anxiety Composite	.42**	.64**	.41
Gender		.03	.00
2. Dyadic Composite	.00	.00	.00
Family Relationships		.01	.00
3. Dyadic X Gender	.00	.04	.00
Family X Gender		-.05	.00
Dyadic X Family		.00	.00
4. Dyadic X Family X Gender	.00	-.08	.00

**$p < .01$.

Time 1 Social Group and Family Relationships

	Step Δ R^2	β	pr^2
1. Time 1 Anxiety Composite	.42**	.64**	.41
Gender		.03	.00
2. Social Group	.01[t]	.09[*]	.01
Family Relationships		-.02	.00
3. Social X Gender	.00	-.06	.00
Family X Gender		-.01	.00
Social X Family		-.04	.00
4. Social X Family X Gender	.00	.06	.00

[t]$p < .10$. *p < .05. **$p < .01$.

Time 1 Peer Composite and Family Relationships

	Step Δ R^2	β	pr^2
1. Time 1 Anxiety Composite	.42**	.64**	.41
Gender		.03	.00
2. Peer Composite	.00	.04	.00
Family Relationships		-.01	.00
3. Peer X Gender	.00	.00	.00
Family X Gender		-.04	.00
Peer X Family		-.02	.00
4. Peer X Family X Gender	.00	-.03	.00

**$p < .01$.

Table 19

Interactions Between Time 3 Chronic Domains Predicting Depression Symptoms: Regressed Change

Time 3 Dyadic Composite and Family Relationships

	Step Δ R^2	β	pr^2
1. Time 1 Depression Composite	.27**	.52**	.27
Gender		-.06	.00
2. Dyadic Composite	.03**	.11*	.01
Family Relationships		.12**	.01
3. Dyadic X Gender	.00	-.05	.00
Family X Gender		.06	.00
Dyadic X Family		.01	.00
4. Dyadic X Family X Gender	.00	.11	.00

*p < .05. **p < .01.

Time 3 Social Group and Family Relationships

	Step Δ R^2	β	pr^2
1. Time 1 Depression Composite	.27**	.52**	.27
Gender		-.06	.00
2. Social Group	.03**	.09*	.01
Family Relationships		.13**	.01
3. Social X Gender	.01	-.07	.00
Family X Gender		.06	.00
Social X Family		-.07[t]	.01
4. Social X Family X Gender	.00	-.02	.00

[t]p < .10. *p < .05. **p < .01.

Time 3 Peer Composite and Family Relationships

	Step Δ R^2	β	pr^2
1. Time 1 Depression Composite	.27**	.52**	.27
Gender		-.06	.00
2. Peer Composite	.03**	.13**	.01
Family Relationships		.11**	.01
3. Peer X Gender	.00	-.05	.00
Family X Gender		.05	.00
Peer X Family		-.03	.00
4. Peer X Family X Gender	.00	.07	.00

**p < .01.

Table 20

Correlations between Time 3 Chronic Interpersonal Variables, Time 3 Episodic Stress Variables, and Depression and Anxiety Outcomes

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. Gender																	
2. MDD Hx	.08																
3. MDD onset	.12	.22															
4. T3 Chronic Peer Stress	-.05	.16	.23														
5. T3 Chronic Family Stress	.02	.21	.16	.31													
6. Fam event pre-MDD 90	.00	-.02	-.04	.01	.17												
7. Fam event pre-MDD 360	.03	.00	.11	.11	.19	.59											
8. Peer event pre-MDD 90	.01	.04	.14	.14	.04	.03	.07										
9. Peer event pre-MDD 360	.05	.04	.10	.12	.09	.03	.11	.55									
10. Fam event pre-quest 90	.02	.00	-.04	.00	.14	1.00	.58	-.01	.02								
11. Peer event pre-quest 90	.02	-.03	.09	.09	.05	.00	-.01	.92	.49	.02							
12. T1 Dep Comp	.03	.31	.22	.31	.29	.00	.03	.09	.04	-.01	.03						
13. T3 Dep Comp	-.05	.17	.27	.31	.27	.00	.00	.06	.05	-.01	.01	.52					
14. Dep change Score	-.06	-.18	.04	-.04	-.02	-.01	-.02	-.01	.01	-.01	.00	-.58	.40				
15. T1 Anx Comp	.11	.20	.15	.18	.21	-.07	-.01	.05	.03	-.09	.04	.62	.39	-.31			
16. T3 Anx comp	.09	.14	.11	.15	.13	-.03	-.02	.03	.02	-.04	.05	.41	.65	.17	.65		
17. Anx Change score	.01	-.13	.00	-.03	-.06	.05	.02	.02	.02	.04	.03	-.29	.26	.56	-.47	.37	
N	486	486	468	486	486	462	445	462	445	409	409	479	435	432	475	413	407

Note. N represents the number of valid values for that variable. Gender coded as 0 = male 1= female, MDD Hx = History of Current or Past Major Depressive Disorder at Time 1 interview, Fam event pre-MDD 90 (360) = Occurrence of a Family Event 90 (360) days before MDD onset, Peer event pre-MDD 90 (360) = Occurrence of a Peer Event 90 (360) days before MDD onset, Fam event pre-quest 90 = Occurrence of a Family event 90 days before Time 3 questionnaires, Peer event pre-quest 90 = Occurrence of a Peer Event 90 days before Time 3 questionnaire, T1 Dep Comp = Time 1 Depression Symptom Composite, T3 Dep Comp = Time 3 Depression Symptom Composite, Dep Change Score = Change in Depressive Symptoms (Time 3 – Time 1), T1 Anx Comp = Time 1 Anxiety Symptom Composite, Time 3 Anx Comp = Time 3 Anxiety Symptom Composite, Anx Change Score = Change in Anxiety Symptoms (Time 3 – Time 1).
All correlation coefficients $r > |.09|$ are significant at the $p < .05$ level; $r > |.15|$ are significant at the $p < .001$ level.

Table 21

Interactions Between Time 3 Peer Chronic Stress and Time 3 Peer Events Predicting Major Depressive Disorder

Order of Entry	Step χ^2	Step R^2	B	S.E.	Wald	Exp(B)	95% C.I.
1. Depression History	33.63**	.18	1.42	.39	13.18	4.12**	1.92-8.84
Peer Events 90 Days			1.06	.47	5.10	2.90*	1.15-7.30
Peer composite			.81	.23	12.28	2.24**	1.43-3.51
2. Peer Comp X Peer Events	.01	.00	.04	.57	.01	1.04	.34-3.17

Note. Chi-square refers to the increment in model improvement by the full step. Step R^2 refers to change in Nagelkerke R^2 values for that step.
* $p < .05$. ** $p < .01$.

Order of Entry	Step χ^2	Step R^2	B	S.E.	Wald	Exp(B)	95% C.I.
1. Depression History	38.85**	.21	1.28	.42	9.10	3.59**	1.56-8.24
T1 Depression Composite			.42	.23	3.30	1.53^t	.97-2.42
Peer Events 90 Days			1.10	.49	5.09	3.01*	1.16-7.84
Peer composite			.65	.25	6.57	1.92*	1.17-3.16
2. Peer Comp X Peer Events	.10	.00	-.18	.59	.10	.83	.26-2.64

$^t p < .10$. * $p < .05$. ** $p < .01$.

Current T1 MDD excluded

Order of Entry	Step χ^2	Step R^2	B	S.E.	Wald	Exp(B)	95% C.I.
1. Depression History	36.57**	.21	1.25	.44	8.02	3.49**	1.47-8.27
T1 Depression Composite			.56	.25	5.14	1.75*	1.08-2.83
Peer Events 90 Days			.90	.52	2.97	2.45^t	.88-6.81
Peer composite			.67	.26	6.35	1.95*	1.16-3.26
2. Peer Comp X Peer Events	.29	.00	-.33	.60	.30	.72	.22-2.35

$^t p < .10$. * $p < .05$. ** $p < .01$.

Table 22

Interactions Between Time 3 Family Chronic Stress and Time 3 Peer Events Predicting Major Depressive Disorder

Order of Entry	Step χ^2	Step R^2	B	S.E.	Wald	Exp(B)	95% C.I.
1. Depression History	25.59**	.14	1.38	.39	12.49	3.98**	1.85-8.56
Peer Events 90 Days			1.19	.47	6.53	3.29*	1.32-8.18
Family Relationships			.35	.19	3.44	1.42^{t}	.98-2.07
2. Family Relationships X Peer Events	.61	.00	.33	.43	.59	1.40	.60-3.27

Note. Chi-square refers to the increment in model improvement by the full step. Step R^2 refers to change in Nagelkerke R^2 values for that step.
$^{t}p < .10$. * $p < .05$. ** $p < .01$.

Order of Entry	Step χ^2	Step R^2	B	S.E.	Wald	Exp(B)	95% C.I.
1. Depression History	34.55**	.19	1.20	.42	8.24	3.33**	1.47-7.58
T1 Depression Composite			.53	.22	5.62	1.70*	1.10-2.63
Peer Events 90 Days			1.21	.48	6.25	3.34*	1.30-8.61
Family Relationships			.29	.21	1.92	1.33	.89-2.00
2. Family Relationships X Peer Events	.35	.00	.26	.45	.34	1.30	.54-3.11

* $p < .05$. ** $p < .01$.

Current T1 MDD excluded

Order of Entry	Step χ^2	Step R^2	B	S.E.	Wald	Exp(B)	95% C.I.
1. Depression History	32.84**	.19	1.16	.44	7.04	3.18**	1.35-7.47
T1 Depression Composite			.67	.23	8.12	1.95**	1.23-3.08
Peer Events 90 Days			.99	.52	3.70	2.69^{t}	.98-7.40
Family Relationships			.32	.21	2.25	1.38	.91-2.08
2. Family Relationships X Peer Events	.13	.00	.16	.46	.12	1.18	.48-2.89

$^{t}p < .10$. ** $p < .01$.

Table 23

Interactions of Time 3 Chronic Stress and Recent Family and Peer Episodic Stress Predicting Depression Symptoms: Regressed Change

Peer Events and Family Relationships

	Step Δ R^2	β	pr^2
1. Time 1 Depression Composite	.29**	.54**	.29
2. Recent Peer Events	.01*	.00	.00
Time 3 Family Relationships		.12**	.01
3. Peer Events X Family Relationships	.00	.00	.00

* $p < .05$. ** $p < .01$.

Peer Events and Peer Relationships

	Step Δ R^2	β	pr^2
1. Time 1 Depression Composite	.29**	.54**	.29
2. Recent Peer Events	.02**	-.01	.00
Time 3 Peer Relationship		.15**	.02
3. Peer Events X Peer Relationship	.00	-.05	.00

** $p < .01$.

Family Events and Peer Relationships

	Step Δ R^2	β	pr^2
1. Time 1 Depression Composite	.29**	.54**	.29
2. Recent Family Events	.02**	-.02	.00
Time 3 Peer Relationship		.15**	.02
3. Family Events X Peer Relationship	.00	-.02	.00

** $p < .01$.

Family Events and Family Relationships

	Step Δ R^2	β	pr^2
1. Time 1 Depression Composite	.29**	.54	.29
2. Recent Family Events	.02*	-.04	.00
Time 3 Family Relationships		.13**	.02
3. Family Events X Family Relationships	.00	.03	.00

* $p < .05$. ** $p < .01$.

Table 24

Interactions of Time 3 Chronic Stress and Recent Family and Peer Episodic Stress Predicting Anxiety Symptoms: Regressed Change

Peer Events and Family Relationships

	Step ΔR^2	β	pr^2
1. Time 1 Anxiety Composite	.43**	.65**	.43
2. Recent Peer Events	.00	.04	.00
Time 3 Family Relationships		.00	.00
3. Peer Events X Family Relationships	.00	.03	.00

**$p < .01$.

Peer Events and Peer Relationships

	Step ΔR^2	β	pr^2
1. Time 1 Anxiety Composite	.43**	.65**	.43
2. Recent Peer Events	.00	.03	.00
Time 3 Peer Relationship		.03	.00
3. Peer Events X Peer Relationship	.00	-.01	.00

**$p < .01$.

Family Events and Peer Relationships

	Step ΔR^2	β	pr^2
1. Time 1 Anxiety Composite	.43**	.65**	.43
2. Recent Family Events	.00	.00	.00
Time 3 Peer Relationship		.03	.00
3. Family Events X Peer Relationship	.00	-.03	.00

**$p < .01$.

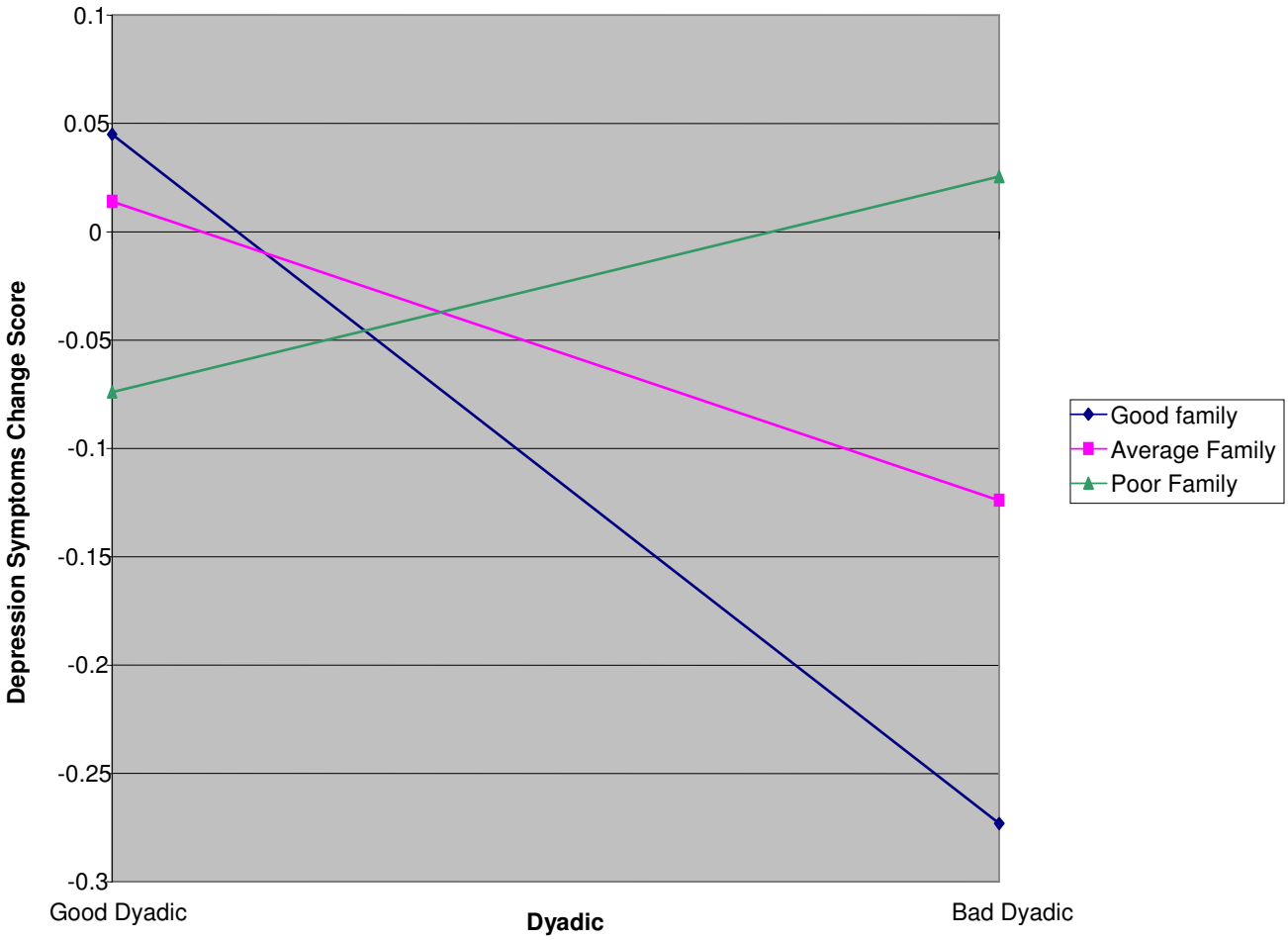
Family Events and Family Relationships

	Step ΔR^2	β	pr^2
1. Time 1 Anxiety Composite	.43**	.65**	.43
2. Recent Family Events	.00	.00	.00
Time 3 Family Relationships		.01	.00
3. Family Events X Family Relationships	.00	-.05	.00

**$p < .01$.

Figure Caption

Figure 1. Interaction between Time 3 dyadic and family functioning in the prediction of depression symptom change score for female participants.


0.1
0.05
0
-0.05
-0.1
-0.15
-0.2
-0.25
-0.3
Depression Symptoms Change Score
Good Dyadic
Dyadic
Bad Dyadic
Good family
Average Family
Poor Family

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Appendix A: Protocol for Recoding Event Dates

Situation	Coding
Event occurred within month of interview, unspecified date ("just happened")	Choose 1st of month if within first 15 days; 15th of month if within second 15 days (e.g., DOI: 4/8 use 4/1)
Says "couple weeks" before a specified time	Choose 2 weeks before date e.g., couple weeks before X-mas (use Dec. 11)
Has a week of a month (e.g. 3rd weeks of Feb 2004)	Choose 4th for first week, 11th for 2nd week, 18th for 3rd week, 25th for fourth week (e.g. 2/18/04)
Specifies a time before a landmark date (e.g. before x-mas 03)	Choose a date 5 days before (e.g., 12/20/03)
Event happened about the time of last interview (e.g., 1 year ago; last January for a January interview; etc)	Choose date of interview and previous year (e.g., last January for a 1/16/04 interview would be 1/16/03)
Specifies part of a year (e.g. early 2002)	Choose Feb 15th of that year (e.g. 2/15/02) for early; July 1 for mid; Nov 15th for late
Says season of a year (e.g. Fall 2003)	Choose Feb 1st for winter, May 1st for spring, August 1st for summer, and November 1st for fall (e.g. 11/1/03)
Specifies part of season of year (e.g., early Fall 2003)	Subtract 1 month for "early" and add one month for "late" to dates specified for seasons (e.g., early Fall 2003-10/1/03)
Has a range of 2 months (e.g., August-Sept 2003; or Dec 2003 or January 2004)	Choose first date of second month (e.g. 9/1/02; 1/1/04)
Has a range of 2 seasons (e.g. spring/summer 03)	Choose first day of second season (e.g. 6/21/03)
Range of exact dates given (e.g., 12/5-12/12/04)	Choose middle date
Has a range of months (e.g., April-June 2003)	Choose middle date of the median month (e.g., 5/15/03)
Specifies beginning of a school year (e.g., beginning junior year)	Choose September 15th of that year
Has a month and year (e.g. Jan of 2004 or about Jan 2004)	Choose the 15th of month
Says "mid" part of a month and year (e.g. mid March 2003)	Choose the 15th of month (e.g. 3/15/03)
Specifies mid to end of a month (e.g. mid to end of Jan 2002)	Choose the 20th of that month

specifies "end" or "late" part of a month and year (e.g. end April 2004)	Choose the 25thof that month (e.g. 4/25/04)
Specifies "early" part of a month and year (e.g., early June 2004)	Choose the 5th of that month (e.g. 6/5/04
Says late part of one month to early part of next month (e.g. late August/early Sept. 2003)	Choose the first day of the latter month (e.g. 9/1/03)
Rated as nonevent (99)	Follow rules as outlined above to ascertain date
Has "?"; No episodics or events; Lists only the year (e.g., 2000)	Leave blank
Has month/date/year (e.g., 12/15/04)	Same
Event happened "this week"	Subtract 4 days from day of interview
Event happened a definite time ago (e.g., 1 month ago, 2 weeks ago)	Subtract interval from target date (e.g., two weeks ago is 14 days from date of interview)
Event happened "one or two weeks ago"	Subtract ten days from time of interval